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VENTILATORY AND ACID-BASE RESPONSES TO LONG-TERM HYPERCAPNIA IN THE FRESHWATER TURTLE, *CHRYSEMYS PICTA BELLII*

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SUMMARY

The response to hypercapnia was studied in western painted turtles, *Chrysemys picta bellii* at 20°C. Ventilation, metabolic rate, arterial blood gases, blood pH and blood plasma ions were monitored periodically on individual turtles exposed to 5·7 % CO_2 for 72 h and then allowed to recover in air.

In response to hypercapnia, there is an immediate 10- to 15-fold increase in ventilation from control levels, which was maintained throughout the entire 5·7 % CO_2 breathing period. The first hour of CO_2 breathing caused an increase in Pa_{CO_2} from 24–39 mmHg with a concomitant decrease in pH and rise in $[HCO_3^-]$. $[HCO_3^-]$ rose from 42 to 50 mmol l^{-1} in the next 24 h of CO_2 breathing and remained at this level for the rest of the hypercapnic period. Small, significant increases in total $[Ca^{2+}]$ and total $[Mg^{2+}]$ were found; however, no changes were observed in the plasma Na^+, K^+ or Cl^- concentrations and the overall change in measured ions could not account for the increased $[HCO_3^-]$. The maximum change in $[HCO_3^-]$ attained in *Chrysemys* exposed to a more severe acidosis (14·3 % CO_2) for up to 18 h was the same as that seen in the animals breathing 5·7 % CO_2 (10 mmol l^{-1}) implying that there is an upper limit for the accumulation of $[HCO_3^-]$ in *Chrysemys* at 20 °C.

The blood pH of turtles recovering in air returned to the control value (7·56–7·74) within the first hour although Pa_{CO_2} did not return to the control value. The HCO_3^- ion concentration also remained elevated throughout the 48-h recovery period, which suggests that ionic compensation is a slower process.

The freshwater turtle employs two mechanisms to reduce the severity of an imposed respiratory acidosis: increased ventilation and changes in the strong ion difference. In spite of these responses, blood pH is not restored to the control value.

INTRODUCTION

In air-breathing vertebrates, adjustments for acid-base disturbances may be accomplished through ventilatory control of Pa_{CO_2} and by changes in the strong ion

Key words: Ionic compensation, calcium, chronic respiratory acidosis.

difference, which is the sum of the strong cations minus the sum of the strong anions (Stewart, 1978). Regulation of blood acid-base state through ventilatory adjustments acts *via* respiratory loss of CO_2 to maintain the appropriate Pa_{CO_2} level. Changes in the strong ion difference (SID) occur by a differential movement of strong cations and strong anions in or out of the blood. Hypercapnia, induced by CO_2 breathing, elicits a hyperventilatory response which has been well documented in mammals, birds and some reptiles (Glass & Wood, 1983). Functionally, this increased ventilation reduces the difference between inspired and lung P_{CO_2} and thereby minimizes the effect of CO_2 breathing on acid-base status. During chronic exposure to CO_2, the ventilatory response may be supplemented by adjustment of SID *via* ionic exchange mechanisms to further restore pH toward normal.

In the freshwater turtle, hyperventilation in response to CO_2 breathing has been described (Jackson, Palmer & Meadow, 1974; Milsom & Jones, 1980). However, the duration of CO_2 exposure in these studies was relatively brief, and compensatory ionic changes were not investigated. In this study we investigated the respiratory and ionic responses associated with long-term hypercapnia in the unanaesthetized, unrestrained turtle, *Chrysemys picta bellii*. The purpose of this study is to integrate the two compensatory mechanisms responsible for regulation of blood acid-base state during a CO_2-induced respiratory acidosis.

METHODS

Animals

Freshwater turtles, *Chrysemys picta bellii* (Gray), of either sex weighing 400–900 g, were obtained from Lemberger Consolidated Scientific, Germantown, Wisconsin. The turtles were kept in an aquarium at 25 °C and fed raw fish and dog food. They were fasted for at least 1 week prior to study.

Test gases

During the control period and recovery, the turtles breathed either compressed air or room air. Gas mixtures produced by Wösthoff pumps containing 5·7 % or 14·3 % CO_2 (P_{CO_2} = 42 and 106 mmHg, respectively) were used during CO_2 breathing.

Blood sampling and analysis

Blood was collected from a catheter (PE 90) inserted in the right subclavian artery distal to the thyroid artery. This area was exposed through a 2-cm hole trephined in the plastron which was eventually plugged with a Plexiglas disc and sealed with dental acrylic. The catheter was exteriorized through the skin at the base of the neck, was filled with heparinized turtle Ringer's solution, and secured to the carapace. Chloramphenicol (100 mg kg^{-1}), was administered post-surgically, and the turtles were allowed to recover in room air for at least 8 h. They were then transferred to a temperature controlled holding tank and allowed to acclimate for 16 h at 20 °C. Previous experiments have shown that 24 h gives adequate time for normal acid-base balance to be re-established.

Turtles breathing 5·7 % CO_2 were studied individually. Ventilation and metabolic rate were monitored and blood samples were collected at specified intervals and analysed for pH, blood gases and plasma ions. Turtles breathing 14·3 % CO_2 were studied in groups and blood samples were taken for blood gas analysis and plasma ion measurements. Ventilation was measured on only one individual of this group.

Blood samples were withdrawn using the following protocol: (1) 5·7 % CO_2 experiment: control, breathing room air, 1, 5, 24, 48 and 72 h in 5·7 % CO_2 and 1, 5, 24 and 48 h during recovery in air; (2) 14·3 % CO_2 experiment: control, breathing room air, 2 and 14–18 h in 14·3 % CO_2.

Two aliquots of blood were collected at each sampling period. One was drawn anaerobically in a heparinized glass syringe and analysed immediately for pH, P_{CO_2} and P_{O_2}, using a water-jacketed microelectrode unit (BMS-MK3, Radiometer, Copenhagen), maintained at 20 °C. The P_{CO_2} and P_{O_2} electrodes were calibrated with gas mixtures prepared by Wösthoff pumps (DIGAMIX Type 2M 303/a-f Bochum, West Germany); the P_{CO_2} values were selected to bracket the expected values for blood P_{CO_2}. The pH meter was calibrated with Radiometer precision buffers (S1500 and S1510). Plasma [HCO_3^-] was calculated using the Henderson-Hasselbalch equation with pK and CO_2 solubility constants derived by Reeves (1976). Total CO_2 was measured in some samples with a water-jacketed Cameron chamber maintained at 20 °C (Cameron, 1971). The purpose of this measurement was to check the accuracy of the published pK values for turtles under the conditions of this experiment. We found that there was little difference between the calculated and the measured [HCO_3^-] values. The second aliquot of blood was drawn in a 1 ml syringe, transferred to a 1·5 ml polypropylene tube and immediately centrifuged. The separated plasma was stored frozen for later analysis of ions. Sodium and potassium were measured with a flame photometer (Instrumentation Lab Model 143), total calcium and magnesium concentrations were determined with an atomic absorption spectrophotometer (Perkin-Elmer Model 280) and chloride concentrations were measured with the chloride titrator (Radiometer CMT 10). For three turtles breathing 14·3 % CO_2, whole blood was also analysed for ionized calcium with a calcium ion selective electrode (Orion Research) and analyser (Orion Microprocessor Ionalyzer/901).

Ventilation and gas exchange

Turtles were unrestrained in a 20 °C water bath covered with a plastic grid. In one corner of the grid was a ventilated plastic breathing chamber which gave the turtles their only access to air. The open bottom of the chamber was submerged in the water thus sealing the chamber. When the turtle breathed, the pressure change in this chamber was sensed by a Statham pressure transducer and recorded on a Grass Polygraph. The chamber was calibrated for volume by injecting air into the chamber from a 10 ml syringe. Flow of air into the chamber was controlled with a rotameter and maintained between 80 and 120 ml min^{-1}. Ventilatory gas leaving the chamber was dried with Drierite, and then passed through the analysis cells of an Applied Electrochemistry oxygen analyser (S-3A) and a Godart Capnograph. The outputs of the analysers were recorded on a dual channel Gould Brush 105 recorder. Calibration of the analysers was performed prior to measurements with gas mixtures which were

checked with a Scholander 0·5 ml analyser. CO_2 production and oxygen consumption were determined during steady-state conditions by integrating the area under the recorder curves by planimetry (OTT-Planimeter Type 30013). Metabolic rate analyses were corrected for differences in gas volume entering and leaving the chamber per unit time when the respiratory quotient was different from 1·0. The value of the respiratory quotient (RQ) was calculated using an equation derived by Otis (1964). The volumes of O_2 consumption and CO_2 production were corrected for body weight and converted to STPD.

Breathing records and O_2 consumption records were matched for periods at least 15 min in length. Total expired volume for an experimental interval was found by determining the sum of all expired tidal volumes. The ventilatory pattern was further analysed using the method employed by Milsom & Jones (1980). A ventilatory period (VP) was the collective duration of all the individual breaths within the 15-min interval. The non-ventilatory period (NVP) was the difference between the total measurement period and the ventilatory period. Mean expired minute ventilation was calculated and expressed as ml BTPS kg^{-1} min^{-1}. Frequency was calculated both as the

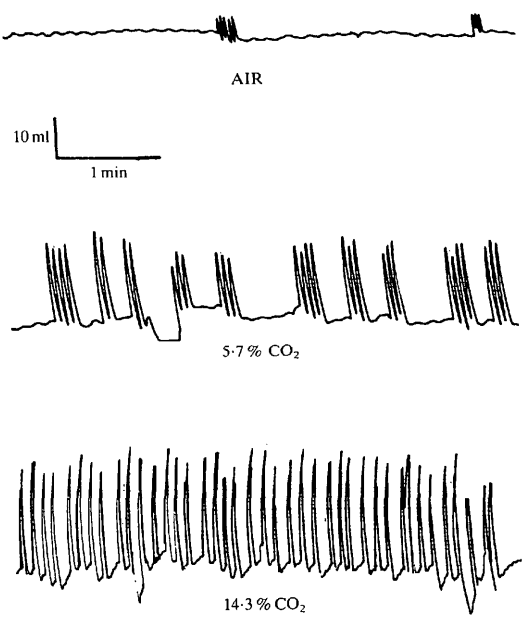

Fig. 1. Representative recordings of ventilatory patterns in a turtle at 20°C breathing air, 5·7 % CO_2 and 14·3 % CO_2 in air. Expiration results in an upward deflection of the flow traces. The scale at the upper left applies in all cases.

frequency during the ventilatory period (f_{VP}) and as the overall frequency during the test interval (f).

RESULTS

Ventilation and metabolic rate

During 5·7 % CO_2 breathing there was a pronounced increase in ventilation which continued throughout the 72 h of exposure (Table 1). The response included an increase in the overall frequency of breathing (f) and an increase in tidal volume. The frequency of breaths within each ventilatory period (f_{VP}) did not change but the fraction of time spent actively breathing increased from 0·09 to 0·23–0·34. When breathing 14 % CO_2, the turtles ventilated continuously. Representative ventilatory records of turtles breathing air, 5·7 % CO_2 and 14·3 % CO_2 are shown in Fig. 1.

Although there was a marked increase in pulmonary ventilation in turtles breathing 5·7 % CO_2, there was no change in metabolic rate (Table 1). The response was thus a sustained increase in the air convection requirement ($\dot{V}E/\dot{V}_{O_2}$) that exceeded the control value by 10–15 times (Fig. 2). Ventilation returned to the control level on resumption of breathing air.

Blood acid-base changes

CO_2 breathing caused an increase in Pa_{CO_2} and a decrease in pH (Table 2). The magnitude of the changes was related to the CO_2 concentration. The relationship between Pa_{CO_2} and ventilation for the 5·7 % CO_2 data is represented on a CO_2 response curve (Fig. 3), which shows that after 5 h of breathing CO_2 the elevated Pa_{CO_2} was matched by the increased ventilation. This new steady state persisted for the remainder of the hypercapnia but rapidly returned to normal upon resumption of breathing air. The HCO_3^- concentration also increased during CO_2 breathing, indicating metabolic compensation for the respiratory acidosis (Table 2).

The relationship between HCO_3^- and pH associated with 5·7 % CO_2 breathing is represented on a pH/[HCO_3^-] diagram (Fig. 4). During the first hour of CO_2 breathing, the Pa_{CO_2} increased to 39 mm Hg along an *in vivo* buffer line, the rise in [HCO_3^-] and decline in pH reflecting the buffering capacity of the extracellular fluid and red cells. The shallow slope of this buffer curve compared to the *in vitro* slope (Ultsch & Jackson, 1982) is probably due to the low buffering capacity of the interstitial fluid. A further increase in [HCO_3^-], apparent by 5 h, indicated a compensatory change in the plasma ionic composition due presumably to exchange processes with other body fluid compartments and/or renal base retention. Plasma [HCO_3^-] had stabilized after 24 h of CO_2 breathing. The acid-base responses of eight additional turtles breathing 14·3 % CO_2 are represented in Table 2. The resulting Pa_{CO_2} and pH changes were more extreme, and plasma [HCO_3^-] increased further after 14–18 h of CO_2 breathing, but was no higher than the values exhibited by the animals breathing 5·7 % CO_2.

Recovery of the turtles breathing 5·7 % CO_2 is represented by the dashed line on the pH/[HCO_3^-] diagram which is a titration back along an *in vivo* buffer line higher than but parallel to the initial one. The P_{CO_2} did not return to the control value and the

HCO_3^- concentration also remained elevated, which suggests a slower time course of the SID compensation.

Plasma ions

No significant changes were observed in the plasma concentrations of Na^+, K^+ or Cl^- during either 5·7 % or 14·3 % CO_2 breathing, although small but significant increases were found for both total calcium and magnesium (Table 3). Ionized calcium was measured in whole blood of three turtles breathing 14·3 % CO_2 and there was a small but significant increase (Table 3).

DISCUSSION

Prolonged CO_2 breathing is a convenient tool for evaluating the ventilatory and ionic responses to an imposed acidosis. In this study of the western painted turtle,

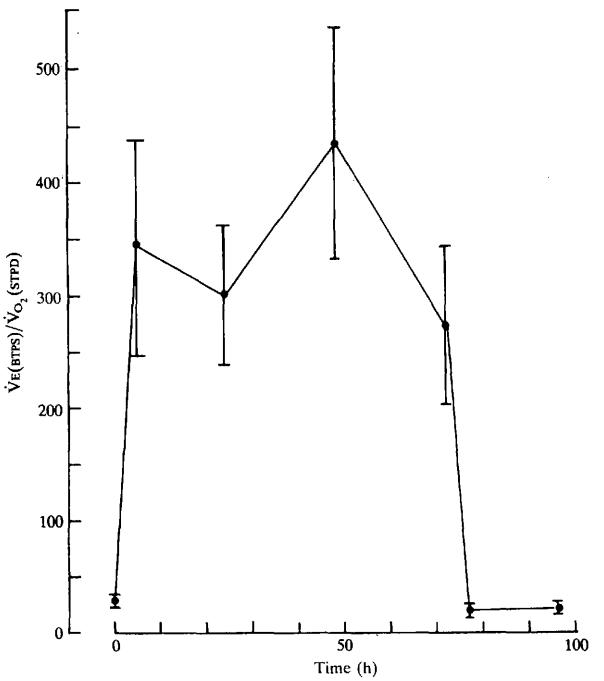

Fig. 2. The mean values (with the standard error bars) of the ratio, $\dot{V}_E/\dot{V}_{O_2}$, plotted against time. 5·7 % CO_2 in air was breathed from 0–72 h.

Table 1. *Ventilation and metabolism data of turtles throughout the experiment beginning with control (air), then 5·7% CO_2 breathing and recovery in air*

riod	N	f_{VP} (min^{-1})	VP / VP+NVP	f (min^{-1})	Vt ($ml\,kg^{-1}$)	$\dot{V}_E$ ($ml\,min^{-1}\,kg^{-1}$)	$\dot{V}_{O_2}$ ($ml\,min^{-1}kg^{-1}$)	$\dot{V}_E(ml)$ / $\dot{V}_{O_2}(ml)$
ntrol	7	25·1 ± 3·8	0·09 ± 0·01	1·9 ± 0·3	9·4 ± 3·1	16·7 ± 4·0	0·58 ± 0·10	29·6 ± 5·8
% CO_2								
h	4	26·1 ± 3·3	0·34 ± 0·06	9·4 ± 2·6	22·3 ± 3·5	235·8 ± 91·4	0·62 ± 0·10	342·2 ± 96·2
4 h	5	26·3 ± 3·8	0·23 ± 0·03	6·1 ± 1·2	26·8 ± 3·1	165·9 ± 46·1	0·56 ± 0·07	301·8 ± 63·0
8 h	6	26·8 ± 6·3	0·28 ± 0·04	7·9 ± 2·9	29·4 ± 3·4	224·6 ± 78·5	0·57 ± 0·11	434·9 ± 101·4
2 h	5	23·0 ± 3·7	0·23 ± 0·05	5·4 ± 1·1	30·1 ± 5·8	160·7 ± 31·1	0·63 ± 0·10	274·2 ± 70·2
% CO_2 Recovery								
h	3	34·3 ± 9·4	0·08 ± 0·01	3·0 ± 1·1	9·6 ± 5·3	18·2 ± 4·1	0·92 ± 0·11	20·5 ± 4·8
4 h	5	23·4 ± 5·5	0·12 ± 0·03	3·0 ± 0·9	4·7 ± 1·3	12·9 ± 4·3	0·65 ± 0·21	22·1 ± 4·7

Values are ± standard error.
All volumes expressed as BTPS except $\dot{V}_{O_2}$ which is STPD.
VP, ventilatory period; NVP, non-ventilatory period; f_{VP}, breathing frequency during the ventilatory period; f, overall breathing frequency; Vt, tidal volume; $\dot{V}_E$, al pulmonary ventilation; $\dot{V}_{O_2}$, oxygen consumption.

Chrysemys picta bellii, we observed significant compensatory increases in ventilation and in plasma [HCO_3^-] during 3 days of 5·7 % CO_2 breathing. These responses both reached stable steady states that persisted for the remainder of the exposure. The ventilation increased during the first few minutes to a plateau after 5 h, and then continued unabated for the next 3 days. This sustained increase in ventilation is unlike the results reported in dogs, in which the acute response declined back to control after 3 days (Jennings & Chen, 1975). In studies on humans breathing CO_2 for extended periods, there was also an abatement of the initial response due to a restoration of arterial plasma and CSF pH (see Dempsey & Forster, 1982). The blood acid-base status of the turtle was not restored to normal during CO_2 breathing, and the constant ventilation suggests that the acidotic environment of the central chemoreceptors also remained uncompensated (Hitzig & Jackson, 1978).

The steady state level of ventilation we observed during 5·7 % CO_2 breathing was some 10–15 times the control level, which is comparable to the 2·5-fold increase

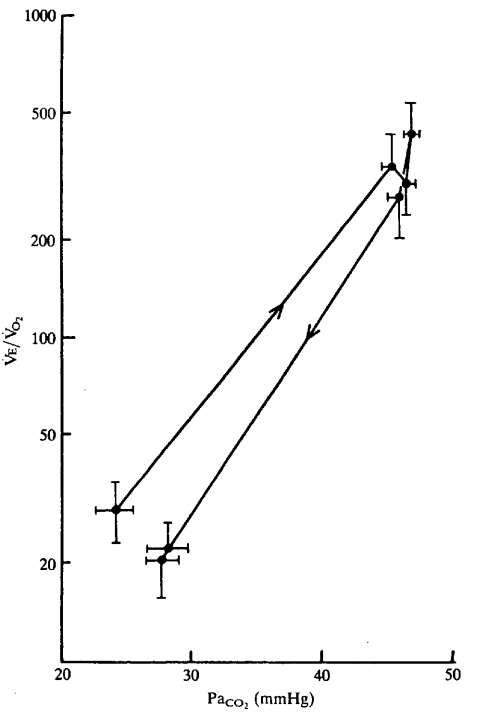

Fig. 3. Ventilatory response to increased Pa_{CO_2} due to breathing 5·7 % CO_2 in air including control and recovery values while turtles breathed air. Note that ventilation is expressed as log $\dot{V}_E/\dot{V}_{O_2}$.

reported by Milsom & Jones (1980) during 5 % CO_2 breathing in the same species, and to the 10-fold increase reported by Jackson *et al.* (1974) during 6 % CO_2 breathing in *Chrysemys*. Freshwater turtles are quite responsive to CO_2, more so than other reptiles that have been studied (Nielsen, 1961; Templeton & Dawson, 1963; Gratz, 1979). Both pulmonary (Milsom & Jones, 1980) and brain (Hitzig & Jackson, 1978) chemoreceptors, which presumably mediate the CO_2 response, have been described in turtles.

The ionic, or metabolic, response to hypercapnia developed more slowly than the respiratory response, and did not reach a stable state until the 24th hour. The magnitude of the compensatory response, estimated by the increase of plasma $[HCO_3^-]$, was some 10 mmol l^{-1} above the initial 1-h response; however, because of the normally high control $[HCO_3^-]$ of the turtle (41 mmol l^{-1}), the compensatory increase had only a small effect on blood pH. In contrast, fish, exposed to a hypercapnic environment that produced an initial respiratory acidosis of similar severity, restored normal or near normal blood pH by a compensatory increase in plasma $[HCO_3^-]$ (Eddy, Lombolt, Weber & Johansen, 1977; Toews, Holeton & Heisler, 1983). Two differences appear to account for the superior performance of fish in this regard. First, fish can elevate $[HCO_3^-]$ by a greater amount than we observed in the turtles (perhaps due to their effective branchial ion exchange mechanisms); in the study of Eddy *et al.* (1977), for example, the plasma $[HCO_3^-]$ of the rainbow trout, *Salmo gairdneri*, increased from 4 to 23 mmol l^{-1}, more than twice as great a change as in the turtles. Second, and of foremost importance, the control $[HCO_3^-]$ of fish is low, about 4–13 mmol l^{-1} so that if similar increases in $[HCO_3^-]$ occur in a fish and a turtle, the elevation of pH will be much greater in the fish. On the

Table 2. *Blood acid-base values of turtles breathing air (control and recovery) and either 5·7 % CO_2 or 14·3 % CO_2 in air*

Period		pH	HCO_3^- (mmol l^{-1})	P_{CO_2} (mmHg)
5·7 % CO_2 breathing, $N = 7$				
Control		7·76 ± 0·03	41·4 ± 1·6	24·3 ± 1·8
1 h		7·55 ± 0·02	42·5 ± 1·7	39·3 ± 1·2
5 h		7·52 ± 0·02	46·2 ± 2·3	45·3 ± 0·8
24 h		7·54 ± 0·02	49·5 ± 2·0	46·4 ± 0·9
48 h		7·56 ± 0·01	51·5 ± 1·5	46·9 ± 0·6
72 h		7·56 ± 0·02	51·1 ± 1·5	45·9 ± 1·0
1 h recovery		7·74 ± 0·04	50·8 ± 0·9	30·3 ± 2·7
5 h recovery		7·76 ± 0·02	49·6 ± 1·7	27·9 ± 1·3
24 h recovery		7·75 ± 0·02	46·6 ± 1·7	28·3 ± 1·9
48 h recovery		7·68 ± 0·03	46·2 ± 1·9	30·8 ± 3·0
14·3 % CO_2 breathing				
	N			
Control	8	7·74 ± 0·02	40·6 ± 1·5	24·9 ± 1·6
2 h	3	7·26 ± 0·07	46·6 ± 2·3	75·6 ± 9·6
14–18 h	8	7·23 ± 0·02	51·2 ± 1·5	98·5 ± 2·2

Values are ± standard error.

other hand, because of the low blood P_{CO_2} of the fish, the animal requires a lower ambient P_{CO_2} than the turtles to produce the same acute acidosis, so that the advantage of the fish over the turtles is its superior pH compensation, not its superior tolerance to elevated P_{CO_2}.

As recently pointed out (N. Heisler, personal communication), a number of fish and amphibians, when exposed to graded levels of hypercapnia, achieve maximal compensatory increases in plasma $[HCO_3^-]$ that are independent of further increases in P_{CO_2}. Once this level is reached, even infused HCO_3^- that improves pH is excreted to return the $[HCO_3^-]$ back to this set level (Heisler, Forcht, Ultsch & Anderson, 1982). The level reached in these animals is about 25–30 mmol l^{-1}, even though their control HCO_3^- concentrations vary from 4 to 22 mmol l^{-1}. Consequently, the magnitude of the compensation depends on control plasma $[HCO_3^-]$, and is therefore greatest in fish with characteristically low concentrations of HCO_3^-. The turtle, *Chrysemys*, on the other hand, departs markedly from this general scheme. Its control plasma $[HCO_3^-]$ is well above the upper compensatory limit of the fish and amphibians, and its compensatory adjustment sends it higher still. Like the other animals, however, *Chrysemys* apparently has an upper limit – in this case, about 50 mmol l^{-1}. This level was reached when turtles breathed 5·7 % CO_2, and no further

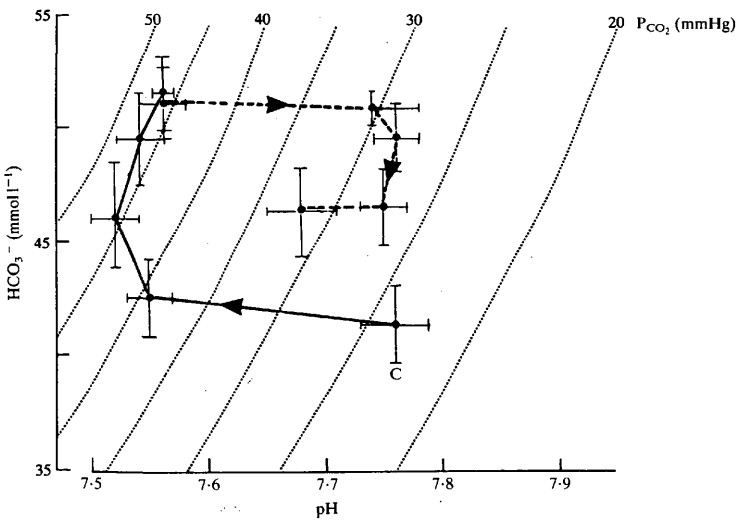

Fig. 4. pH *versus* HCO_3^- diagram depicting arterial blood acid-base changes that occurred in seven animals during the experiment. C represents the control breathing period in air and the arrows follow the time course of the experiment: 1, 5, 24, 48, 72 h breathing 5·7 % CO_2 in air and 1, 5, 24, 48 h recovery in room air.

elevation was observed when they breathed 14·3 % CO_2 (Table 2). This [HCO_3^-] may reflect the maximum ability of the kidney to retain filtered HCO_3^-.

The increased [HCO_3^-] we observed must be associated with an increase in the strong ion difference SID; however, our analysis of plasma strong ions failed to reveal any consistent changes that could account for the response. One possibility is that we did not measure the particular ion or ions that were involved, although this seems unlikely. A more plausible explanation, is that the net SID effect was the result of a number of subtle changes in the ions measured, and the resolution of the analyses was not adequate to detect these changes. We did see significant increases in plasma [Ca^{2+}] and [Mg^{2+}] but by themselves these were too small to account for the observed compensation, although there did appear to be a preferential increase in ionized Ca^{2+} in the turtles exposed to 14·3 % CO_2. Some fish and amphibia show an increased SID when exposed to hypercapnic conditions. The rainbow trout increased [HCO_3^-] and decreased [Cl^-] by about 20 mmol l^{-1} in response to water bubbled with CO_2 (Eddy *et al.* 1977). In tiger salamander, *Ambystoma tigrinum*, exposed to 3 % CO_2, plasma [HCO_3^-] increased from 15 to 22 mmol l^{-1} and was associated with a rise in K^+ from 3·0 to 3·5 mmol l^{-1} and a decrease in [Cl^-] from 90 to 84 mmol l^{-1} (Stiffler, Tufts & Toews, 1982). This ability to compensate for an acid-base disturbance through various ionic exchange mechanisms may reflect the primary means by which these

Table 3. *Plasma ion data of turtles breathing air (control and recovery), 5·7 % CO_2 in air and 14·3 % CO_2 in air. Ionized Ca^{2+} was also measured on three turtles breathing 14·3 % CO_2 in air*

Period	K^+	Na^+	Cl^-	Total Ca^{2+}	Total Mg^{2+}
5·7 % CO_2 breathing ($N = 7$)•					
Control	2·2 ± 0·3	124·9 ± 2·2	79·7 ± 3·3	6·2 ± 0·2	4·2 ± 0·1
1 h	2·2 ± 0·3	123·1 ± 2·9	81·4 ± 2·4	6·2 ± 0·2	4·0 ± 0·1
5 h	2·1 ± 0·3	128·7 ± 2·4	81·0 ± 3·0	6·4 ± 0·1	4·0 ± 0·1
24 h	2·2 ± 0·2	121·4 ± 3·0	79·0 ± 2·2	7·2 ± 0·2	4·3 ± 0·1
48 h	2·2 ± 0·2	120·1 ± 4·4	80·0 ± 2·7	7·2 ± 0·2	4·3 ± 0·1
72 h	2·5 ± 0·2	118·7 ± 3·0	79·1 ± 2·2	7·4 ± 0·3	4·3 ± 0·1
1 h recovery	2·7 ± 0·2	118·1 ± 4·0	79·3 ± 2·2	7·2 ± 0·2	4·5 ± 0·1
5 h recovery	2·6 ± 0·2	120·3 ± 3·4	78·4 ± 2·3	6·6 ± 0·2	4·4 ± 0·1
24 h recovery	2·4 ± 0·2	115·8 ± 3·9	79·3 ± 1·9	6·6 ± 0·2	4·2 ± 0·1
48 h recovery	2·4 ± 0·2	120·1 ± 5·9	80·4 ± 2·0	6·6 ± 0·2	4·0 ± 0·9
14·3 % CO_2 breathing					
Period N					
Control 8	2·8 ± 0·2	126·3 ± 1·9	85·0 ± 1·9	5·6 ± 0·4	3·3 ± 0·3
2 h 3	2·8 ± 0·2	123·8 ± 2·6	83·0 ± 2·1	6·6 ± 0·1	3·4 ± 0·2
14 h 5	2·7 ± 0·3	133·4 ± 1·1	87·0 ± 1·5	6·6 ± 0·1	5·3 ± 0·2
18 h 3	3·1 ± 0·1	125·8 ± 3·7	84·6 ± 2·1	7·8 ± 0·3	5·0 ± 0·3
Period $N = 3$ Ionized Ca^{2+}					
Control 3·4 ± 0·1					
2 h 4·0 ± 0·3					
18 h 6·0 ± 0·2					

Values are ± standard error.
• Ca^{2+} and Mg^{2+}, $N = 6$.

organisms respond to changes in blood acid-base state. Turtles normally depend on the efficient respiratory control of P_{CO_2} to maintain acid-base homeostasis. During hypercapnia, however, this means of control can only minimize the acidosis, and cannot prevent it from occurring.

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