Respiration Physiology, 77 (1989) 337–350
Elsevier

RESP 01567

Factors terminating nonventilatory periods in the turtle, *Chelydra serpentina*

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(Accepted for publication 2 May 1989)

Abstract. Pa_{O_2}, Pa_{CO_2} and pHa were measured via an extracorporeal loop in conscious snapping turtles (*Chelydra serpentina*) breathing air or hypoxic (10, 15% O_2), hyperoxic (30% O_2), or hypercapnic (2% CO_2) gases. Turtles breathed into an inverted funnel ventilated with the test gas. Breathing was recorded with a differential pressure transducer. In all turtles, nonventilatory periods were interrupted by breathing episodes containing multiple breaths. In normoxia, Pa_{O_2} at the end of nonventilatory periods ranged from 22–128 mm Hg, although Pa_{CO_2} showed a less than 5 mm Hg variation about the mean. There was a positive correlation between Pa_{CO_2} at the end of the nonventilatory period and the number of breaths in the succeeding period of ventilation. Pa_{CO_2} at the end of nonventilatory periods did not change significantly in hyperoxia, although mean Pa_{O_2} was significantly increased. In hypoxia, on the other hand, mean Pa_{O_2} was significantly reduced and Pa_{CO_2} at the end of the nonventilatory period was slightly, but significantly lower. Nonventilatory periods were shorter when turtles breathed 15% O_2 (9.3 ± 1.2 min) or 10% O_2 (5.5 ± 0.3 min) than when they breathed air (17.6 ± 3.4 min). The results indicate that, in undisturbed turtles, the most important stimulus triggering a breathing episode is the rise in Pa_{CO_2} to a critical value during the preceding nonventilatory period. An increase in hypoxic drive shortens the nonventilatory period. However, in normoxia, Pa_{O_2} at the end of many nonventilatory periods probably does not fall sufficiently to stimulate O_2-sensitive chemoreceptors.

Chemoreception; Hypercapnia; Hypoxia; Periodic breathing; Turtle; Ventilation

Chelonian reptiles typically demonstrate an intermittent pattern of ventilation, in which the length of the nonventilatory period represents an important respiratory variable (Milsom and Jones, 1980; Glass *et al.*, 1983; Shelton *et al.*, 1986). For aquatic chelonians, which are normally submerged, such intermittent ventilation involves a suite of behaviours ranging from simple neck extension to actively swimming to the surface (Lenfant *et al.*, 1970; West, Bevan and Butler, unpublished data). That respiratory drive reaches the level of perception is suggested by reports that submerged *Pseudemys scripta* can be trained to press a bar in order to be allowed access to air (Van Sommers, 1963).

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Fluctuations in respiratory blood gases and pH are an inevitable consequence of intermittent ventilation in both terrestrial and aquatic chelonians. Pa_{O_2} and pHa fall, while Pa_{CO_2} rises, during the nonventilatory period (Burggren and Shelton, 1979). It seems reasonable to assume that some combination of these stimuli may be important in determining the length of the nonventilatory period by eventually triggering ventilation, at least in undisturbed animals. Chelonians possess several receptor groups by which changes in such variables could be measured. For example, the increase in Pa_{CO_2} accompanying the nonventilatory period could be sensed by central chemoreceptors (Hitzig and Jackson, 1978), carotid artery chemoreceptors (Ishii and Ishii, 1986), or by the modulation of pulmonary stretch receptor discharge (Jones and Milsom, 1979). The concomitant fall in Pa_{O_2} may provide a stimulus for chemoreceptors located within the carotid, pulmonary, or aortic arches (Benchetrit *et al.*, 1977; Ishii *et al.*, 1985; Ishii and Ishii, 1986).

There are few studies in which the relationship between respiratory blood gases and pH and the termination of breath holding in undisturbed, freshwater chelonians has been addressed. In actively diving *Pseudemys scripta*, no clear correlation was found between Pa_{O_2}, Pa_{CO_2}, pHa, and the initiation or termination of breathing periods (Burggren and Shelton, 1979). In *Chelys fimbriata*, a sedentary turtle, a reduction in Pa_{O_2} by nitrogen breathing resulted in a shortening of the nonventilatory period as did a 75% increase in alveolar Pa_{CO_2} (Lenfant *et al.*, 1970). Hypoxia also decreased the diving time in *Chrysemys picta* (Glass *et al.*, 1983). However, the likelihood of a hypoxic stimulus making the major contribution to the termination of the nonventilatory period in undisturbed turtles seems slight in view of the typically high levels of Pa_{O_2} and arterial oxygen saturations reported at the end of voluntary periods of submergence (Lenfant *et al.*, 1970; Burggren and Shelton, 1979).

Therefore, we set out to test the hypothesis that the length of the nonventilatory period in the undisturbed snapping turtle, *Chelydra serpentina* is determined by Pa_{CO_2} rising to a threshold value, which stimulates breathing behaviour. Pa_{O_2}, Pa_{CO_2} and pHa were measured continuously via an extracorporeal loop in animals breathing air, or hypoxic or hypercapnic gas mixtures, and their values determined at the initiation of ventilatory periods.

Material and methods

Animals and surgical preparation. Seven snapping turtles, *Chelydra serpentina* (average body mass, 3.09 kg) used in the study were obtained from a commercial dealer and were maintained for several weeks at 20–25 °C on a diet of beef liver. Turtles were prepared for the experiments by performing upstream, occlusive cannulations of the sciatic artery and accompanying sciatic vein (50 cm of polyethylene tubing, PE 90; Intramedic). The animals were under cold-induced and local (2% lidocaine injections) analgesia during the procedure. Muscle and skin layers of the incision were closed separately, and the wound was further sealed with an external application of cyanoacrylate glue. Blood

circulation collateral to the cannulation sites was sufficient to allow the turtles full use of the cannulated hindlimb throughout the study. Turtles were given a minimum of 24 h to recover from surgery and to acclimatize to the experimental tank.

Experimental measurements. Breathing episodes and the associated changes in blood gases and pH were measured while each turtle moved freely and undisturbed within a 60 L, water-filled tank at room temperature, 22–24 °C. The depth of water was 35–40 cm. Turtles were visually screened from the experiments at all times. The water surface was covered with a styrofoam panel with a single 10 cm circular opening through which a turtle could breathe. Breathing events were signalled by airflow passing through the narrow end of a funnel that was inverted over the breathing hole. Movement of air through the funnel due to inspiration or expiration was indicated as an air pressure difference between two sides of a mesh paper screen placed across the funnel opening. A Grass PT5A differential pressure transducer was used to record breathing, and transducer output was displayed on one channel of a Narco BioSystems MK-IV physiograph. Room air was passed through the funnel at a constant rate of 250 ml · min^{-1} to ensure the turtles did not rebreathe their own exhalations.

Experimental protocol. Continuous recording of arterial blood P_{O_2}, P_{CO_2} and pH in undisturbed turtles was accomplished by using an extracorporeal vascular loop incorporating sciatic arterial and venous cannulas (see Burggren *et al.*, 1989). Blood withdrawn from the arterial cannula at 0.27–0.55 ml · min^{-1} with a Cole-Parmer peristaltic pump was passed through a modified Instrumentation Laboratories Micro 13 pH/Blood Gas analyzer, into a gas bubble trap, and was returned to the animal via the venous cannula. All tubing was siliconised with Prosil-28. The analyser electrodes were held at room temperature. Output from the pH/blood gas analyser was recorded on three additional channels of the Narco physiograph, simultaneous with the recording of breathing events.

The extracorporeal a-v loop was run for 6–8 h per day for three consecutive days on each turtle. Blood sampled from the extracorporeal loop showed no signs of erythrocyte lysis. Oxygen, CO_2 and pH electrodes were calibrated at the beginning and end of the day and at 2–3 h intervals in between. Drift in the electrode output between calibrations was assumed to be linear over time, and values were corrected accordingly. The actual respiratory values (arterial P_{O_2}, P_{CO_2}, and pH) at the initiation of breathing were determined directly from the inflections of the recorded tracings (see fig. 1A,B), thus, the time delay due to the residence time of blood in the extracorporeal loop was not corrected. Electrode response time to step changes in the measured variables was determined by switching between room-temperature saline equilibrated at different partial pressures or pHs (P_{O_2}, 52.5–98.5 mm Hg; P_{CO_2}, 24.5–36.5 mm Hg; pH, 6.90–7.41). The flow chosen was the lowest flow used in the experiments, 0.27 ml · min^{-1}. Under these conditions, the time constants (63% response) of the O_2, CO_2 and pH electrodes were 105, 90 and 41 sec, respectively. This includes the time taken (22 sec) to flush 0.1 ml of electrode dead space, but excluded any other time

delays. In normoxia, nonventilatory periods were more than an order of magnitude longer than the electrode time constants, and longer than three time constants (95% response) under all conditions except in the case of the O_2 electrode when animals breathed 10% O_2. Therefore, no attempt was made to correct measured values to compensate for electrode response time.

Normoxic, control conditions were established by passing room air through the breathing funnel. Breathing frequency and changes in arterial blood gases were also measured while animals breathed hypoxic (15%, 10% O_2) and hypercapnic (2% CO_2) gas mixtures delivered into the funnel at the same flow rate via a Wösthoff pump.

Blood analysis. Three to five ml blood samples were withdrawn from six of the experimental animals at the end of experiments in which *in vivo* blood gas values were monitored. Hemoglobin (Hb) concentration was measured on a 25 μl blood sample using a spectrophotometric hemoximeter (Model OSM 2, Radiometer). Hematocrit was determined on 10 μl of blood after centrifugation at 10000 × g for 3 min. Blood oxygen capacity of a 20 μl blood sample equilibrated with atmospheric air was determined according to the method of Tucker (1967). Blood oxygen equilibrium curves were determined by equilibrating 1 ml of blood with $O_2/N_2/CO_2$ gas mixtures and then using the hemoximeter to measure % O_2 saturation. Oxygen equilibrium curves were generated at a $P_{a_{CO_2}}$ of 28 and 57 mm Hg. The pH of the blood near the P_{50} of each curve was determined with an I. L. Micro 13 blood gas analyzer, allowing calculation of the Bohr effect.

Blood O_2 saturation and O_2 content *in vivo* were calculated by using the oxygen equilibrium curves. Bohr effect and O_2 capacity for each animal's blood and the arterial Pa_{O_2} as measured by the extracorporeal loop. In one animal in which *in vivo* data were collected, there was no information for the *in vitro* properties of blood, so the average of the values of the other six animals were used for calculation of % O_2 saturation and O_2 content of arterial blood.

Data traces were analysed with the aid of a digitising tablet (Jandel Scientific) and appropriate software. The data were analysed to provide means ± SE where appropriate. Treatment effects were evaluated using analysis of variance (ANOVA). The fiducial level of significance was considered to be $P < 0.05$.

Results

Blood gases and ventilation in normoxic conditions. Data were obtained from six undisturbed turtles under normoxic conditions. All turtles showed an intermittent pattern of ventilation, in which nonventilatory periods (17.6 ± 3.4 min) were interrupted by breathing periods containing multiple breaths (5.2 ± 1.2 breaths). When undisturbed in shallow water, the only behaviour manifested by *Chelydra* was neck extension to the surface for the purpose of breathing, suggesting that non-respiratory related drives were not significant in influencing the duration of the nonventilatory period under these conditions.

TABLE 1

Pa_{O_2}, Pa_{CO_2} (mm Hg) and pHa measured at the end of nonventilatory periods (NVP), the duration of the nonventilatory period (minutes) and the number of breaths performed during ventilatory periods in undisturbed, normoxic snapping turtles. Values are given ± SE. R = range, CV = coefficient of variation. $N = 6$, $n = 130$.

Turtle No.	Pa_{O_2}	Pa_{CO_2}	pHa	No. of breaths	Duration of NVP
1	77.5	41.8	7.674	4.9	17.1
	(28–128)	(39–44)	(7.65–7.70)	(2–8)	(7–32)
2	92.8	33.4	7.685	3.9	11.1
	(74–107)	(31–37)	(7.62–7.75)	(2–7)	(5–21)
3	54.4	41.1	7.632	5.25	23.1
	(40–103)	(38–45)	(7.60–7.67)	(2–12)	(11–39)
4	64.0	41.9	7.666	6.2	15.3
	(22–90)	(37–49)	(7.61–7.73)	(1–17)	(5–29)
5	73.9	38.0	7.668	6.9	23.2
	(33–99)	(34–43)	(7.62–7.71)	(3–13)	(11–50)
6	64.6	39.1	7.651	4.0	17.9
	(41–83)	(36–41)	(7.62–7.69)	(2–7)	(4–31)
Total					
Mean	69.5	39.2	7.661	5.2	17.6
SE	8.8	1.5	0.014	1.2	3.4
R	(22–128)	(31–49)	(7.60–7.75)	(1–17)	(4–50)
CV	28.5	8.5	0.42	51.9	43.8

Breathing periods were initiated at a Pa_{O_2} of 69.5 ± 8.8 mm Hg, Pa_{CO_2} of 39.2 ± 1.5 mm Hg and pHa of 7.661 ± 0.014 ($N = 6$, $n = 130$; table 1), and were associated with an increase in Pa_{O_2}, a fall in Pa_{CO_2}, and a corresponding increase in pHa (fig. 1A).

Maximum Pa_{O_2} attained after breathing periods was 92.6 ± 7.4 mm Hg with a corresponding Pa_{CO_2} of 32.9 ± 2.7 mm Hg and pHa of 7.722 ± 0.007. Pa_{O_2} fell between breathing periods at a rate of 1.7 ± 0.2 mm $Hg^{-1} \cdot min^{-1}$. The fall in Pa_{O_2} was associated with a rise in Pa_{CO_2} at a rate of 0.5 ± 0.1 mm $Hg \cdot min^{-1}$, and a corresponding fall in pHa at a rate of 0.005 ± 0.001 unit $\cdot min^{-1}$ (table 2). Pa_{O_2} at the end of nonventilatory periods varied from 22–128 mm Hg, Pa_{CO_2} from 31–49 mm Hg and pHa from 7.60–7.75 ($N = 6$, $n = 130$; table 1). Figure 2 (solid circles) illustrates the comparative variability of Pa_{O_2} and Pa_{CO_2} by plotting the deviation from the mean partial pressure at the termination of the nonventilatory period in each animal for individual nonventilatory periods. Pa_{O_2} varied from nearly 80 mm Hg above, to over 40 mm Hg below the mean value. Pa_{CO_2} showed a less than 5 mm Hg variation about the mean.

Blood gases and ventilation in hypoxia, hyperoxia and hypercapnia. Ventilation of the breathing funnel with 30% O_2 resulted in an elevation of both maximum and minimum Pa_{O_2}, but did not significantly affect Pa_{CO_2} values (figs. 1B, 2; table 2). The Pa_{CO_2} at the end of nonventilatory periods, 39.2 ± 1.8 mm Hg, was not significantly different

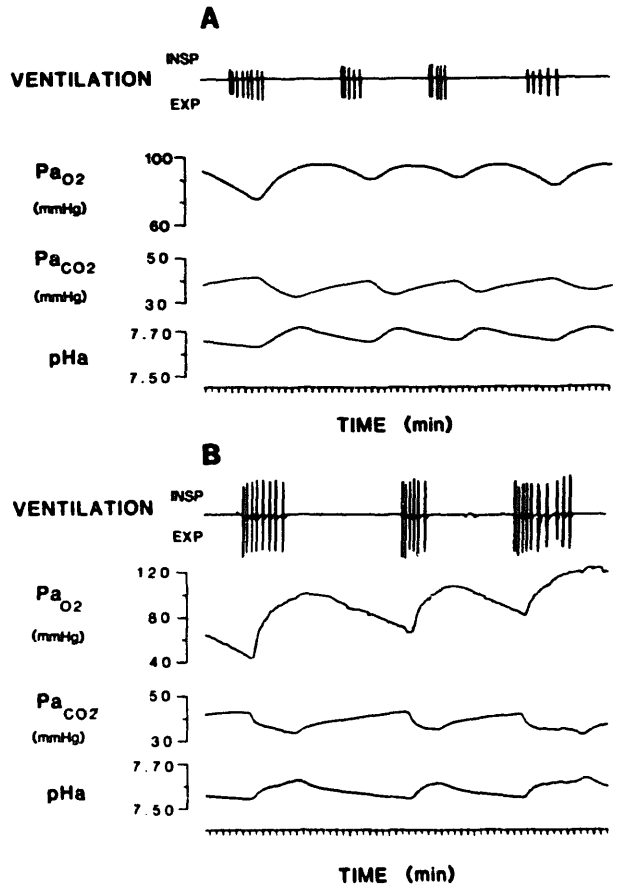

Fig. 1. Ventilatory patterns and the changes in Pa_{O_2}, Pa_{CO_2} and pHa associated with ventilatory and nonventilatory periods in *Chelydra serpentina* (2.3 kg). In A the turtle is breathing air. In B the ventilating gas was switched to 30% O_2 before the first ventilatory period illustrated. The traces are uncorrected for the time delay due to blood flow in the extracorporeal loop and electrode response time.

from the normoxic value, although mean Pa_{O_2} at the start of ventilation was raised to 103.4 ± 10.6 mm Hg (N = 3, n = 7).

Under hypoxic conditions (10% O_2), Pa_{O_2} fell to 49.5 ± 8.5 mm Hg (N = 4, n = 25) at the end of nonventilatory periods. The corresponding Pa_{CO_2} (34.5 ± 2.2 mm Hg) was slightly, but significantly, lower than under normoxic conditions (39.2 ± 1.5 mm Hg) (fig. 2, table 2).

Under hypercapnic conditions (2% CO_2 in air), ventilation was initiated at a Pa_{CO_2} not significantly different from that during air-breathing (38.9 ± 1.8 mm Hg; N = 3, n = 13) although the corresponding Pa_{O_2} was elevated to 78.8 ± 14.1 mm Hg, compared to 69.5 ± 8.8 mm Hg while breathing air (fig. 2, table 2). The average length of nonventilatory periods in hyperoxia was 20.4 ± 4.0 min (N = 3, n = 7), compared with 17.6 ± 3.4 min (N = 5, n = 102) while air-breathing. Breathing 15% and 10% O_2 reduced the nonventilatory period to 9.3 ± 1.2 min (N = 4, n = 21) and 9.3 ± 1.2 min (N = 4, n = 25), respectively. When breathing 2% CO_2 the nonventilatory period (16.9 ± 6.5 min) was not significantly different from that in normoxia (table 2).

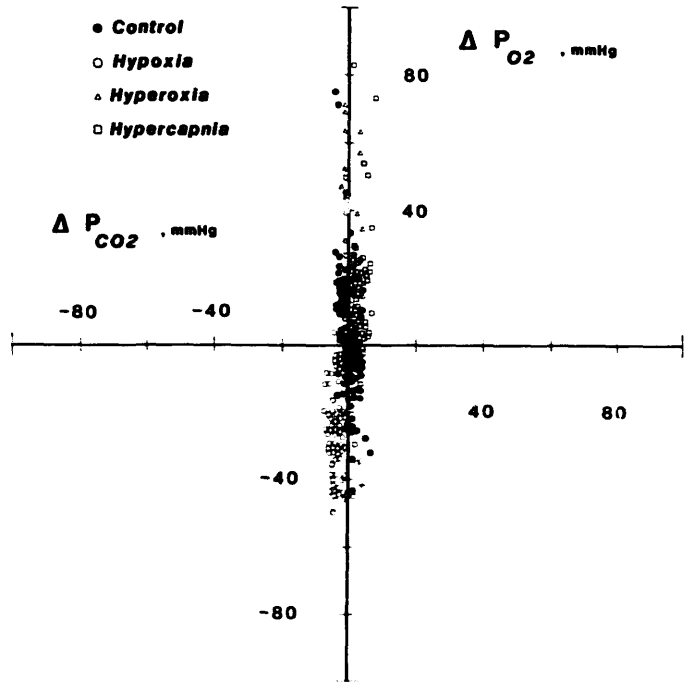

Fig. 2. Distribution of differences in Pa_{O_2} and Pa_{CO_2} at the end of the nonventilatory period from the mean value for each animal. ΔPa_{O_2} and Pa_{CO_2} (in mm Hg) were calculated by subtracting the mean value calculated in each animal from the value achieved at the end of individual nonventilatory periods. Therefore, an x,y coordinate of 5.40 mm Hg on the graph indicates that at the end of the nonventilatory period in question Pa_{CO_2} was 5 mm Hg above, and Pa_{O_2} was 40 mm Hg above the average values in that animal.

Blood gases and the ventilatory variables. The number of breaths in a breathing episode showed a significant positive correlation with the Pa_{CO_2} at the initiation of the episode (fig. 3A). The number of breaths was statistically unrelated to the Pa_{O_2} at the start of the breathing episode (fig. 3B).

Arterial oxygen content and ventilation. Data obtained by *in vitro* analysis of blood samples drawn from six turtles were pooled to generate the oxygen equilibrium curve and table of blood properties illustrated in fig. 4. The P_{50} at a pH of 7.65 was 25.6 mm Hg, and the Bohr shift was quite large at -0.895. The *in vitro* P_{50} and Bohr shift predict that, in normoxic conditions, mean P_{50} at the start of the nonventilatory period was 22.1 mm Hg (pHa = 7.72), and was right-shifted to 25.0 mm Hg by pHa falling to 7.66 by the end of the nonventilatory period. A compensation of Pa_{O_2}'s recorded in normoxia with the curve suggests that blood was fully saturated at the start of the nonventilatory period in animals breathing air (Pa_{O_2} = 92.6 ± 7.4 mm Hg). At the end of the nonventilatory period Pa_{O_2} had fallen to 69.5 ± 8.8 mm Hg, representing a fall in arterial oxygen saturation of only 7%.

TABLE 2

Arterial blood gas and pH values, and the length of the nonventilatory period (NVP) in *Chelydra serpentina* under hyperoxic, normoxic, hypoxic and hypercapnic conditions. Maximum and minimum P_{O_2} and pHa refer to the values recorded at the end of ventilatory periods and nonventilatory periods respectively, and correspond to minimum and maximum Pa_{CO_2} values. Slopes refer to the average rate of change of the variable recorded during nonventilatory periods. SE = standard error of mean, *N* = number of animals, *n* = number of observations.

	P_{O_2}			P_{CO_2}			pHa			NVP min	*N*	*n*
	Max mm Hg	Min mm Hg	Slope mm Hg · min	Min mm Hg	Max mm Hg	Slope mm Hg · min^{-1}	Max U	Min U	Slope U · min^{-1}			
30% O_2	126.6	103.4	−1.5	30.1	39.2	0.5	7.720	7.637	−0.005	20.4	3	7
SE	10.7	10.6	0.3	2.7	1.7	0.1	0.004	0.014	0.001	4.0		
AIR	92.6	69.5	−1.7	32.9	39.2	0.5	7.722	7.661	−0.005	17.6	6	130
SE	7.4	8.8	0.2	2.7	1.5	0.1	0.007	0.014	0.001	3.4		
15% O_2	64.0	46.3	−2.0	31.9	36.7	0.6	7.761	7.712	−0.006	9.3	4	21
SE	6.9	11.5	0.3	3.0	2.9	0.1	0.031	0.028	0.002	1.2		
10% O_2	58.8	49.5	−1.9	30.5	34.5	0.7	7.759	7.717	−0.008	5.5	4	25
SE	7.5	8.5	0.4	2.8	2.2	0.2	0.019	0.024	0.002	0.3		
2% CO_2	97.5	78.8	−1 4	32.6	38.9	0.5	7.726	7.672	−0.004	16.9	3	13
SE	11.4	14.1	0.1	1.9	1.8	0.1	0.017	0.016	0.001	6.5		

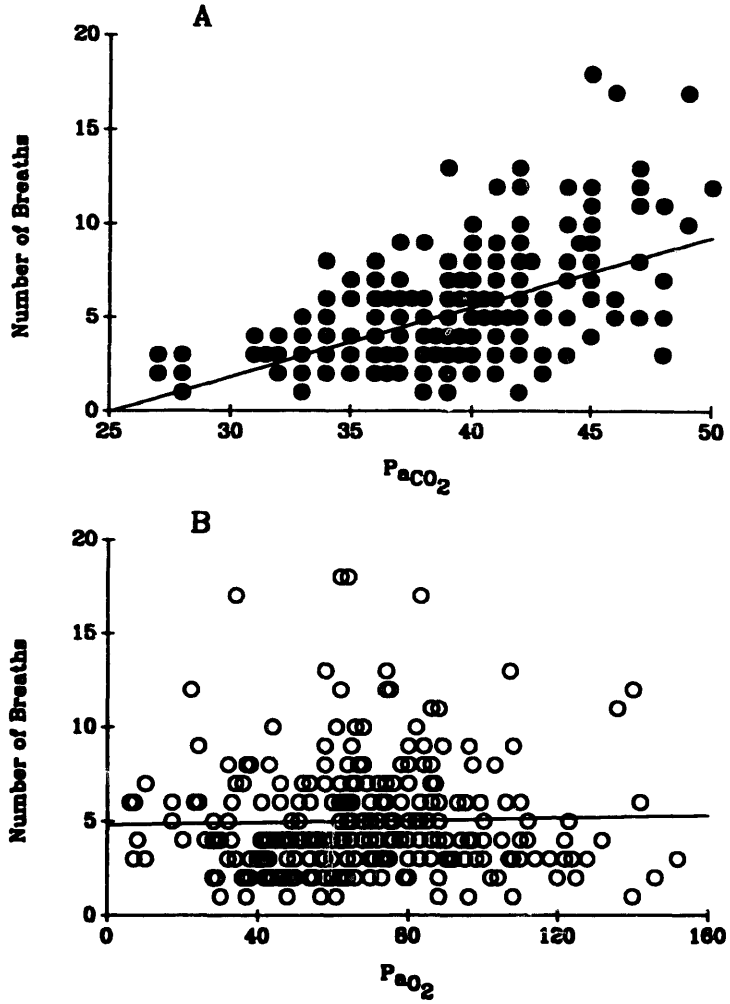

Fig. 3. Number of breaths during the ventilatory period plotted in relation to the Pa_{CO_2} (A), and the Pa_{O_2} (B), at the end of the preceding nonventilatory period. The solid line represents the least-squares regression for each relationship. Only the regression in A (Breaths *vs* Pa_{CO_2}) was significant ($r = 0.57, P < 0.01, n = 280$ breathing episodes).

Discussion

The data we have obtained in the turtle *Chelydra serpentina* strongly indicate that in this species the most important chemical stimulus determining the end of a nonventilatory period in undisturbed, normoxic animals is the Pa_{CO_2} or the corresponding pHa, rather than Pa_{O_2}. This conclusion is based on the following evidence:

1. In normoxia, the maximum excursion of Pa_{CO_2} associated with ventilatory and nonventilatory periods (ca. 6.3 mm Hg) was much smaller than the corresponding excursions in Pa_{O_2} (ca. 23.1 mm Hg), reflecting the greater capacity of blood for CO_2 transport, and the different shapes of the O_2 and CO_2 equilibrium curves. However, Pa_{CO_2} at the end of the nonventilatory periods showed very little scatter from the mean compared with the corresponding Pa_{O_2} values (see fig. 2), suggesting that Pa_{CO_2} repre-

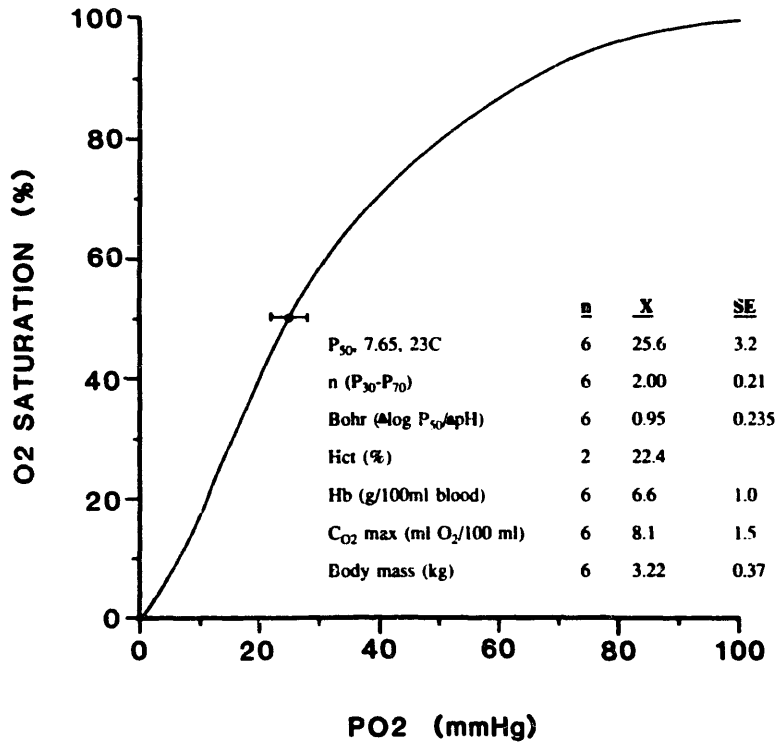

Fig. 4. *In vitro* oxygen equilibrium curve and related characteristics representative of blood in *Chelydra serpentina*. The horizontal bar at the P_{50} represents the standard error of estimates in six turtles.

sents an important controlled variable. The scatter evident in the Pa_{CO_2} indicated that hypercapnic ventilatory drive was not uniform at the end of the nonventilatory periods. This was reflected in the fact that Pa_{CO_2}, but not Pa_{O_2}, showed a significant correlation with the number of breaths in the subsequent breathing episode.

2. In hyperoxia, nonventilatory periods were terminated at a Pa_{CO_2} not significantly different from that in normoxia, in spite of an elevation of mean Pa_{O_2} at the end of the nonventilatory period to a value higher than the maximum postventilation Pa_{O_2} achieved under normoxic conditions.

3. Under hypoxic conditions, mean Pa_{O_2} was markedly depressed at the end of the nonventilatory period but the corresponding Pa_{CO_2} values were only slightly, but significantly reduced, suggesting an interaction between respiratory drive from central and peripheral chemoreceptors under these conditions.

4. The mean Pa_{O_2} achieved by the end of the nonventilatory period in air-breathing, 69.5 ± 8.8 mm Hg, probably does not represent an adequate stimulus for peripheral, hypoxia-sensitive, chemoreceptors.

The presence of central CO_2 sensitivity in turtles (Hitzig and Jackson, 1978) suggests that elevation of cerebral spinal fluid (CSF) Pa_{CO_2} or $[H^+]$ at the end of the nonventilatory period could be the most important stimulus for triggering ventilation. In *Pseudemys scripta*, perfusion of the lateral 4th ventricles with mock CSF of low pH resulted in an increase in ventilation. A reduction in the strong ion difference of 2 meq · L^{-1} resulted in a four-fold increase in the $\dot{V}_E/\dot{V}_{O_2}$ ratio, in spite of a resultant

respiratory alkalosis (Hitzig and Jackson, 1978). Therefore, central chemosensitivity dominated the possible change in input from CO_2-sensitive peripheral receptors such as those described in the dorsal carotid artery of *Testudo hermanni* by Ishii *et al.* (1986).

In normoxic *Chelydra serpentina*, Pa_{CO_2} rose from a mean of 32.9 to 39.2 mm Hg during the nonventilatory period. Assuming free access of CO_2 to the CSF from arterial blood, a CSF osmolarity of 280 mOsm/L and bicarbonate concentration of 32 mM (Hitzig and Jackson, 1978), and using a CO_2 solubility value and carbonic acid dissociation constant calculated from Heisler (1984) and Maas *et al.* (1984) (in Truchot, 1987), this rise is calculated by the Henderson-Hasselbalch equation to represent a fall in CSF pH of 0.08 units, from 7.72 to 7.64 at 23 °C. A fall in CSF of some 0.02 unit was found to increase ventilation in *Pseudemys scripta* approximately four-fold (Hitzig and Jackson, 1978). Therefore, if *Chelydra serpentina* possess central chemoreceptors of similar sensitivity, a powerful stimulus to breathe is present at the end of the nonventilatory period. Under normal circumstances, peripheral CO_2 sensitivity (Ishii *et al.*, 1986), and perhaps the modulation of the tonic firing of CO_2-sensitive pulmonary stretch chemoreceptors by increasing Pa_{CO_2} (Jones and Milsom, 1979), may also contribute to the ventilatory drive.

The shortening of the nonventilatory period while breathing hypoxic gas mixtures (10, 15% O_2) suggests that, at least under these circumstances, receptors sensitive to hypoxia may also contribute to the patterning of ventilation (Boyer, 1963; Glass *et al.*, 1978). In our experiments a reduction in Pa_{O_2} at the end of the nonventilatory period from 69 ± 8.8 to 46.3 ± 11.5 mm Hg by ventilation with 15% O_2, produced a reduction in the length of the period from 16 ± 2.4 min to 9.3 ± 1.2 min, suggesting that the hypoxic threshold lies between some 50–70 mm Hg in *Chelydra serpentina*. A similar shortening of the nonventilatory period was found in hypoxic *Chelys fimbriata*, in which the hypoxic threshold was estimated to correspond to a Pa_{O_2} of approximately 50 mm Hg (Lenfant *et al.*, 1970). Although mean Pa_{O_2} at the end of the nonventilatory period was approximately 70 mm Hg, (60 mm Hg in *Chelys fimbriata*, Lenfant *et al.*, 1970), the wide variability of Pa_{O_2} (22–128 mm Hg) at the end of the nonventilatory period suggests that hypoxia may provide a variable, although minor, contribution to the drive to breathe in undisturbed animals, while perhaps assuming a greater importance during extended breath holding, for example, in the face of a potential threat at the water's surface.

Receptors sensitive to hypoxia are present at several sites in the chelonian cardiovascular system, including the pulmonary artery (Benchetrit *et al.*, 1977), the aorta (Frankel *et al.*, 1969; Ishii *et al.*, 1985), the carotid artery (Ishii *et al.*, 1986) and the truncus (Ishii *et al.*, 1985). No workers have related receptor discharge rates to Pa_{O_2}. It seems reasonable to assume, however, that they may show characteristics similar to central arterial chemoreceptors in the toad *Bufo marinus*, which markedly increase their firing rate at levels of Pa_{O_2} below 50 mm Hg (Van Vliet and West, unpublished data).

The possibility of interaction between the hypoxic and hypercapnic drives terminating the nonventilatory period is suggested by the fact that Pa_{CO_2} at the end of the period was significantly lower when Pa_{O_2} was correspondingly depressed by breathing hypoxic