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Ventilatory Control of Arterial P_{O_2} in the Turtle *Chrysemys picta bellii*: Effects of Temperature and Hypoxia

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Summary. Ventilation, pulmonary O_2 uptake, arterial blood gases and pH were measured in fresh water turtles, *Chrysemys picta bellii,* during voluntary diving and surfacing at temperatures of 10, 20 or 30 °C. At each temperature, the animals were also exposed to declining levels of inspired O_2 concentration with blood samples taken at various stages of breath holding and during episodes of breathing.

The breathing pattern of *Chrysemys* consists of a series of breaths followed by a breath hold period which usually coincides with a period of submergence. The ventilatory response to hypoxia at all temperatures involved a decrease in the diving time as well as an increase in the tidal volume. The breathing frequency during ventilatory periods decreased slightly during severe hypoxia. The increase of ventilation in response to hypoxia was most pronounced at 30 °C; ventilation approximately doubled as arterial P_{O_2} decreased from 60 to 30 Torr and increased more than tenfold as arterial P_{O_2} approached 10 Torr. In comparison, the ventilatory response of animals at lower temperatures occurred at much lower levels of arterial P_{O_2}; at 10 °C ventilation did not increase relative to normoxic control values until arterial P_{O_2} fell to about 5 Torr.

The observed reduction in the ventilatory response to environmental hypoxia at lower temperatures can probably be attributed to the sevenfold reduced pulmonary O_2 uptake at 10 °C as compared to 30 °C in combination with the shift in P_{50} of the blood oxygen dissociation curves from 29 (30 °C) to 5 Torr (10 °C). The present data suggest that desaturation of the blood during hypoxia is a leading factor for the increase in ventilation as an attempt to maintain normal O_2 uptake.

Introduction

Most studies on the control of breathing in reptiles lack any detailed information on arterial blood gas tensions and pH (Wood and Lenfant 1976). This presents a difficulty for both the evaluation and interpretation of the ventilatory responses to low inspired P_{O_2}, because of the considerable P_{O_2} differences which may exist between inspired gas, alveolar gas and arterial blood (Burggren and Shelton 1979).

In contrast to the continuous ventilation of birds and mammals, the reptilian breathing pattern consists of breathing episodes interrupted by breath hold periods of variable duration. In aquatic reptiles the breath hold period usually coincides with diving. Therefore, a complete description of the ventilatory responses of reptiles should include data on breathing time relative to the time spent on breath holding (Glass and Johansen 1979; Milsom and Jones 1980), information which has rarely been reported in conjunction with blood gas data in earlier studies.

On this background, the ventilatory responses to decreases in arterial P_{O_2} were measured in the semi-aquatic freshwater turtle, *Chrysemys picta bellii,* which inhabits portions of the northern United States and southern Canada (Pritchard 1979). Pulmonary ventilation and O_2 uptake were measured along with arterial blood gas tensions and pH. All measurements, including the construction of *in vitro* blood O_2 dissociation curves, were performed at three different body temperatures

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(10, 20 and 30 °C) to encompass the wide thermal range that these animals experience in nature (Ernst 1972).

Materials and Methods

Animals

Specimens of *Chrysemys picta bellii* had been kept in captivity for several months prior to the period of experimentation. All turtles were feeding well and were housed in large glass aquaria equipped with heat lamps and dry areas for basking. Several days (3 days minimum) before measurements, food was withheld and the turtles were acclimated to the experimental temperature. Three independent groups of animals were established as follows: 10 °C, body weight 600±27 g (mean±S.E., $N=6$); 20 °C, 708±23 g ($N=8$); 30 °C, 579±26 g ($N=7$). These turtles were used for measurements of ventilatory responses to hypoxia. In addition, four cannulated turtles were acclimated at each temperature for 2–3 days in order to obtain blood for the determination of *in vitro* O_2 dissociation curves.

Surgical Preparation for Blood Sampling

Blood samples were drawn from chronically implanted catheters. Before surgery, the turtles were anaesthetized by one hour of cold exposure (5 °C) followed by halothane inhalation. A tracheal tube was inserted and connected to a small animal respirator and a halothane evaporator. Muscular relaxation occurred following an initial 5–10 min period of 4% halothane in air, after which the concentration was reduced to 2%.

The cannulation procedure resembled that described by Jackson et al. (1974). A 3.5 cm diameter hole was cut in the plastron to expose the left brachial artery. A P.E. 50 catheter with side holes at the tip was inserted into the artery at a point close to the left leg. Though the cannulation was occlusive, the diameter of the catheter was only about one half of that of the artery, thereby avoiding any serious impedence of blood flow through the numerous side branches coming off the brachial vessel. The catheter was fed out at a point behind the leg and secured to the plastron and carapace with tissue glue and adhesive tape. The excised piece of the plastron was reinserted and fixed in place with dental acrylic. Catheters were flushed with heparinized saline (100 I.U. ml^{-1} in Ringer; de la Lande et al. 1962) and a small dose of tetracycline and chloramphenicol was injected (5 and 10 mg kg^{-1}, i.m.) to prevent postsurgical infections. The turtles recovered from the surgery for at least 24 h at the experimental temperature prior to experimentation.

Measurement of Oxygen Uptake

The experimental set-up is shown in Fig. 1. The turtle moved freely within a thermostatted aquarium, the water surface of which was covered by a grid except at a funnel-shaped breathing hole. The funnel was flushed at a constant flow delivered by a model R 1 flow control pump (Applied Electrochemistry Inc., Sunnyvale, California). The pump was fed gases from the outlet of a Wösthoff gas mixing pump (Bochum FRG), which delivered either room air or hypoxic gases. All gases were thermostatted to the experimental temperature before reaching the inlet port of the experimental chamber. Flowrate through the system (60 to 300 $ml \cdot min^{-1}$) was frequently measured by means of a pneumotachograph (Grenvik et al. 1966). A differential O_2 concentration analyzer (Model S-3A, Applied Electrochemistry, Inc.) monitored the difference in fractional

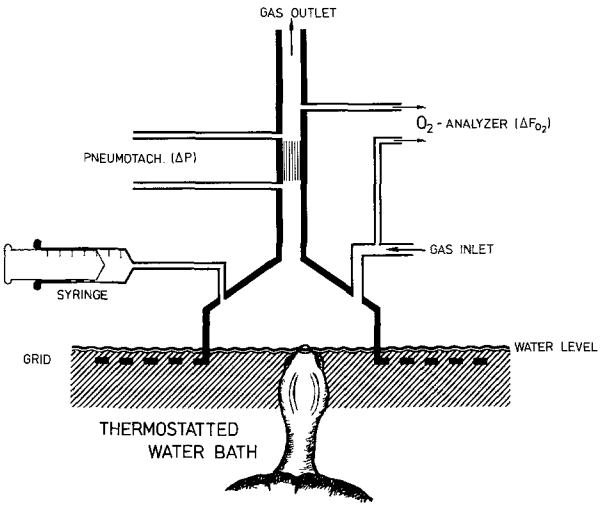

Fig. 1. The experimental set-up. The turtle enters a funnel for breathing. Inhalation and exhalation cause pressure difference across a tube system (pneumotach) at the funnel outlet. The signals are processed to provide tidal volume

O_2 concentration (F_{O_2}) between the inlet and outlet of the funnel (Fig. 1).

Oxygen uptake ($\dot{V}_{O_2}$) was calculated as:

$$\dot{V}_{O_2} = \dot{V}_{STPD} \cdot (FI_{O_2} - FE_{O_2}),$$

where $\dot{V}_{STPD}$ is the flow rate through the funnel corrected to STPD; FI_{O_2} and FE_{O_2} are the fractional O_2 concentrations in inflowing and outflowing gas, respectively. The fractional concentration difference was usually about 0.005 during breathing. For each experimental run and each inspired gas concentration, a mean value for $(FI_{O_2} - FE_{O_2})$ was determined.

Measurement of Ventilation

Ventilation was measured by pneumotachography (Godart pneumotachograph with a OO Godart/Statham transducer probe). The Godart model provides automatic integration of the flow signals for inspired and expired tidal volumes. The zero balance of the pneumotachograph was adjusted to cancel the constant signal resulting from gas flow through the system. Calibration of the pneumotachograph was achieved by withdrawal or injection of known gas volumes using a syringe (Fig. 1).

Experimental Protocol

Upon being placed in the experimental chamber, the turtle soon learned to surface into the breathing hole. The arterial catheter was fed out through the grid, and the experimental chamber was shielded to avoid visual disturbances to the animal during sampling of blood and calibration procedures.

A steady resting baseline ventilation was always recorded within 24 h after placing the animal into the experimental chamber, and this baseline did not change during subsequent days of the test period. Each hypoxic gas mixture ($FI_{O_2} = 0.10$, 0.05, 0.03 or less) was flushed into the breathing hole for 2 to 6 h depending on the body temperature, since steady responses developed faster at higher temperatures. Experiments were discontinued if inhalation of a hypoxic gas caused struggling.

Blood Gas Analysis and O_2 Dissociation Curves

Several blood samples were obtained for each run starting 1 h after the initial introduction of a hypoxic gas. The samples were analyzed for P_{O_2}, P_{CO_2} and pH by electrodes thermostatted to the body temperature of the turtle (Radiometer BMS-systems, Copenhagen, Denmark). The P_{O_2} and P_{CO_2} electrodes were calibrated before and following each measurement with gas mixtures (Wösthoff pumps) which had been humidified at the experimental temperature. The pH electrode was calibrated with Radiometer precision buffer solutions S1500 and S1510. Blood samples used for the analysis of gas tensions were reinjected into the turtle to minimize blood losses.

Oxygen dissociation curves were constructed *in vitro* (at 10, 20 and 30 °C) on blood samples obtained from catheterized animals, which had been resting quietly at the respective preacclimation temperature. The mixing method (Haab et al. 1960) was employed for curve construction and was carried out as detailed by Scheid and Meyer (1978). Desaturated and fully saturated blood samples for mixing were achieved by equilibration in thermostatted tonometers supplied with gas mixtures from Wösthoff gas mixing pumps. At each temperature the CO_2 concentration of equilibration gas mixture was adjusted so that P_{CO_2} of the equilibrated blood was close to the mean *in vivo* value for arterial P_{CO_2}. Each curve was constructed on basis of 10 to 15 different levels of saturation.

Calculations

Calculation of ventilation was based on inspired volume. The resting ventilation baseline for room air breathing was calculated from hours of recording whereas ventilation for hypoxic mixtures was calculated from either 10–20 min sections of a recording or from entire recordings lasting up to several hours, depending on body temperature; i.e. very prolonged measurements were required at 10 °C. It should be emphasized that the analyzed episodes were measured during daytime, because ventilation was decreased at night. The analyzed episodes were taken to represent steady state conditions achieved after at least one hour of exposure to a given gas mixture. Because several blood samples were obtained for each run, the N value is greater for the blood sample data than for the ventilatory variables.

All data were grouped according to inspired oxygen content. Thus, at each temperature, the group with the highest arterial P_{O_2} represents the turtles breathing room air, whereas groups with decreasing arterial P_{O_2} represent $FI_{O_2}=0.10$, 0.05, and 0.03 or less. Experiments on a single turtle were sometimes repeated on different days and have been considered as separate runs in the data treatments.

Plasma bicarbonate concentrations were calculated from the Henderson-Hasselbalch equation using α_{CO_2} and pK'_1 values derived drom Reeves (1976).

Results

The breathing pattern of *Chrysemys* consists of an uninterrupted series of breaths followed by a variable period of breath holding which usually corresponded to a dive (Fig. 2). The duration of the breath hold periods increased with decreases of body temperature, and dives of up to 1 h could occur at 10 °C. Despite the intermittent breathing pattern arterial P_{O_2} was fairly stable at 20 and 30 °C decreasing by about 5 Torr during 1 to 3 min dives. More prolonged dives were often observed

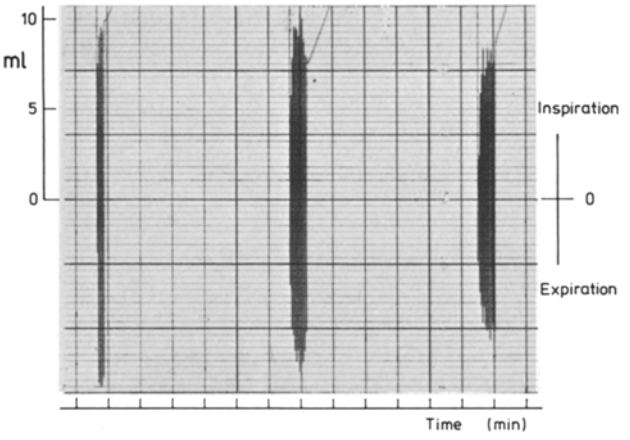

Fig. 2. The breathing pattern of *Chrysemys* consists of breathing episodes interrupted by breath holds (dives). Recording for a 709 g specimen at 20 °C. The reset function of the pneumotachograph only operates during breathing. Therefore, the upward drift during breath-holding represents an artefact

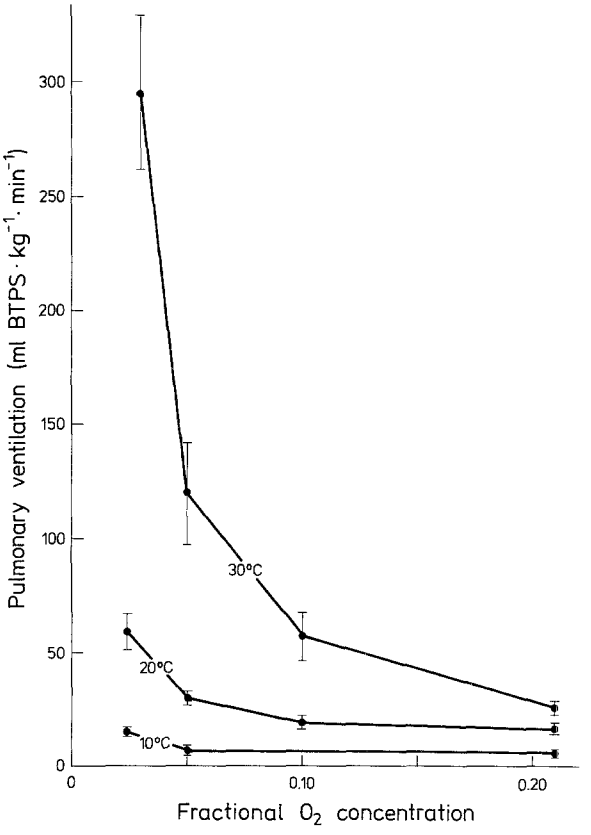

Fig. 3. Effects of hypoxia on pulmonary ventilation at three body temperatures. The data have been grouped according to inspired F_{O_2}. Starting with room air and ending with the most severe hypoxic condition, the N values for ventilation are: 10 °C: 14, 5, 8; 20 °C: 19, 11, 11, 8; 30 °C: 13, 6, 7, 6. For P_{O_2} the corresponding N values are: 10 °C: 38, 17, 20; 20 °C: 27, 19, 17, 8; 30 °C: 19, 11, 11, 9. Mean values (±S.E.) are indicated. The N values are the same for other figures showing effects of hypoxia on ventilatory variables

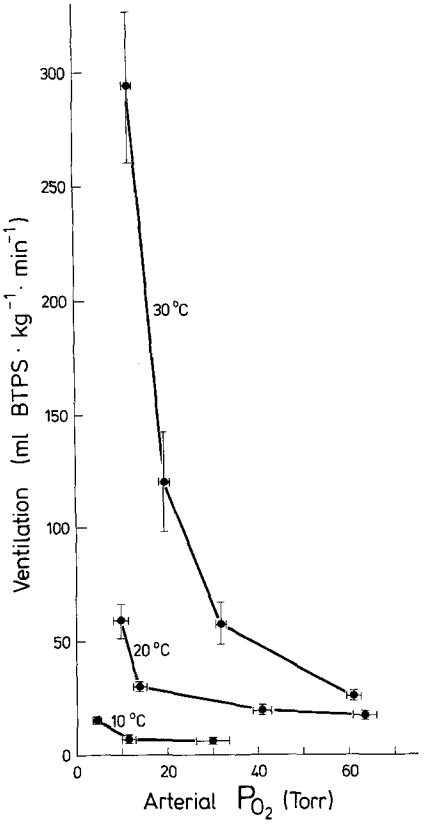

Fig. 4. Effects of hypoxia on pulmonary ventilation in relation to arterial P_{O_2}. The data have been grouped according to inspired F_{O_2} as in Fig. 3, but values for F_{O_2} have been replaced by corresponding values for arterial P_{O_2}

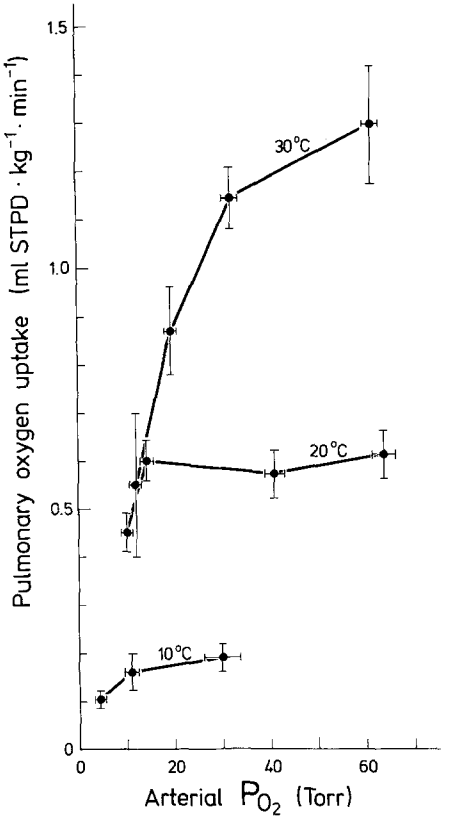

Fig. 5. Oxygen uptake during normoxic and hypoxic conditions at three body temperatures

at 10 °C and these resulted in decreases of arterial P_{O_2} from about 40 Torr during breathing to 10 Torr at the end of 1 h dives. For the most part, however, dives at 10 °C were shorter (about 9 min) and arterial P_{O_2} more stable. For the purpose of the present study, arterial P_{O_2} will be reported as the mean values of measurements made on blood samples taken during breathing as well as diving.

The effect of hypoxia on the frequency of breathing as well as on tidal volume were most pronounced at 30 °C causing large increases in ventilation (Figs. 3 and 4). In contrast, hypoxia had little effect on ventilation at 10 °C. Thus, at 30 °C, ventilation approximately doubled as arterial P_{O_2} decreased from 60 to 30 Torr whereas at 10 °C, ventilation did not increase relative to the control values until arterial P_{O_2} fell to less than 10 Torr (Fig. 4). When the turtle breathed room air, arterial P_{O_2} was only about 30 Torr at 10 °C as compared to about 60 Torr at 30 °C (Fig. 4).

Ventilation increased with temperature as did pulmonary O_2 uptake. The relatively high O_2 uptake at 30 °C was markedly impaired by hypoxia, decreasing to less than 50% of the control value when arterial P_{O_2} was lowered from 60 to 12 Torr. The lower resting O_2 uptake at 10 °C was maintained until arterial P_{O_2} was below 10 Torr (Fig. 5).

The effects of hypoxia on breathing frequency and tidal volumes are shown in Figs. 6 to 8. The duration of breath holds (dives) decreased with body temperature and were also markedly reduced by severe hypoxia (Fig. 5). Even so, breath holding was not entirely abolished even under the most hypoxic conditions. This is further evident in Fig. 6 which shows the ratio of the time spent breathing to total time. It is important to note that, regardless of the body temperature, *Chrysemys* spends less than 10% of its total time breathing at the surface under normoxic conditions. Thus, potentially a tenfold increase in ventilation could be achieved by reduction of breath holds. The decrease of the breath holding time in response to severe hypoxia produced an increase in the overall breathing frequency, i.e. the breathing frequency calculated on basis of longer episodes including breath hold periods as well as periods of ventilation. In addition, inspired tidal volumes increased,

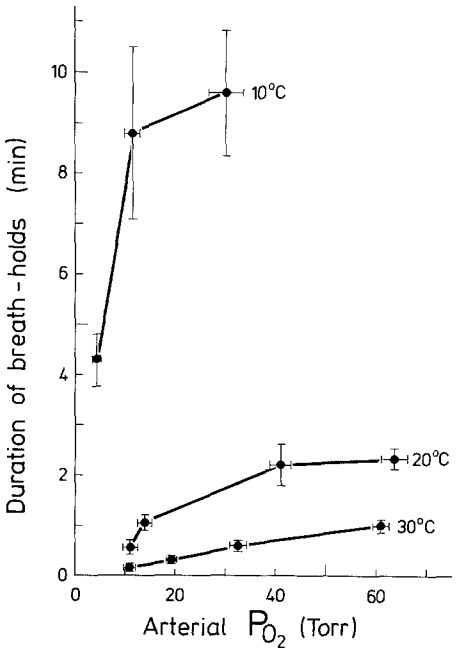

Fig. 6. Effects of hypoxia on breath-holding (diving) time

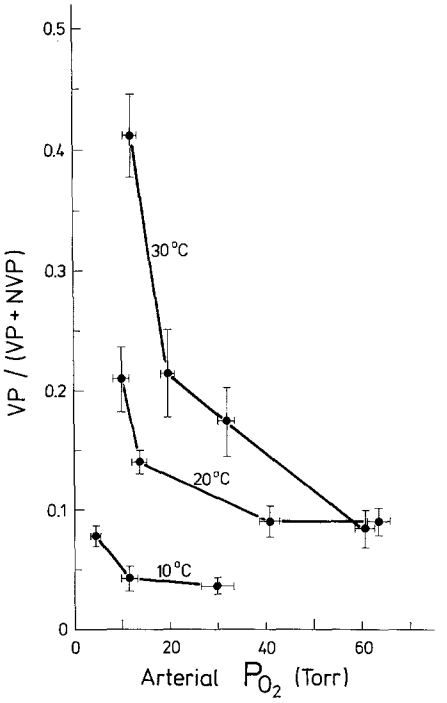

Fig. 7. Effects of hypoxia on the ratio of breathing time to total time, i.e. the diving time as well as breathing time. VP denotes "ventilatory period" and NVP non-ventilatory (diving) period

with the most pronounced changes occurring at 30 °C (Fig. 8).

The breathing frequency within breathing episodes increased slightly with temperature from 16.1 ± 1.7 breaths·min^{-1} (mean $\pm$S.E., $N=14$) to

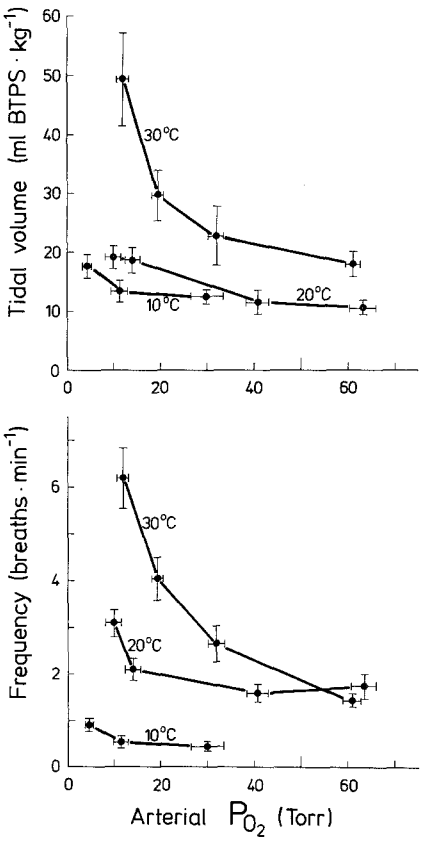

Fig. 8. Effects of hypoxia on overall breathing frequency and on tidal volume

21.3 ± 2.4 breaths·min^{-1} at 30 °C ($N=13$). During severe hypoxia, the frequency decreased by about 20%, probably as a consequence of increases in tidal volume, when more time is required for inspiration and expiration (Milsom and Jones 1980).

The hyperventilation induced by hypoxia caused an increase in arterial pH at 10 and 20 °C. At 30 °C, arterial pH was increased when arterial P_{O_2} was lowered from 60 to 30 Torr, but further decreases of P_{O_2} were accompanied by a progressive fall in pH (Fig. 9). Arterial P_{CO_2} during normoxia was higher at higher temperatures and decreased, as compared to normoxic control values, when hyperventilation occurred during hypoxia. This effect was most pronounced at 30 °C (Fig. 10). Plasma bicarbonate concentrations changed little during hypoxia at 10 and 20 °C, whereas at 30 °C the animals showed a pronounced fall in plasma bicarbonate, resulting in declining arterial pH values in spite of the concomitantly reduced P_{CO_2} (Fig. 11).

The P_{50}-values of *Chrysemys* blood increased with temperature from 5 Torr at 10 °C (pH = 7.96, $P_{CO_2}=17.5$ Torr) to 16 Torr at 20 °C (pH = 7.78, $P_{CO_2}=20$ Torr) and to 28 Torr at 30 °C (pH = 7.63,

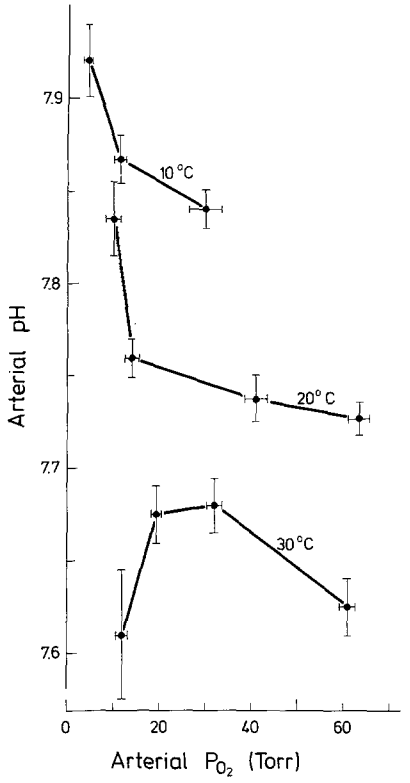

Fig. 9. Effects of hypoxia on pH of arterial blood. The N values for pH are (starting with normoxia and ending with the most severe hypoxic condition): 10 °C: 38, 17, 19; 20 °C: 27, 19, 18, 11; 30 °C: 19, 11, 11, 10. Mean ±S.E.

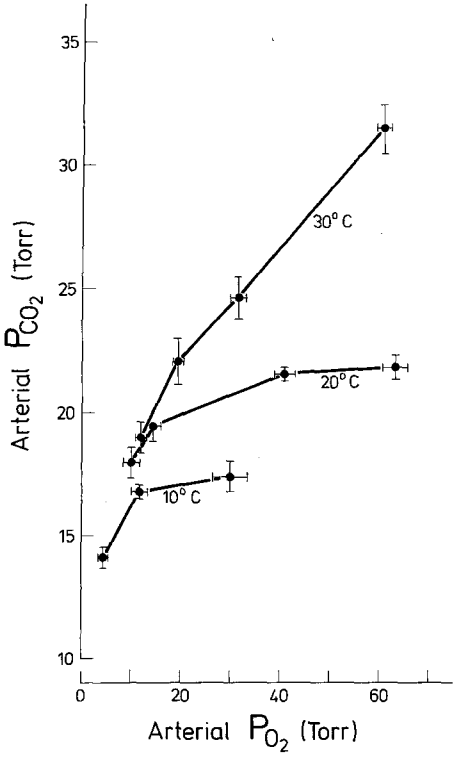

Fig. 10. Effects of hypoxia on arterial P_{CO_2}. N values as for Fig. 9

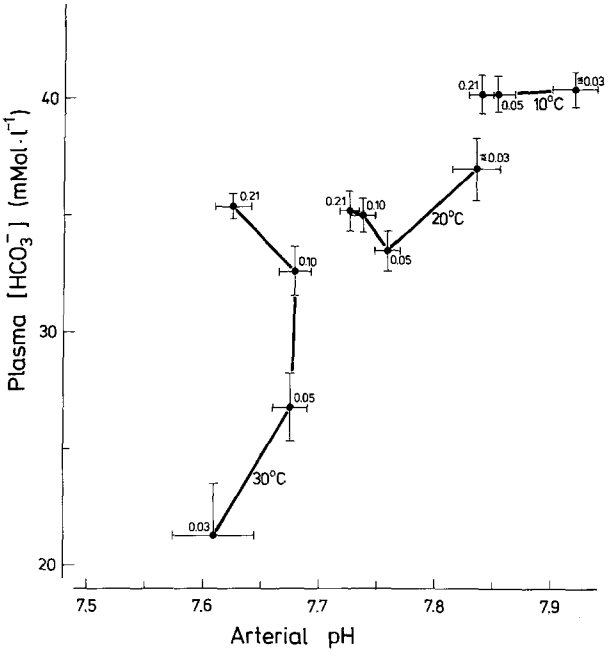

Fig. 11. Relationships between arterial pH and calculated plasma bicarbonate concentrations. N values for pH and also for plasma bicarbonate are equal to N values indicated in the text for Fig. 9. Inspired fractional gas concentrations are shown for each group

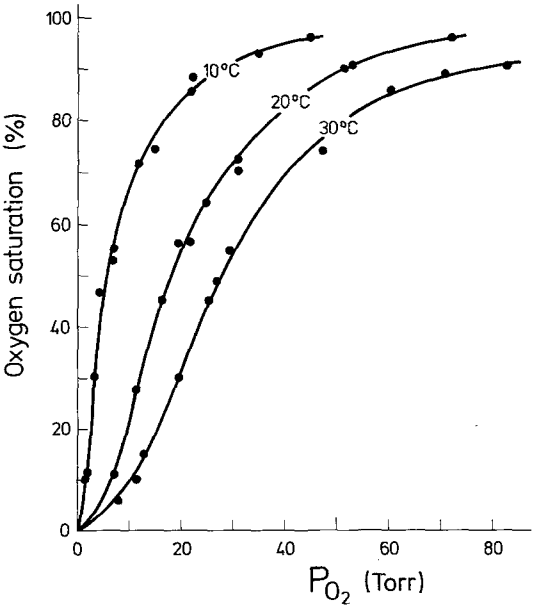

Fig. 12. Oxygen dissociation for *Chrysemys* blood at 10, 20 and 30 °C. Individual points for curve construction are indicated

P_{CO_2} = 29 Torr). Detailed O_2 dissociation curves are shown in Fig. 12.

Discussion

The pronounced temperature-dependence of the ventilatory responses of turtles to low ambient O_2 concentrations have been reported earlier (Jackson 1973; Benchetrit et al. 1977), and the relationships between ventilation and inspired O_2 concentrations in *Chrysemys* are in close agreement with those earlier reported for the fresh water turtle *Pseudemys scripta* (Jackson 1973). With the additional blood gas and pH data of the present study, a more detailed account of the response patterns can be put forth.

Clearly, arterial P_{O_2} in the normoxic 10 °C turtles was shifted to a much lower set of values, relative to both the 20 and 30 °C specimens. In fact, in voluntarily diving turtles at 10 °C, arterial P_{O_2} could decrease to about 10 Torr. These changes in arterial P_{O_2} with temperature are expected on basis of model predictions for the relationships between arterial P_{O_2} and the position of the O_2 dissociation curve of the blood, when the blood is not fully saturated (Rossoff et al. 1980; Wood 1982). The lack of complete saturation of the turtle blood at all temperatures is due to intracardiac shunting of blood with reentry of part of the systemic venous return into the systemic arteries (White 1976).

In a study on the ventilatory responses to hypoxia in the turtle *Pseudemys scripta,* Jackson (1973) made the point that the turtle must defend a greater resting O_2 demand when body temperature increases. Indeed, in *Chrysemys,* the pulmonary O_2 uptake is 6.8 times greater at 30 °C than at 10 °C. Also, the blood at 30 °C is considerably less saturated for a given P_{O_2} than it would be at lower temperatures, because of the increase of P_{50} of the blood with increased temperature. Clearly, both circumstances work against the maintenance of normal O_2 uptake under hypoxic conditions.

These ventilatory responses to hypoxia are compared to the positions of the O_2 dissociation curves of the blood in Fig. 13. The arterial blood of the turtles at 30 °C is only about 15% saturated when the greatest ventilatory response occurs (Pa_{O_2} = 12 Torr), whereas with the same arterial P_{O_2} the blood is still about 70% saturated at 10 °C, and ventilation has changed little relative to the normoxic control value. At all temperatures the turtles increase ventilation in response to a decline in arterial O_2 saturation to about 50%. This may indicate that a receptor system is monitoring O_2 saturation rather than P_{O_2}. This type of control has also been suggested in relation to respiration in humans, but studies on human subjects with abnormally high affinity hemoglobins indicate that arterial P_{O_2} is the regulated variable, because the ventilatory responses of these subjects were normal when arterial P_{O_2} was used as the independent variable, indicating that the high O_2 affinity of the blood has no effect on respiratory control (Hebbel et al. 1977). In addition, the carotid oxygen recep-

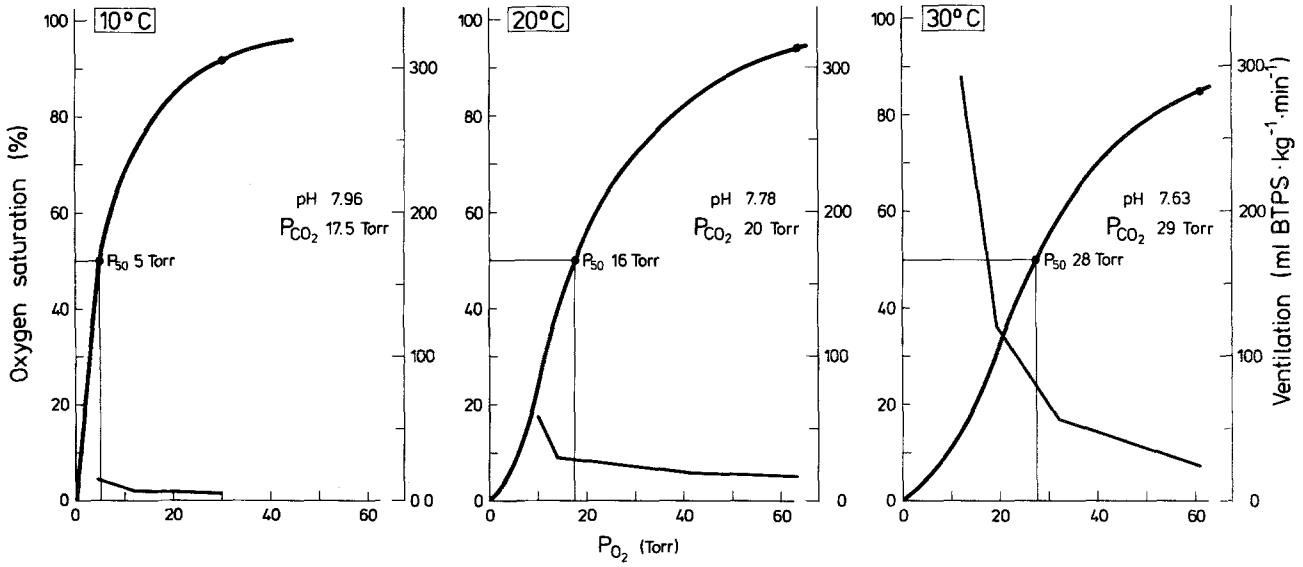

Fig. 13. Ventilatory responses to hypoxia compared to the positions of O_2 dissociation curves for *Chrysemys* blood at 10, 20 and 30 °C. Values for P_{50} and *in vivo* points are indicated. Note that the blood is somewhat desaturated for normoxic turtles at all temperatures. Also note that ventilatory responses occur in hypoxic turtles when arterial P_{O_2} approaches P_{50} of the blood

tors in mammals respond to changes in arterial P_{O_2} and not to changes in O_2 saturation of the blood, although additional aortic receptors in mammals may respond both to decreased P_{O_2} and desaturation of the blood (Lahiri and Gelfand 1981).

The receptors involved in the ventilatory responses to hypoxia in turtles have not yet been identified. Frankel et al. (1969), working on *Pseudemys scripta,* reported an ovoid structure above the bifurcation of the common carotid artery. Despite its inviting location, there was no evidence given for the functional significance. Alternatively, Benchetrit et al. (1977) provided data on the tortoise *Testudo horsfieldi,* suggesting that vagally innervated receptors monitor the P_{O_2} of pulmonary arterial blood. As the O_2 receptors of turtles may be different from those of mammals it is possible that they are monitoring oxygen saturation of the blood. Nevertheless, ventilatory responses to hypoxia of *Chrysemys* cannot be completely explained on basis of a relationship between ventilation and O_2 saturation of the blood. It is a complication that the absolute changes in ventilation were very small at 10 °C. A small ventilatory response to hypoxia may not be a problem in turtles at low temperature, because the low metabolism at these temperatures can be maintained for long periods by utilization of anaerobic stores. For example, *Chrysemys picta bellii* may survive for several months without O_2 when at 3 °C (Jackson and Ultsch 1982). It seems that control of breathing in turtles at low temperature mainly depends on receptor systems monitoring acid-base status (Jackson et al. 1974; Hitzig and Jackson 1978). Thus, in *Pseudemys scripta* at 10 °C Jackson et al. (1973) measured a 3-fold increase in ventilation, when CO_2 inhalation caused a decrease in arterial pH by 0.35 units. Later studies on *Pseudemys scripta* (Hitzig 1977; Hitzig and Jackson 1978) related these responses to the presence of central nervous receptors sensitive to the acid-base status of the cerebrospinal fluid (CSF). Subnormal pH-values of the CSF caused large increases in ventilation and also in the ratio of ventilation to O_2 uptake (air convection requirements). The magnitude of these responses was independent of body temperature if expressed as the change in air convection requirements relative to a given change in pH of the CSF (Hitzig and Jackson 1978). Undoubtedly, the central nervous receptors in turtles are crucial in control of breathing and there is evidence that the information from this receptor system may override the information provided by peripheral receptor systems (Hitzig and Jackson 1978). Nevertheless, the relative roles of central and peripheral receptor systems in turtles need to be further quantified.

The predominant effect of hypoxia on the acid-base status of *Chrysemys* at 10 and 20 °C is a respiratory alkalosis due to hyperventilation. This alkalosis occurs during mild hypoxia at 30 °C, but severe hypoxia is characterized by a fall of pH and in plasma bicarbonate concentrations (Fig. 11). This very likely reflects a depression of O_2 uptake to such an extent that an adequate metabolic rate requires anaerobic production of lactic acid, since the acid-base shift is clearly metabolic in origin. The hyperventilation at 30 °C is presumably aimed not only at acquisition of sufficient oxygen but also towards the removal of excess CO_2 generated through production of lactic acid. In addition, the fall in pH could augment the ventilatory response to hypoxia, due to stimulation of central nervous system receptors.

In many instances, the breathing patterns of reptiles have been regarded as erratic and irregular (Pough 1969; Frankel et al. 1969; Naifeh et al. 1970). This gives the impression that the control of breathing in turtles is inferior to that of birds and mammals. As pointed out by McDonald (1976) and by Jackson (1978), irregularities in the breathing patterns can easily be brought about by disturbances or by the utilization of methods for ventilation measurement that involve restraints and/or unnatural positioning of the animals (see also, Cragg 1978). Our method for measuring ventilation allowed the turtles to move freely, and we can only conclude that the ventilatory responses of *Chrysemys* to hypoxia reveal a flexible but precise control of breathing. The same conclusions were reached by Milsom and Jones (1980) in a detailed analysis of the breathing pattern of *Chrysemys* at 22–23 °C. Moreover, their study and ours are in general agreement as to the duration of breath holds, tidal volumes and other ventilatory variables. Their study also showed that decreases in the duration of the breath hold period are involved in the ventilatory responses to hypercapnia. In turtles, an obvious strategy in increasing ventilation is to step up the amount of time spent breathing, as the breath hold period represents a large reserve for pulmonary gas exchange. Certainly, the reduction in breath hold duration in hypoxic *Chrysemys* is not unprecedented among Chelonian reptiles (*Chelydra:* Boyer 1963; *Pseudemys scripta:* Frankel et al. 1969; *Pelomedusa:* Glass et al. 1978).

In conclusion, increases in ventilation occur when pulmonary O_2 uptake falls below normal resting levels in *Chrysemys*. The response is more

marked as body temperature increases, owing to the greater O_2 uptake and increased potential for acid-base disequilibrium. It seems apparent that, despite decreases in arterial P_{O_2}, ventilation only increases when normal O_2 uptake cannot be readily achieved. An advantage of this strategy is that large changes in arterial pH, due to hyperventilation, can be avoided at least until O_2 uptake becomes endangered. Ventilatory responses to hypoxia occurred when arterial P_{O_2} approached P_{50} of the blood, which decreased from 29 Torr at 30 °C to 5 Torr at 10 °C. The increases in ventilarion during hypoxia were small at 10 °C, whereas increases were more than 10-fold at 30 °C.

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