J. exp. Biol. (1982), **96**, 295–306
With 3 figures
Printed in Great Britain

RESPIRATION AND ACID-BASE PHYSIOLOGY OF THE SPOTTED GAR, A BIMODAL BREATHER

III. RESPONSE TO A TRANSFER FROM FRESH WATER TO 50% SEA WATER, AND CONTROL OF VENTILATION

BY NEAL J. SMATRESK* AND JAMES N. CAMERON

Departments of Zoology and Marine Studies, The University of Texas at Austin, Port Aransas Marine Laboratory, Port Aransas, Texas 78373, *U.S.A.*

(*Received* 1 *April* 1981)

SUMMARY

Transfer from fresh water to 50% sea water (SW) at 26 °C increased the blood osmolarity of spotted gar (*Lepisosteus oculatus*) from 275 to 310 mosmol during the first 24 h. It then returned slowly to freshwater levels by 5 days after the transfer. The arterial pH dropped sharply, from 7·69 in fresh water to 7·46 in 50% SW, as a result of a small elevation in the blood CO_2 partial pressure, and a marked metabolic acidosis. The respiratory (CO_2) portion of the acidosis appeared to be a result of the reduction in branchial ventilation, and possibly permeability as well. The metabolic portion of the acidosis was not due to the accumulation of lactic acid, but probably involved a disruption of the extracellular strong ion difference in the saltier medium. The metabolic acidosis did not diminish during 5 days.

The rate of air breathing rose from 7 to 20 bph during 50% SW exposure. The control of pulmonary ventilation was directly responsive to the availability of O_2, in general increasing when O_2 was limiting (e.g. 50% SW transfer, hypoxia) and decreasing in hyperoxia. CO_2 had no affect on the rate of air breathing. Withdrawal from 5–20% of total lung volume elicited an immediate air breath during hypoxia, but the response was inconsistent in normally aerated water. Lung inflation with O_2 prolonged the interval between air breaths, but inflation with N_2 did not change the rate of air breathing. Thus, pulmonary ventilation was secondarily controlled by lung volume. Gill ventilation frequency fell in 50% SW, despite a respiratory and metabolic acidosis, while gill ventilation increased in response to treatment with acetazolamide. Hyperoxia caused a marked depression of gill ventilation, despite a respiratory acidosis. The gill ventilation rate appears to be most closely linked to oxygen, but may be affected indirectly by CO_2 through the Root or Bohr effects.

* Present address: University of Pennsylvania, School of Medicine, Department of Physiology, Philadelphia, PA 19104, U.S.A.

INTRODUCTION

Some teleost fish can maintain relatively constant internal osmotic pressures in aquatic environments ranging from fresh water to sea water, (Maetz, 1971). The transport mechanisms in gills which are responsible for the regulation have received considerable attention (Maetz, 1971). Most of these studies have focused on ion fluxes and exchange mechanisms; there have been comparatively few studies on the respiratory and acid-base responses to hyperosmotic environments (Milne & Randall, 1976; Bath & Eddy, 1979).

Transfer from a hypo- to a hyper-osmotic environment upsets the osmotic and ionic balance of a freshwater fish. The entry of salts in a hyperosmotic environment occurs primarily across the gills and gut. A water-breathing fish has conflicting demands to meet in such a situation: the demand for oxygen increases in a hyperosmotic environment (Rao, 1971) so the fish must maintain or increase ventilation and perfusion of the gills. The gills, however, are a major site for passive ion influxes, and it would be advantageous to reduce the branchial ventilation or perfusion to minimize changes in the internal osmotic pressure. A water breather will have to compromise between the demands for oxygen uptake and the need to maintain osmotic balance until branchial ion exchanges are able to handle the increased salt load. An air-breathing fish, on the other hand, may be able to 'shut down' gill ventilation or perfusion and use its lungs for gas exchange to maintain osmotic balance. This strategy would help osmoregulation, but might adversely affect acid-base regulation. If the gills were 'shut down' the animal could encounter a respiratory acidosis.

Both the alligator gar and the Florida spotted gar are bimodal breathers that are frequently seen in marine environments. Juvenile Florida spotted gar survived for at least 2–3 weeks in 79% prior to experimentation in work by Zawodny (1975). Zawodny concluded that while blood osmolarity rose from about 220 to 340 mosmol the gar survived by reducing their unidirection water efflux and by actively extruding Na^+ and Cl^-.

In addition to determining the ventilatory responses to dilute sea water, we studied the effects of hyperoxia, acetazolamide and changes in lung gas concentrations and volume on ventilation. The combined effects these treatments had an acid-base balance and oxygen supply to the gar, along with previous work (Smatresk & Cameron, 1982*a*, *b*) allowed us to generally describe the control of pulmonary and branchial ventilation in spotted gar (*L. oculatus*).

METHODS

50% *sea-water transfer*

Sampling cannulae were implanted in both dorsal and ventral aortae, as described in a preceding paper (Smatresk & Cameron, 1982*a*), and subsequently allowed to recover for at least 24 h in a flow-through chamber. The temperature for all the experiments was 26 ± 1 °C. Two constant-volume reservoirs were employed to provide a flow of either fresh water or 50% sea water (550 mosmol) to the fish chambers. After the

recovery period in fresh water (FW), resting determinations of pH, P_{CO_2}, C_T, and P_{O_2} were made as described earlier. The air-breathing intervals and gill ventilation frequencies were measured using an opercular catheter connected to a pressure transducer and recorder. The blood lactate concentration was measured enzymatically (Sigma 826-UV kit) using 200 μl plasma samples. The osmolarity of the plasma was measured using duplicate samples in a vapour-pressure osmometer (Wescor). All measurements were repeated after the transfer to sea water (SW) at intervals of 1, 2, 4, 8, 24, 48, 72, 96 and 120 h, using 11 fish.

Acetazolamide treatment

Four fish were treated with acetazolamide (Diamox, Lederle Labs.), which inhibits carbonic anhydrase activity, and elicits a respiratory acidosis (Holder *et al.* 1955; Maren, 1962). The injections were made in order to achieve an *in vivo* concentration of $0{\cdot}5 \times 10^{-5}$ g/g wet weight via a catheter implanted in the dorsal aorta. Blood values for P_{O_2}, P_{CO_2}, and pH were then taken several times between 4 and 7 h after the injections. Gill ventilation and air-breathing frequencies were also recorded during this interval via opercular catheters. The fish were held in darkened plexiglass chambers provided with an air space for breathing and a flow of dechlorinated tap water (at 27 °C). Surgical methods were described by Smatresk & Cameron (1982*a*).

Hyperoxia

Four fish were observed in hyperoxia (water $P_{O_2} \simeq 500$ torr, temperature 26 ± 1 °C). These data, although few, are included in this section because the responses were pronounced and similar among the fish, and because the observations help to clarify ventilatory control mechanisms. Blood was analysed for P_{O_2}, P_{CO_2}, pH, and C_T from the dorsal aorta in the fish (five measurements total). An opercular catheter was used to measure gill ventilation, and air-breathing rates.

Lung-gas Manipulation

In normoxia and hypoxia, lung-gas concentrations and volumes were manipulated. By utilizing two lung catheters (PE-50) implanted in the lung through the pneumatic duct, gases could be added to the lung while maintaining a constant lung volume. In both normoxia and hypoxia the gar's responses to lung deflation (withdrawal of from 2 to 30 ml of lung gas), lung inflation (addition of gas until the animal floated at the surface of the water), elevated lung P_{CO_2}, and elevated or depressed lung P_{O_2} were studied. Lung P_{CO_2} was elevated by adding humidified CO_2 (from 10 to 50 ml) while simultaneously withdrawing an equal volume of gas from the second catheter. Lung P_{O_2} was changed by adding 100% humidified O_2, or 100% humidified N_2 while maintaining lung volume with the other catheter, elevating the P_{O_2} in the lung as high as 600 torr, and lowering it to between 10–20 torr. The effects of these changes were also analysed as decreasing, increasing or producing no change in the resting air breathing frequency.

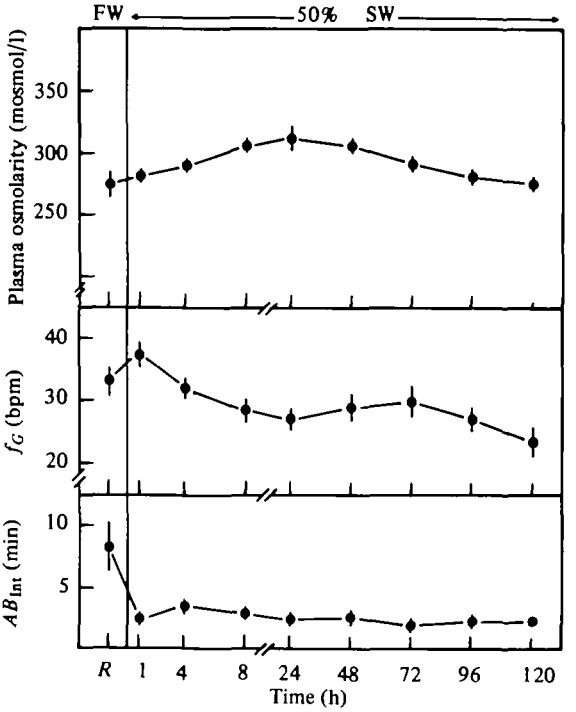

Fig. 1. The time course of changes in gill ventilation (f_G), plasma osmolarity and the air breathing interval (AB_{Int}) upon transfer from freshwater (fw) to 50% sea water (sw).

RESULTS

50% sea-water transfer

Ventilation

The changes in the air-breathing interval and the ventilation frequency for resting fish in FW, and at several time intervals after the transfer to 50% SW (1/2 SW) are shown in Fig. 1 (N = 8, 16, where 8 = number of fish and 16 = total number of measurements). The 1/2 SW breathing intervals were significantly shorter than the FW intervals at all sample periods (*t* test, $P < 0.05$), and the ventilation frequency was significantly depressed 24 h after the transfer ($P < 0.05$).

Blood osmolarity

The blood osmolarity increased temporarily (Fig. 1), and was significantly different from FW values 24 h following transfer. Over the next 4 days the blood osmolarity gradually returned to the control value. The number of determinations at each time interval was five fish, ten measurements, except that at 4 h eight measurements were made from four fish.

Acid-base balance

Following the exposure to 1/2 SW there were marked changes in the blood acid-base balance (Fig. 2). Both the pH and C_T fell after 1/2 SW exposure, while the

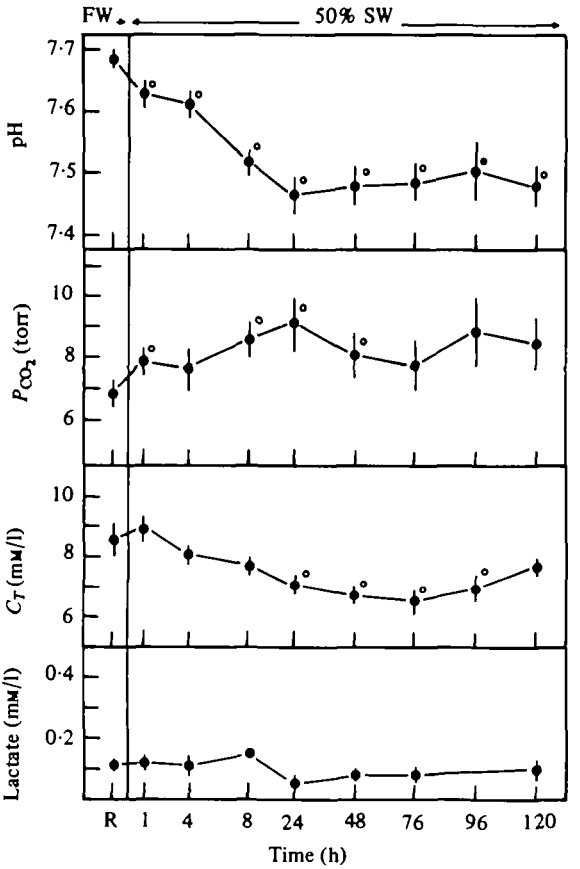

Fig. 2. The time course of blood changes in pH, P_{CO_2}, C_T, and lactate upon transfer from fresh water (fw) to 50 % sea water. An open circle following a point indicates that the point is significantly different from the fresh water value.

P_{CO_2} was elevated. This acidotic condition was not compensated in 5 days' exposure, and two fish studied were still acidotic after 12 days' exposure. Analysis of the acid-base variables on a pH-HCO_3^- (Davenport) diagram (Fig. 3) showed that the acidosis had both a respiratory and a metabolic component, but the metabolic component was clearly not due to lactic acid (Fig. 2), since its concentration did not change.

The *in vivo* pK calculated for the blood in fresh water was 6·236, using means of pH, C_T and P_{CO_2}, mean osmolarity of 275 mosmol/l, and temperature of 26 °C. Exposure to 1/2 SW produced an insignificant change in the calculated pK to 6·229.

Ventilation

Acetazolamide

Treatment with acetazolamide (Table 1) produced a marked respiratory acidosis in all fish tested and arterial P_{O_2} was elevated slightly but not significantly. The gill ventilation frequency rose significantly (t test, $P < 0{\cdot}05$) and the rate of air breathing more than doubled (significant, t test, $P < 0{\cdot}05$).

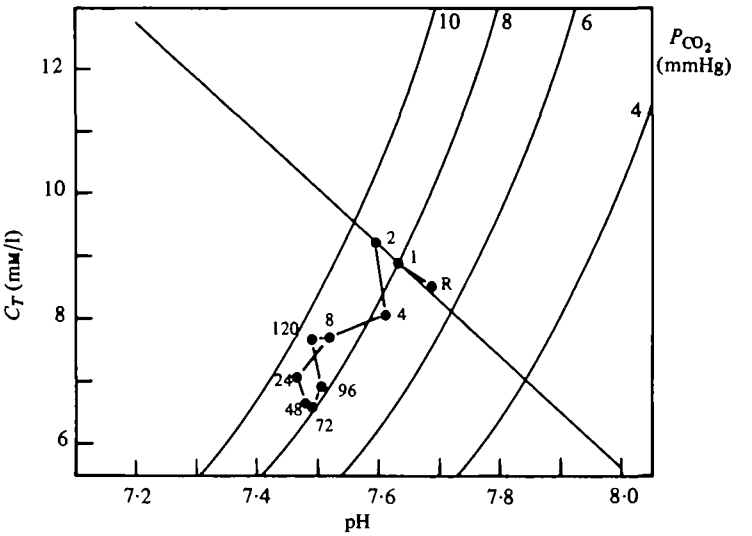

Fig. 3. Davenport diagram of the simultaneous changes in pH, P_{CO_2}, and C_T in fresh water (R) and at several time intervals after transfer to 50% sea water (time given in hours).

Table 1. *The effects of acetazolamide on ventilation and blood variables*

(Values shown are mean ± S.E., and in parenthesis are given either the number of fish or both the number of fish, and the total number of measurement. Arterial total CO_2 (C_T) was calculated using the Henderson–Hesselbalch equation, an aW_2 of 0·428 and a pK calculated from gar whole blood (*in vivo*) of 6·236 at 26 °C.

	Control	Acetazolamide
Gill ventilation (bpm)	33·8 ± 1·9 (N = 11)	46·3 ± 11·3 (N = 4, 16)
Air breathing interval (m)	7·8 ± 1·7 (N = 11)	3·7 ± 1·2 (N = 4, 16)
Arterial P_{O_2} (torr)	29·3 ± 1·3 (N = 4, 27)	33·3 ± 1·9 (N = 4, 27)
Arterial P_{CO_2} (torr)	8·1 ± 0·2 (N = 4, 27)	14·2 ± 2·4 (N = 4, 27)
Arterial pH	7·60 ± 0·00 (N = 4, 27)	7·49 ± 0·00 (N = 4, 27)
Arterial C_T (mM·l^{-1})	8·0 (calculated)	11·5 (calculated)

Hyperoxia

Hyperoxia completely inhibited air breathing in all four fish tested, while the gill ventilation rate dropped significantly (paired t test, $P < 0.05$) from 32·5 ± 1·5 in normoxia to 23·2 ± 1·5 bpm in hyperoxia. In one fish arterial P_{O_2} rose from 22 to 39, P_{CO_2} rose from about 6 to 16, pH fell from 7·76 to 7·53 and C_T rose from 10·6 to 18·0. In the other fish from which blood samples were obtained arterial P_{O_2} changed from 25 to 32·5, P_{CO_2} from 6·5 to 14·5, pH from 7·72 to 7·50 and C_T from 10 to 17.

Lung gas manipulation

When fish were in hypoxic water removal of between 5 and 20 ml of gas (5–20% of total lung volume in a 1 kg fish) (N = 8) would elicit an immediate air breath. In normoxia (N = 8), four fish did not respond to lung deflation and the other fish required as much as 40 ml of gas removal before taking an air breath. This extreme

Table 2. *Summary of the responses of the gar to various treatments*

The data for hypoxia, temperature change and hypercapnia were reported in the previous papers (Smatresk & Cameron, this issue). The symbols f_G and f_L refer to the frequency of gill and lung ventilation, C_{a,O_2} to the arterial blood oxygen content.

Stimulus	f_G	f_L	C_{a,O_2}	Respiratory acidosis	Metabolic acidosis
Hypoxia	−	+	−	No	No
Hyperoxia	−	−	+	Yes	No
Hypercapnia	+	+?	−?	Yes	Alkalosis
1/2 sea water	−	+	−	Slight	Yes
Acetazolamide	+	+	−?	Yes	No
Activity	+	+	−	Yes	Yes
30 °C, Normoxic	−	+	−	No	No

+, An increase, −, a decrease relative to 20 °C, Normoxic.

deflation also caused the animals to sink, and struggling often ensued. When the lung was filled with O_2 and kept inflated, air breathing ceased. When the lung was inflated with N_2, air breathing continued unabated. Changes in the CO_2 of the lung did not affect pulmonary ventilation.

A summary of the responses of both gill and lung ventilation to treatments reported above and results reported in previous papers (Smatresk & Cameron, 1982*a*, *b*) is given in Table 2. The activity studies were made along with other studies, and reflect periodic episodes at struggling or spontaneous activity. These studies were done at different temperatures, and so the results have been normalized (except for the temperature elevation to 30 °C) in terms of changes made from 20 °C, normoxic and normocapnic conditions.

DISCUSSION

50% sea-water transfer

The salt challenge represented by the transfer from fresh water to 50% sea water appears to have been met by a complex series of responses involving ventilation of gills and lungs, changes in blood electrolytes, and changes in blood gas and acid-base parameters. Most of these responses appear to be interrelated.

Since the gills are the primary site of passive salt influx, the reduction of gill ventilation appears to be adaptive in terms of reducing the osmotic load presented to the gills. The obvious effects on gas transfer are partially overcome by the corresponding increase in the ventilation of the lung (Fig. 1), but there is still a slight decline in the arterial oxygen content. The reduction in gill ventilation is not entirely effective, however, and the blood osmolarity undergoes a transient rise lasting several days, and probably remains significantly different in ion ratio from the freshwater fish (Zawodny, 1975). It may be these changes in ion ratios, perhaps related to differential ion permeability, that result in the permanent depression of the blood pH in 1/2 sea water relative to fresh water. That is, a prime independent variable in the acid-base equilibrium in blood is the 'strong ion difference' (Stewart, 1978), so the pH set-point of the blood is strongly dependent on the ionic control mechanisms.

Ventilatory response

The ventilatory reduction, during 1/2 SW exposure, may have caused the small respiratory (CO_2) acidosis of 1–2 torr we observed (Figs. 2, 3). The reduction of ventilation may, at least in part, account for the reduction of branchial permeability observed by Zawodny (1975) (i.e. a reduction in the unidirectional water efflux of from 17·4 in FW to 4·8 ml.100 $g^{-1}.h^{-1}$ in 75% SW).

The perfusion of the gills by blood may have also been reduced or altered, but we had no way of estimating any such changes in this study. Bath & Eddy (1979) have reported a reduction of oxygen consumption and a reduction in the oxygen transfer factor across the gills in trout after SW exposure. They also observed that gill surface area was actually reduced, perhaps by dehydration of the branchial epithelium. The trout sacrificed oxygen consumption to a certain extent to reduce permeability, whereas the gar compensated its branchial reduction in oxygen consumption by increasing the frequency of air breathing, and so, unlike the trout, their oxygen consumption was probably maintained.

Blood acid-base balance

The branchial 'shut down' and the ensuing respiratory acidosis were not altogether surprising in view of Zawodny's earlier (1975) work that showed a reduction in gill permeability following SW transfer. What was not particularly expected was the prolonged metabolic acidosis, as shown in the Davenport diagram (Fig. 3). It does not appear that this component of the acidosis was truly 'metabolic' in the sense of arising from acidic metabolites, especially of anaerobic metabolism, since lactate was not elevated (Fig. 2). There is every indication that the animals remained fully aerobic by balancing the branchial reduction with increased air breathing. The 'metabolic' component is more likely the result of differential changes in the permeability to various strong ions (i.e. completely dissociated electrolytes) which are a principal determinant of the acid-base status (Stewart, 1978). Although we did not measure specific ions in this study, Zawodny (1975) found that in the Florida spotted gar, blood chloride concentration increased from 97 to 177 mequiv·l^{-1} after transfer to 75% SW, while sodium rose only from 122 to 164 mequiv·l^{-1}. Thus, there is a surprising change in the difference from 122–97 = 25 to 164–177 = −13! Of course the difference between sodium and chloride does not constitute the entire strong ion difference, but this change was probably indicative of a reduction in strong ion difference, especially since there was no change in potassium, and is completely consistent with the idea of 'metabolic' acidosis resulting from ion imbalance. The mechanism by which this change in strong ion difference could come about must remain a matter for speculation, but it probably involves both differential changes in passive (influx) permeability in the hyperosmotic environments, and changes in the rates of electroneutral ion exchanges, such as Cl-for-HCO_3 and Na-for-NH_4 (or H^+). There may also be shifts of ions between the intra- and extra-cellular fluid compartments that contribute.

The extracellular pH was still depressed 5 days after the change to 1/2 SW, but the HCO_3^- concentration may have shown slight compensation, since it was not significantly different from FW after 5 days of 1/2 SW. Trout do not immediately

demonstrate a significant acidosis when transferred to SW (Bath & Eddy, 1979; Milne & Randall, 1976), but do become acidotic after several days' exposure. The trout are clearly not able to shut down their gills as completely as a gar can, since they have no auxiliary means of oxygen supply, and so a smaller change in P_{CO_2} is not surprising. The blood HCO_3^- concentration in trout was also reduced (Bath & Eddy, 1979). This relationship between pH and salinity has also been observed in crabs (Mangum *et al.* 1976).

Osmotic balance

The early increase and later regulation of blood osmolarity indicated that osmoregulation upon transfer to 1/2 SW was not entirely handled by a reduction of gill permeability, or at least as it is affected by ventilation. Drinking probably accounted for a portion of the elevation in blood salts (Zawodny, 1975). The time course of 4 or 5 days for return to normal is similar to that observed in trout (Houston, 1959). This slow time-course suggests that slower mechanisms, like increases in the numbers of active transport sites on the gills, may be responsible for the slow restoration of osmotic balance (Thomson & Sargent, 1977).

Ventilation

Lung ventilation

The delivery of O_2 to the tissues was threatened during exposure to 1/2 SW, and was probably compromised by the severe respiratory acidosis produced by acetazolamide. These, and the other stimuli that reduced the blood O_2 content; aquatic hypoxia, activity and temperature elevation (see Table 2) all stimulated pulmonary ventilation (Table 2). Increasing the availability of O_2 via aquatic hyperoxia inhibited air breathing. *Amia* (Johansen, Hanson & Lenfant, 1970), *Neoceratodus* (Johansen, Lenfant & Grigg, 1967), *Saccobranchus* (Hughes & Singh, 1971) and *Lepisosteus osseus* (Crawford, 1971), all faculatative air breathers, respond similarly to hypoxia by increasing their breathing rates.

CO_2, on the other hand, had no consistent effect on pulmonary ventilation. During hyperoxia the blood P_{CO_2} was elevated, but the rate of air breathing was depressed. When the fish were subjected to aquatic hypercapnia the air breathing rate did not change significantly. It is likely that the gar's pulmonary ventilation does not directly respond to P_{CO_2}, but P_{CO_2} and pH may indirectly modulate air breathing rates through their effects on blood O_2 via the Bohr and Root effects (Smatresk & Cameron, 1981*a*).

Changes in lung gases and lung volume

Reduction of the lung volume stimulated an air breath in hypoxia but did so less predictably in normoxia. The ease with which the response can be elicited in hypoxia suggests that the sensitivity of the response is increased at low P_{O_2} concentrations. The rapidity of the response, though, indicates that there are pulmonary stretch receptors. *Amia* has also been shown to respond rapidly to lung deflation (Johansen *et al.* 1970) by air breathing. These receptors may be of great importance in changing the air-breathing rate. The low lung R value (R = 0·10) will cause lung volume to fall

during the course of an air breath, as O_2 is absorbed without equal amounts of CO_2 excreted. As pulmonary perfusion and lung $\dot{M}_{O_2}$ increase, the lung will deflate more rapidly, stimulating the receptors, and thereby an air breath. Air breathing is not wholly dependent on these receptors, though, because keeping the lung fully inflated with N_2 does not inhibit air breathing. The stretch receptors may also be useful in helping the lung to maintain buoyancy, which is another important function of the gar's air bladder. As the gar consumes O_2 from the lung it becomes less buoyant; by taking an air breath, the gar maintains neutral buoyancy.

Pulmonary ventilation was generally unresponsive to acute changes in lung gas tension, which suggests that there are probably no peripherally located pulmonary chemoreceptors. Inflation of the lung with O_2 did inhibit the rate of air breathing, but this may have been due to the combined effects of inhibiting the stretch reflex and increasing O_2 delivery. Filling the lung with N_2, on the other hand, did not stimulate air breathing in normoxic or hypoxic water, as has been seen in *Electrophorus* (Johansen *et al.* 1968*b*) and *Trichogaster* (Burgren, 1979).

Gill ventilation

Water-breathing fish virtually always increase gill ventilation as water P_{O_2} declines (Shelton, 1970). The gar, though, showed an optimal gill-ventilation rate in normoxic water and ventilation declined during both hyperoxia and hypoxia (Table 2). *Amia* (Johansen *et al.* 1970) and *Lepisosteus osseus* (Crawford, 1971) also demonstrated maximal gill ventilation rates at normoxic P_{O_2} levels. This complex ventilatory behaviour to changing O_2 tensions suggests that central integration of chemoreceptor responses from several loci determines the gill ventilation rate.

There is a fair amount of evidence for peripheral loci for O_2-sensitive chemoreceptors in water-breathing fish (Randall & Smith, 1967; Laurent & Rouzeau, 1969; Daxbeck & Holeton, 1978; Smith & Jones, 1978). The rapid response of gill ventilation to hyperoxia, along with the reduction of gill ventilation during hypoxia (despite the maintenance of blood P_{O_2} by air breathing) is indicative of external P_{O_2} receptors in gar as well.

As was true for lung ventilation, CO_2 did not have a consistent effect on gill ventilation (Table 2). Both hyperoxia and sea-water transfer produced a respiratory acidosis, and depressed gill ventilation. The acidosis caused by acetazolamide and hypercapnia (Smatresk & Cameron, 1982*b*), however, stimulated ventilation slightly. It would appear that CO_2 was not by itself an important stimulus to gill ventilation, and that its effects were either indirect, via changes in blood O_2 content, or were subordinate to other stimuli (e.g. P_{O_2} or osmolarity).

The branchial response of air breathing fish to changes in external P_{CO_2} levels has not been well documented. Hughes & Singh (1971) found that hypercapnia (2% CO_2) stimulated branchial ventilation, but very high CO_2 may have inhibited it in *Saccobranchus*. *Neoceratodus* exhibited an inhibition of branchial ventilation upon exposure to 2% CO_2 in water (Johansen *et al.* 1967) and *Amia* elevated branchial respiration at concentrations up to 3% CO_2. The obligate air breathers *Protopterus* and *Synbranchus* both reduce gill ventilation in response to any hypercapnic stimulus (Johansen, 1970; Johansen & Lenfant, 1968). *Electrophorus* (Johansen, 1970), another obligate air

breather, does not respond to aquatic hypercapnia. The quick response of most of these air breathers to hypercapnia caused Johansen (1970) to hypothesize that they have peripheral chemoreceptors sensitive to pH or P_{CO_2}. Rapid responses to aquatic P_{CO_2} were not observed in the gar, which supports the contention that the effects of CO_2 (or pH) may be indirect.

The variable behaviour of gill ventilation to the various stimuli makes it difficult to characterize its control. The gar, unlike water-breathing fish, is not dependent on its gills for meeting its O_2 demands, giving the branchial ventilatory control system the potential for more flexibility in responding to environmental changes. This was particularly evident during the exposure to 1/2 SW when external P_{O_2} remained constant, but gill ventilation decreased. The reduction might have helped the gar to osmoregulate more easily by reducing branchial ion influx and water efflux rates. In this case the regulation of gill ventilation is probably more subservient to the needs of osmoregulation than to O_2 demand. Thus, interactions of branchial ventilation with other control mechanisms could allow the gar to make the most efficient use of its gill not only for O_2 transport (or retention) but for osmotic, ionic or acid-base regulation.

This research was supported in part by an NSF grant, PCM77-24358, to J.N.C.

REFERENCES

BATH, R. N. & EDDY, F. B. (1979). Ionic and respiratory regulation in rainbow trout during rapid transfer to seawater. *J. comp. Physiol.* **134**, 351–357.

BURGGREN, W. W. (1979). Bimodal gas exchange during variation in environmental oxygen and carbon dioxide in the air breathing fish *Trichogaster trichopterus*. *J. exp. Biol.* **82**, 197–213.

CRAWFORD, R. H. (1971). Aquatic and aerial respiration in the bowfin, longnose gar and Alaska blackfish. Ph.D. dissertation, University of Toronto.

DAXBOECK, C. & HOLETON, G. F. (1978). Oxygen receptors in the rainbow trout, *Salmo gairdneri*. *Can. J. Zool.* **56**, 1254–1259.

HOLDER, J., HEINEMANN, H. O., FISHMAN, A. P. & SMITH, H. W. (1955). Urine pH and carbonic anhydrase activity in the marine dogfish. *Am. J. Physiol.* **183**, 155–162.

HOUSTON, A. H. (1959). Osmoregulatory adaptation of steelhead trout to seawater. *Can. J. Zool.* **37**, 729–748.

HUGHES, G. M. & SINGH, B. N. (1971). Gas exchange with air and water in an air breathing catfish, *Saccobranchus* (*Heteropneustes*) *fossilis*. *J. exp. Biol.* **55**, 667–682.

JOHANSEN, K. (1970). Air breathing in fishes. In *Fish Physiology*, vol. IV (ed. W. S. Hoar and D. J. Randall), New York: Academic Press.

JOHANSEN, K., HANSON, D. & LENFANT, C. (1970). Respiration in a primitive air breather, *Amia calva*. *Resp. Physiol.* **9**, 162–174.

JOHANSEN, K., LENFANT, C. & GRIGG, G. F. (1967). Respiratory control in the lungfish, *Neoceratodus forsteri* (Krefft). *Comp. Biochem. Physiol.* **20**, 835–854.

JOHANSEN, K. & LENFANT, C. (1968). Respiration in the African lungfish. II. Control of Breathing. *J. exp. Biol.* **49**, 453–468.

JOHANSEN, K., LENFANT, C., SCHMIDT-NIELSEN, K. & PETERSON, J. A. (1968). Gas exchange and control of breathing in the electric eel, *Electrophorus electricus*. *Z. vergl. Physiol.* **61**, 137–163.

LAURENT, P. & ROUZEAU, J. D. (1969). Afferent neural activity from the pseudobranch of teleosts. *Resp. Physiol.* **14**, 307–331.

MAETZ, J. (1971). Fish gills: mechanisms of salt transfer in freshwater and seawater. *Phil. Trans. R. Soc. Lond.* B **262**, 209–249.

MANGUM, C. P., SILVERTHRONE, S. U., HARRIS, J. L., TOWLE, D. W. & KRALL, A. R. (1976). The relationship between blood pH, ammonia excretion and adaptation to low salinity in the blue crab, *Callinectes sapidus*. *J. exp. Zool.* **199**, 129–126.

MAREN, T. H. (1962). Ionic composition of cerebrospinal fluid and aqueous humor of the dogfish, *Squalus acanthias*. II. Carbonic anhydrase activity and inhibition. *Comp. Biochem. Physiol.* **5**, 201–215.

MILNE, R. S. & RANDALL, D. J. (1976). Regulation of arterial pH during freshwater to seawater transfer in *Salmo gairdneri*. *Comp. Biochem. Physiol.* **53** A, 157–160.

RANDALL, D. J. & SMITH, J. C. (1967). The regulation of cardiac activity in a hypoxic environment. *Physiol. Zool.* **40**, 104–113.

RAO, M. M. G. (1971). Influence of salinity and activity on the weight-dependent oxygen consumption of rainbow trout. *Mar. Biol.* **205**, 205–212.

SHELTON, G. S. (1970). The regulation of breathing. In *Fish Physiology*, vol. IV (ed. W. S. Hoar and D. J. Randall). New York: Academic Press.

SMATRESK, N. J. & CAMERON, J. N. (1982*a*). Respiration and acid-base physiology of the spotted gar, a bimodal breather. I. Normal values, and the response to severe hypoxia. *J. exp. Biol.* **96**, 263–280.

SMATRESK, N. J. & CAMERSON, J. N. (1982*b*). Respiration and acid-base physiology of the spotted gar, a bimodal breather. II. Responses to temperature change and hypercapnia. *J. exp. Biol.* **96**, 281–293.

SMITH, F. M. & JONES, D. R. (1978). Localization of receptors causing hypoxic bradycardia in trout (*Salmo gairdneri*). *Can. J. Zool.* **56**, 1260–1265.

STEWART, P. A. (1978). Independent and dependent variables of acid-base control. *Resp. Physiol.* **33**, 9–26.

THOMSON, A. J. & SARGENT, J. R. (1977). Changes in the levels of chloride cells and $(Na^+ + K^+)$-dependent ATPase in the gills of yellow and silver eels adapting to sea water. *J. exp. Zool.* **200**, 33–40.

ZAWODNY, J. F. (1975). Osmoregulation in the Florida spotted gar, *Lepisosteus platyrhincus*. M.Sc. Thesis, University of Miami.