Respiratory and Cardiovascular Adjustments to Exercise in Reptiles

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1 Introduction

An important aspect of the reptilian response to exercise is a heavy reliance on anaerobic energy production to supplement aerobic metabolism during even modest activity. The aerobic and anaerobic capacities of reptiles, and the implications of these capacities for vigorous activity, have been recently discussed (Bennett and Dawson 1976; Bennett 1978, 1980). Like other terrestrial vertebrates, reptiles have been shown to increment the rate of oxygen consumption in proportion to the intensity of locomotory exercise (Fig. 1a; Moberly 1968a; Chodrow and Taylor 1973; Prange 1976; Bennett

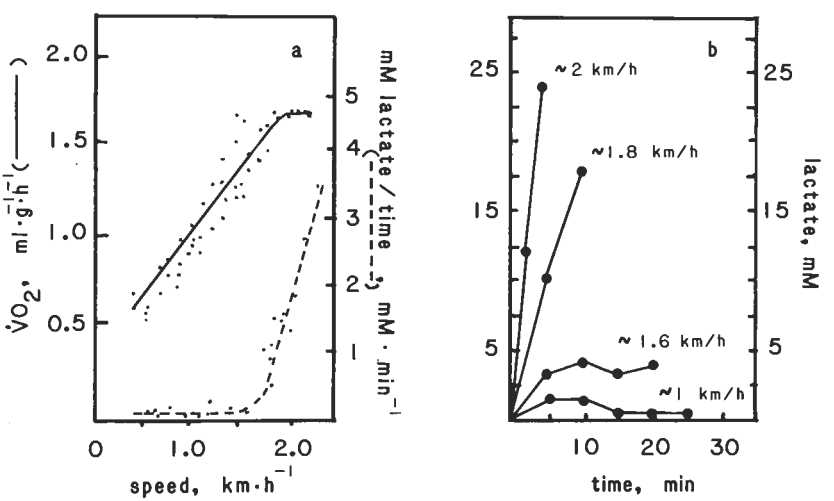

Fig. 1a,b. Oxygen consumption and lactate production in *V. exanthematicus* during exercise. **a** Rate of oxygen consumption (*solid line*) and rate of lactate production (*broken line*) as a function of treadmill running speed. **b** Change in blood lactate concentration with time at different exercise intensities. VO_{2max} in this 700 g lizard was reached at approx. 1.8 km h^{-1}. Redrawn from Seeherman et al. 1983

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and Gleeson 1979; Gleeson 1979, 1981; Gleeson et al. 1980; Butler et al. 1984). Although both reptiles and mammals are capable of incrementing oxygen consumption five- to tenfold during exercise, predictive equations suggest that the maximum aerobic scope of a 100 g reptile is only 8% that of a comparable-sized mammal (Bennett and Dawson 1976; Taylor et al. 1981). The significance of this is that reptiles are capable of supporting only modest levels of activity aerobically. A 1 kg lizard will reach maximal rates of oxygen consumption ($\dot{V}O_{2\,max}$) at running speeds as low as 0.5–1.0 km h^{-1} (Fig. 1a), while maximal aerobically-supported running speed for a 1 kg mammal would be 6–10 km h^{-1} (Garland 1982). This limited aerobic capacity of reptiles means that nearly all vigorous activities have an anaerobic component to their energy production. While some species may be capable of utilizing nearly their entire aerobic scope prior to supplementation with lactate production (Mitchell et al. 1981a), others experience lactate accumulations at nearly all levels of activity (Fig. 1b). The mechanism for anaerobic energy production during exercise is highly developed in this vertebrate group. Lactate accumulations of 20–30 m*M* following brief activity are common, and blood concentrations in excess of 50 m*M*, with blood pH falling as low as 6.6, have been reported to occur in crocodiles trying to escape capture (Bennett et al. 1985). Adjustments to metabolic acidosis of this magnitude are a major aspect of the reptilian response to exercise.

Physiological responses to activities having both aerobic and anaerobic components range from biochemical changes at the cellular level to changes in organismal gas exchange. We have chosen to highlight the changes that occur in respiratory and cardiovascular function during sustainable and burst activity. The data available on these subjects are mostly derived from observations on lizards and turtles. There are few data available on other groups of reptiles, and one of the goals of this review is to stimulate research of the activity physiology of these animals.

2 Ventilatory Adjustments to Exercise

2.1 Introduction

Exercise presents three challenges to the respiratory system of reptiles. Incrementing aerobic metabolism above resting levels results in increased rates of CO_2 production, and this CO_2 must be excreted by increasing ventilation in proportion to this elevated rate to maintain acid-base balance. A second challenge is that of increasing ventilation sufficiently to maintain a favorable gradient for oxygen diffusion to arterial blood. Most lizards are capable of maintaining arterial oxygen content during exercise, although they generally do not maintain the high levels of saturation typical of mammals. The third challenge is maintaining acid-base balance in the face of hydrogen ion produced as an end product of anaerobic metabolism. Buffering this hydrogen ion production is the role of the bicarbonate buffer system. Reptilian respiratory systems are surprisingly efficient at meeting these challenges despite simple lung morphologies (Tenney and Tenney 1970; Perry and Duncker 1978; Perry 1983). Readers are referred to Wood and Lenfant (1976) for a review of reptilian respiratory mechanics, ventilatory characteristics of resting animals, and the influence of temperature on ventilation. The data

on ventilatory function in active animals are much less extensive. We will describe the general features of reptilian ventilatory responses to sustainable and nonsustainable exercise. A working definition of sustainable exercise is that requiring rates of VO_2 less than $VO_{2\,max}$ which does not result in continual lactate accumulation. Such exercise is illustrated by the lower curves (speeds ≤ 1.6 km h^{-1}) in Fig. 1b. Rapid and continual lactic acid production has been reported at running speeds requiring 50%–95% $VO_{2\,max}$, the variation due to differences among species and experimental protocols (Bennett and Gleeson 1979; Gleeson 1980; Mitchell et al. 1981a; Seeherman et al. 1983). This division between sustainable and nonsustainable exercise allows independent focus on the maintenance of blood gases during carbonic acid loads (CO_2 production), on the one hand, and on acid-base regulation during noncarbonic acid loads (lactic acid production), on the other.

2.2 Ventilation During Sustainable Exercise

Rates of CO_2 and O_2 exchange increase five- to ninefold during sustainable exercise, and ventilation usually has an even greater factorial increment. For example, minute ventilation increases tenfold in sea turtles during terrestrial exercise, although VO_2 and VCO_2 increase only three- and sevenfold, respectively (Jackson and Prange 1979). This increase in ventilation is due entirely to increases in frequency, as tidal volume does not change. Such adjustments maintain blood gases and acid-base balance surprisingly well, given that sea turtles are unable to locomote and ventilate at the same time. This stability is even more remarkable in swimming *Chelonia*, which surface and take but one breath, whereas when walking they pause and take several. Yet, swimming turtles are also able to increase ventilation sufficiently to regulate blood gases within the range found at rest (Butler et al. 1984). Similar data on ventilatory frequency and volumes are not available for other reptiles during sustainable exercise.

An indirect assessment of ventilatory changes during exercise is provided by changes in the effective lung ventilation (V_{eff}). This calculated variable is analogous to rate of alveolar ventilation in mammals, and estimates the functional rate of lung ventilation (Mitchell et al. 1981a). Lung ventilation increases 10- to 15-fold in three lizard species during sustainable exercise (Mitchell et al. 1981a; Gleeson and Bennett 1982), but the data do not indicate how changes in frequency and tidal volume accomplish these increases. In *Varanus exanthematicus*, lung ventilation overcompensates for the metabolic demand for increased gas exchange across the lung; the factorial increase in V_{eff} exceeds the factorial increments in both VO_2 and in VCO_2 (Fig. 2a). The slight hyperventilation relative to VCO_2 results in a 5–7 torr depression in arterial and presumably also lung P_{CO_2} (Fig. 2b). Thus, sustainable exercise can cause a mild respiratory alkalosis in this species. Alkalosis occurs during such exercise in *Varanus*, but lactate production by *Iguana* early in exercise counters a respiratory alkalosis and arterial pH is maintained (Mitchell et al. 1981a).

All species of lizards studied are able to maintain Pa_{O_2} at or above resting levels throughout the period of exercise (Mitchell et al. 1981b; Gleeson and Bennett 1982). Increasing the rate of lung ventilation (V_{eff}) relative to the rate of oxygen consumption during sustainable exercise (Fig. 2a) increases lung P_{O_2} as the metabolic demand for oxygen increases (Fig. 2b). Thus, the gradient for pulmonary oxygen transport is

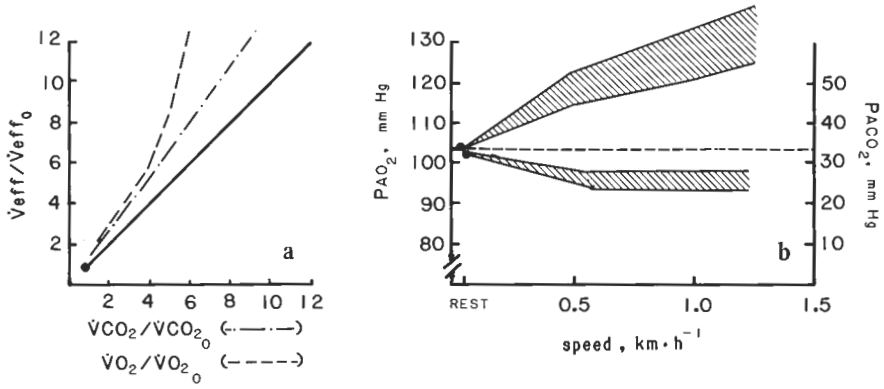

Fig. 2a,b. Changes in ventilation and lung gases during exercise in *V. exanthematicus*. a (left) Changes in ventilation relative to changes in gas exchange. Changes in lung ventilation from rest are expressed as a ratio (V_{eff}/V_{eff_0}), as are changes in VCO_2 and VO_2. Ventilatory change relative to VO_2, *dashed line*; relative to change in VCO_2, *broken line*. *Solid line* is line of identity, *area above line* represents hyperventilation. **b** (right) Changes in lung gas partial pressures during sustainable exercise, *upper region* reflects change in lung oxygen partial pressure; *lower region*, lung CO_2 partial pressure. Data calculated from Mitchell et al. 1981a,b

enhanced and maintains Pa_{O_2} and arterial O_2 content at or slightly above resting levels during exercise (Gleeson et al. 1980; Mitchell et al. 1981b). Any potential limitation in pulmonary oxygen transport during exercise due to insufficient or inefficient exchange surfaces is, therefore, offset by an increase in the lung-arterial P_{O_2} gradient (Mitchell et al. 1981b). An increase in lung-arterial P_{O_2} gradients is contrary to changes seen in man (Whipp and Wasserman 1969). The large gradient in resting lizards is likely due to cardiac shunts, which are known to exist in resting animals (Berger and Heisler 1977). A constant or increasing right-to-left cardiac shunt cannot explain the increasing pulmonary diffusion gradient for oxygen during exercise, however, as shunting venous blood of reduced oxygen content during exercise would result in a lower Pa_{O_2}, which does not occur (Figs. 2b and 3). The data suggest that in *Varanus* and *Iguana*, a decreasing right-left shung may occur as VO_2 increases during exercise (discussed further in Sect. 3). An alternative explanation for the increased gradient during exercise is provided by Mitchell et al. (1981b), who suggest that it might compensate for a limited lung surface area that otherwise would become limiting to oxygen uptake during exercise.

The ventilatory adjustments that occur during sustainable exercise are sufficient to maintain arterial blood gases and pH close to resting levels. Mitchell et al. (1981a) found that after prolonged exercise on a treadmill, running lizards possessed Pa_{CO_2} tensions slightly lower than at rest, hydrogen ion concentrations slightly more alkalotic than during rest, and no blood lactate accumulation. In a study which detailed the transient changes that occur during sustainable exercise, Gleeson and Bennett (1982) obtained a more complete picture of blood gas regulation during sustainable exercise. Arterial blood of the water monitor *Varanus salvator* was sampled at intermediate periods during treadmill running lasting 45 min at 85% $VO_{2\,max}$. Under these conditions, arterial oxygen tensions were maintained at a slightly elevated level relative to rest for

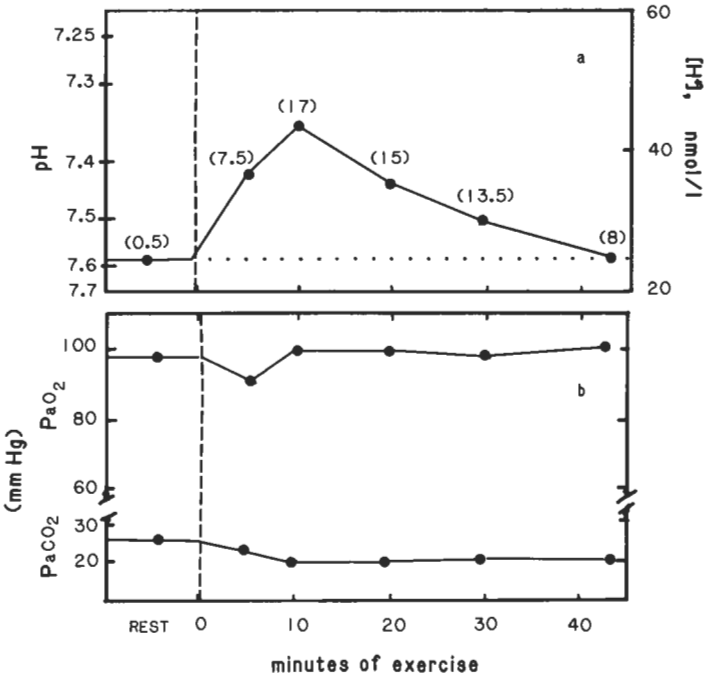

Fig. 3a,b. Blood gas and pH changes during exercise at 85% VO_{2max} in *V. salvator*. **a** Changes in arterial pH, with mmol l^{-1} changes in arterial lactate indicated in *parentheses*. **b** Arterial P_{CO_2} and P_{O_2} during exercise. Redrawn from Gleeson and Bennett 1982

all but the first few minutes of exercise (Fig. 3). The transient depression in Pa_{O_2} may indicate that the increased minute ventilation mentioned above may require a few minutes to be established and doesn't track VO_2 kinetics. Supplementary anaerobic metabolism during the initial minutes of exercise resulted in lactate accumulations of 18 m*M* and 0.2 U depression of pH after 10 min of running. Arterial P_{CO_2} declined approx. 8 torr as V_{eff}/VCO_2 increased 50%. As exercise continued, however, the metabolic acidosis was corrected by both metabolic and respiratory mechanisms. The elevated lactate levels were reduced metabolically by either oxidative or gluconeogenic mechanisms, both of which require equimolar quantities of hydrogen ions. Plasma bicarbonate levels, depressed as a result of the earlier metabolic acidosis, also begin to return towards resting concentrations. Late in the exercise period, arterial pH returns to normal (Fig. 3) and may even be slightly alkalotic, while Pa_{O_2} and Pa_{CO_2} change little relative to rest. The hyperventilation which occurs during such exercise persists during recovery in several species and results in significant alkalosis (Gleeson and Bennett 1982). The ability of *Varanus* to correct for the transient metabolic acidosis while exercising at such a high percentage of its VO_{2max} clearly indicates that ventilatory adjustments during sustainable exercise are more than adequate for excretion of the CO_2 resulting from increased rates of aerobic metabolism.

2.3 Ventilation During Exhaustive Exercise

Reptiles are noted for their ability to undergo brief bursts of exercise of very high intensity. Lizards, for example, are capable of running for a short time at speeds equal to the sprint speeds of similar-sized mammals (Garland 1985). Such exhaustive exercise is dominated by a metabolic acidosis resulting from lactate production. Large amounts of nonmetabolic CO_2 produced as the bicarbonate system buffers the fixed acid. Metabolic CO_2 production may be of secondary importance, as fatigue may occur even before maximal rates of aerobic metabolism are reached (Gleeson and Bennett 1982).

Exhaustive exercise elicits an immediate increase in lung ventilation. Minute ventilation increases five- to sixfold during such exercise in lizards and snakes (Dmi'el 1972; Bennett 1973a; Wilson 1971). Effective lung ventilation (V_{eff}) during 5 min of treadmill running increases to rates approaching 20 times standard rates in the lizard *Varanus salvator*. Increases in minute ventilation that occur during maximal exercise in lizards are due almost entirely to increases in tidal volume, as ventilatory frequency changes little if at all (Bennett 1973a). This pattern is in contrast to the response seen in sea turtles to sustainable exercise (Prange and Jackson 1976; Jackson and Prange 1979). Data on the ventilatory responses by snakes to exercise are less clear, but it appears that both frequency and tidal volume increase to some degree (Dmi'el 1972).

Both lizards and snakes hyperventilate relative to their aerobic demands for gas exchange during vigorous exercise. In the lizard *V. salvator*, ratios of V_{eff} to VO_2 indicate that this species increases ventilation threefold relative to increases in oxygen consumption. Such is also the case for other lizards and snakes (Dmi'el 1972; Wilson 1971). Ventilation is sufficient to maintain oxygenation of arterial blood, and thus, prevent an even greater reliance on anaerobic metabolism during exercise. During exhaustive exercise, saurian blood Pa_{O_2} remains constant or may rise slightly (Mitchell et al. 1981b; Gleeson and Bennett 1982) and arterial saturation may decline 10% or less (Bennett 1973b). However, ventilation in turtles is not sufficient to maintain oxygen saturation of arterial blood (Gatten 1975). During 2 min exercise at 30 °C the red eared turtle *Pseudemys scripta* experiences a fall in arterial blood saturation from 90% at rest to 34%. A similar decrement occurs in the box turtle *Terrepene ornata*. Failure to replenish arterial oxygen stores reinforces reliance of these turtles on anaerobic metabolism and probably limits their ability to sustain vigorous activity. Hypoventilation or apnea during exercise appears not to be the cause of this decreased arterial oxygen. *Pseudemys* apparently increase ventilation sufficiently to excrete the CO_2 necessary to allow the bicarbonate buffering system to maintain arterial pH precisely as blood lactate increases 6 m*M* (Gatten 1975), and it is unlikely that this regulation could be accomplished without hyperventilation. An increased shunt fraction during exhaustive exercise could explain these data.

The metabolic acidosis that occurs during exhaustive exercise makes carbon dioxide excretion of central importance for maintenance of acid-base balance. CO_2 is derived from both tissue respiration and from biocarbonate buffering of lactic acid, which frequently increases 15–30 m*M* in a few minutes. The resultant high rates of CO_2 excretion result in respiratory exchange ratios ($R_e = VCO_2/VO_2$) that peak at values of 2.0 or greater in some species (Bennett and Gleeson 1979; Gleeson 1980; Gleeson and Bennett 1982). Respiratory exchange ratios remain elevated at levels greater than 1.0

for a considerable portion of the recovery period, long after arterial lactate concentrations ([L]a) have peaked. Continued production of high levels of CO_2 is presumably due to washout of dissolved CO_2 from body tissues as arterial blood with a lower P_{CO_2} flows through it (Gleeson and Bennett 1982). The available data suggest that R_e can be used as a noninvasive indicator of the period of [L]a accumulation in lizards; decreasing values of R_e are indicative that [L]a has peaked and is declining, even though R_e may still be greater than 1.0 (Gleeson and Bennett 1982).

The magnitude of the metabolic acidosis during and after exhaustive exercise can be considerable. Lactate accumulations in snakes and lizards of 20–30 m*M* are not unusual (Bennett and Dawson 1976; Ruben 1976; Gleeson 1982). Crocodiles attempting to avoid human capture elevate [L]a more than 50 m*M*, the highest concentration ever reported in an active animal (Bennett et al. 1985). Such accumulations are very disruptive to tissue acid-base balance; blood pH in lizards may decline from around 7.5 to 7.0 (Gleeson and Bennett 1982), and crocodile blood pH may drop to 6.6 (Bennett et al. 1985; Seymour et al. 1985).

Table 1 summarizes the changes that occur in several reptiles in response to this acidosis. Most reptiles become hypocapnic during or shortly after exhaustive exercise, the result of an hyperventilation relative to VCO_2. Bicarbonate concentrations become depressed in roughly equimolar amounts to lactate accumulation (Mitchell et al. 1981a; Gleeson and Bennett 1982).

The stimulus for the hyperventilation relative to VCO_2 that accompanies both exhaustive and sustained exercise is uncertain. One possibility is that the metabolic acidosis that occurs during the onset of sustainable exercise, or throughout exhaustive exercise, provides the stimulus for hyperventilation via peripheral or central chemoreceptors. Mitchell and Gleeson (1985) recently tested this possibility by infusing lactate into resting varanid lizards. They found that respiratory compensation to the acidosis was minor and that ventilation followed changes in VCO_2 closely, regulating Pa_{CO_2} while pH fell. These results suggest that the lactacidosis attendent to exercise in reptiles may play only a minor role in stimulating the ventilatory response to exercise, leaving other factors associated with exercise as the stimulus for hyperventilation.

The recovery period following exhaustive exercise is a period of hypoventilation relative to both VO_2 and VCO_2. The result in *Varanus salvator* is that Pa_{O_2} declines relative to resting values, and both Pa_{CO_2} and $[HCO_3^-]$ slowly increase as metabolically

Table 1. Acid-base changes following exhaustive exercise in lizards and crocodile

Variable	Units	*V. salvator*[a]	*V. exanthematicus*[a]	*I. iguana*[a]	*C. porosus*[b]
R after exercise	–	1.77	1.23	1.38	–
Pa_{O_2}	mmHg	n/c	+12	+ 9	n/c
Pa_{CO_2}	mmHg	– 4	–13	– 4	n/c
pH		– 0.25	– 0.27	– 0.44	– 0.32
$[HCO_3^-]$a	m*M*	–13	–	–	–11
[L]a	m*M*	+17	+19	+14	+23

[a] Data from Gleeson and Bennett 1982
[b] Data from Seymour et al. 1985
n/c, no change

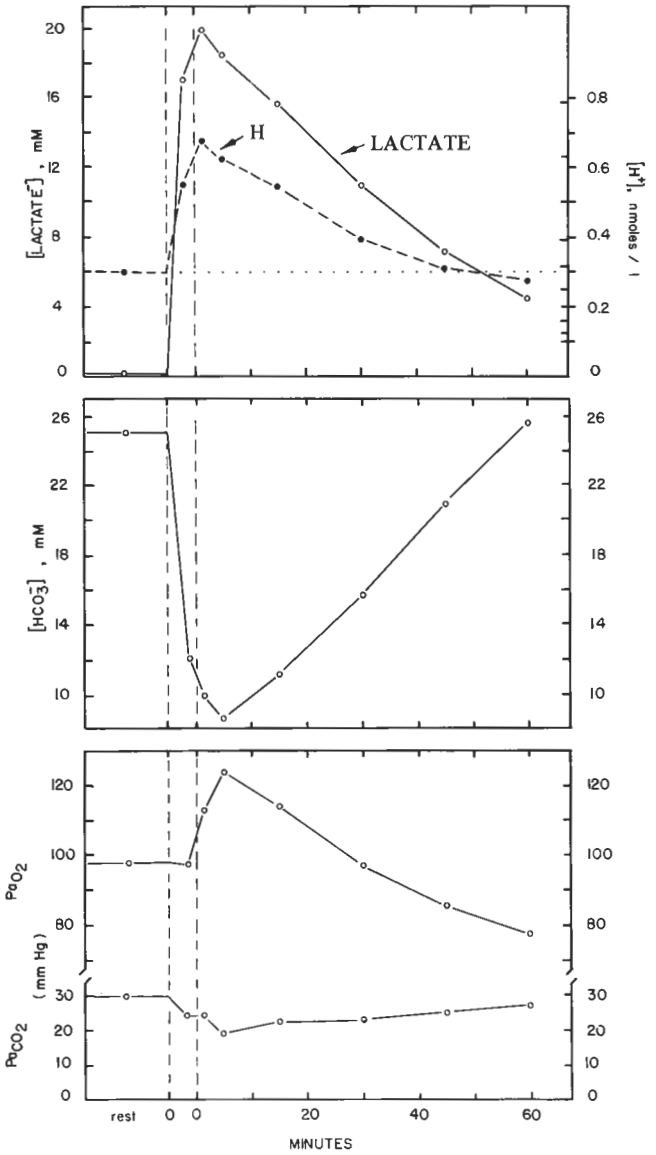

Fig. 4. Selected blood gas and acid-base variables before and after 5 min exhaustive treadmill exercise in *V. salvator* (by permission, from Gleeson and Bennett 1982)

produced CO_2 is retained and replenishes the depleted bicarbonate buffer system (Fig. 4). Values of R_e during this period may decline to 0.3–0.5. In three lizard species pH returns to preactive levels before R_e does, resulting in an alkalosis that persists for variable lengths of time following exercise. Recovery towards preexercise acid-base status is also facilitated by the metabolic removal of hydrogen ions from blood and tissues in the course of lactate catabolism. Varanid lizards become slightly alkalotic as they metabolize buffered solutions of Na^+-lactate infused into the blood (Mitchell and Gleeson 1985). Muscle gluconeogenesis in reptiles has been shown to be a possible pathway for this mechanism of metabolic recovery (Gleeson 1984).

3 Cardiovascular Adjustments to Exercise

3.1 Introduction

An increment in cardiovascular function accomodates the increased rate of gas exchange that occurs between tissue and lung during exercise. Increased blood flow facilitates the delivery of oxygen to active tissues and provides for the removal of CO_2 produced as a respiratory end product and as a consequence of lactic acid buffering. Increases in pulmonary and systemic blood flow are critically important during exercise, and towards that end changes in cardiac function play a central role. Readers are referred to White (1976) for a summary of the literature on aspects of reptilian cardiac anatomy, innervation, and function at rest.

Our understanding of how the reptilian cardiovascular system functions during exercise is rather limited. Comprehensive data on the components of cardiac output (heart rate, stroke volume) and oxygen extraction (arterial and mixed venous oxygen content. Ca_{O_2} and Cv_{O_2}, respectively) in active reptiles exist for only two lizards (Tucker 1966; Gleeson et al. 1980). Partial data exist for several other species. Confounding our understanding of reptilian cardiovascular physiology is the uncertainty over the significance of intracardiac shunts during exercise. It is apparent that right-to-left ventricular shunts occur in resting lizards, turtles, and snakes (Seymour and Webster 1975; White 1976; Berger and Heisler 1977; Seymour et al. 1981). The admixture of systemic venous blood to systemic output would reduce arterial P_{O_2} and oxygen saturation somewhat, but it is unlikely that such a shunt would seriously compromise oxygen delivery in a resting animal. Right-left shunts during running or diving, however, may be very important. Reducing pulmonary blood flow independently of systemic flow may be of some advantage to diving reptiles, and there is evidence that such a flow redistribution does occur. The sea snake *Acrochordus* responds to voluntary and forced dives by reducing pulmonary cardiac output considerably. During forced activity underwater of 2 min duration, *Acrochordus* reduces right ventricular output to 21% of its resting, eupneic rate (Seymour et al. 1981). During this period, heart rate remains unchanged and Pa_{O_2} declines, suggesting that systemic output continues at approximately preexercise rates. Seymour and Webster (1975) estimate right-left shunts in nonexercising sea snakes to be as high as 66% of total venous return. Butler et al. (1984) have shown that during periods of intermittant breathing, pulmonary blood flow in sea turtles declines between breaths, while left aortic flow remains unchanged. It is probable, therefore, that pulmonary artery and dorsal aorta blood flow may not be equal under conditions of underwater exercise in reptiles.

While right-left shunts may be advantageous during underwater exercise, shunting of venous blood during exercise on land is counterproductive to efficient gas exchange. Studies designed to evaluate the significance of and changes in cardiac shunts during exercise have not yet been performed. Data on changes in arterial and venous oxygen contents during exercise in lizards (Gleeson et al. 1980; Mitchell et al. 1981b) suggest that the shunt fraction must decrease during exercise, but these data are indirect. It would be most valuable to have direct measurements of shunt fraction, coupled with complementary data on oxygen extraction and systemic cardiac output, from exercising reptiles.

In the sections which follow we have summarized the available literature on the changes in cardiac output and oxygen extraction from arterial blood during exercise. As indicated above, these data are few, and knowledge of how they might be affected by cardiac shunts is unknown. Partly for these reasons, we have also included a discussion of the data on changes in oxygen pulse, as these changes may reflect changes in shunt fraction or aspects of cardiovascular regulation worthy of additional study.

3.2 Cardiac Output

Estimates of systemic cardiac output (Q) by the Fick equation ($Q = \dot{V}O_2/Ca\text{-}\tilde{v}O_2$) indicate that cardiac output increases in proportion to $\dot{V}O_2$ during exercise in the lizards *Varanus* and *Iguana* (Gleeson et al. 1980). Cardiac output itself is the product of changes in heart rate (HR) and stroke volume (SV), each of which are known to change independently during exercise.

Heart rate increases during exercise in nearly all reptiles studied, and is probably the primary mechanism for incrementing cardiac output in all groups. The single example where HR does not increase during activity is in the sea snake *Acrochordus* during forced activity while submerged (Seymour et al. 1981), suggesting that diving bradycardia may override an exercise-induced tachycardia. Factorial increments in HR range from 0.5–4 times, with twofold increments common. Gatten (1974a) has calculated from data on 14 species of reptiles at 30 °C that heart rate increments account for 19% (range 9–37%) of the total increment in oxygen delivery during forced, exhaustive exercise. During sustainable exercise on a treadmill HR increments may account for 30%–60% of the total increment in oxygen delivery (Gleeson et al. 1980). In *V. exanthematicus*, HR increments are the primary mechanism for increasing cardiac output at all levels of exercise (Fig. 5). Both the HR increment and its percentage contribution to total oxygen transport during exercise vary as a function of body temperature and may not always be optimal at preferred body temperatures (Moberly 1968b; Wilson and Lee 1970; Greenwald 1971; Bennett 1972; Dmi'el and Borut 1972; Gatten 1974a).

In *Varanus* and *Iguana*, increments in stroke volume have a secondary role in incrementing cardiac output. In *Iguana*, stroke volume decreased by 20% from rest to exercise. In this case, increments in heart rate are entirely responsible for increases in Q

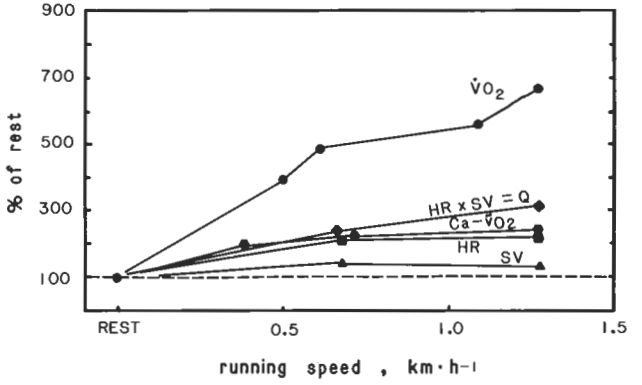

Fig. 5. Cardiovascular changes during treadmill exercise in *V. exanthematicus*. All values expressed as a percent of resting values, which were: $\dot{V}O_2$ = 0.19 ml g^{-1} h^{-1}; HR = 45 min^{-1}; SV = 0.0023 ml g^{-1} $beat^{-1}$; $Ca\text{-}\tilde{v}O_2$ = 2.6 vol%; Q = 6.7 ml g^{-1} h^{-1}. Data from Gleeson et al. 1980

(Tucker 1966; Gleeson et al. 1980). In *Varanus*, stroke volume increases 30% during exercise, so that stroke volume changes account for 40% of Q changes, while heart rate increments account for the remaining 60%. In this species, increments in SV contribute 10%–20% to the overall increase in oxygen transport (Fig. 5).

At preferred body temperatures exercise causes cardiac output to increase 100% to 200% over preexercise rates. Lizards appear to rely equally on increments in cardiac output and oxygen extraction to balance O_2 delivery needs, as do other vertebrates (Gleeson and Baldwin 1981). Data from *Varanus* suggest that the relationship between increments in VO_2 and Q is roughly linear throughout the range of VO_2 increments.

3.3 Oxygen Extraction

Delivery of oxygen to actively respiring tissues is restricted by the low oxygen carrying capacity reptilian blood, which typically has low hemoglobin concentrations and low hematocrits relative to the blood of endotherms. Hematocrit is usually within the 20%–30% range (Pough 1979). Blood oxygen capacities vary widely, but are low by mammalian standards, averaging 5.6 vol% in turtles, 8.4 vol% in lizards, and 10.2 vol% in snakes (Pough 1976).

Maximizing oxygen extraction from the blood during exercise requires that arterial oxygen content (Ca_{O_2}) during activity be maintained or increased, and that the volume of oxygen that passes through the capillary beds of the respiring tissue without participating in oxidative processes be minimized. Unlike the situation in mammals and birds, it is theoretically possible for reptiles to increase Ca_{O_2} as well as decrease $C\tilde{v}_{O_2}$ during activity. Most arterial systemic blood in reptiles is only 70%–90% saturated with oxygen at rest, with lizards tending to the lower end and turtles the higher end of that range (Pough 1979; Stinner 1981). Optimizing pulmonary ventilation-perfusion during exercise would accomplish an increased Ca_{O_2}, as would any reduction in R–L shunting of deoxygenated blood within the pulmonary circuit or heart itself. Right-left shunting has been shown to be highly variable in *Iguana* and *Varanus* (White 1959; Tucker 1966; Baker and White 1970; Berger and Heisler 1977; Wood et al. 1977), which raises the possibility that the degree of shunting is under physiological control.

Arterial C_{O_2} is maintained during exercise at resting levels in the lizards *Iguana* and *V. exanthematicus* (Tucker 1966; Gleeson et al. 1980). Maintenance of Ca_{O_2} occurs despite increases in arterial P_{O_2} known to occur during exercise (Mitchell et al. 1981b; Gleeson and Bennett 1982). This result can occur under one of two situations; decreasing shunt fraction coupled with a oxygen dissociation curve right-shifted by decreasing pH (interested readers should read both Mitchell et al. 1981b; Wood 1982), or a constant shunt fraction coupled with an increased O_2 saturation of pulmonary venous blood (made possible by the increased lung P_{O_2}; Mitchell et al. 1981b), which would offset a lower $C\tilde{v}_{O_2}$ of shunted blood. Mixed venous C_{O_2} decreases during exercise from 3–4 vol% at rest to approx. 1.8 vol% in both species. Increments in oxygen extraction account for 45%–55% of the total increment in oxygen delivery that occurs during exercise (Fig. 5). Although varanid lizards are capable of nearly twice the maximal $Ca\text{-}\tilde{v}_{O_2}$ of *Iguana*, this change contributes less to incrementing total oxygen delivery than in *Iguana*.

3.4 Oxygen Pulse

Oxygen pulse has been calculated for a number of reptile species according to the following rearrangement of the Fick equation:

$$VO_2/HR = (Ca\text{-}\tilde{v}_{O_2} \times \text{stroke volume}) = \text{oxygen pulse}.$$

Oxygen pulse has units of volumes of oxygen delivered (or consumed) per heart beat, and in all cases has been calculated from data on oxygen consumption and HR, non-invasive measures that are relatively easy to measure in active and resting animals. Oxygen pulse (OP) is the product of the two variables most difficult to measure in active animals, arteriovenous oxygen content difference ($Ca\text{-}\tilde{v}_{O_2}$) and stroke volume (SV). Because changes in oxygen pulse may be due to changes in either or both variables, knowledge of OP changes themselves are of limited value. The changes that do occur, however, point to what we feel may be a very fertile area for future research in reptilian cardiovascular physiology.

Interesting changes in OP occur as body temperature is varied. Body temperature is an important variable in reptilian biology, affecting many aspects of physiology and behavior. The changes that occur in maximal oxygen pulse (OP_{max}) with temperature suggest that its components are temperature sensitive. Oxygen pulse in resting reptiles changes little over a wide range of body temperature, i.e., resting oxygen consumption and heart rate have similar thermal dependencies. During maximal exercise, however, OP increases four to five times above resting levels, and is maximal at low body temperatures in most reptiles (Fig. 6). Maximal oxygen pulse decreases as body temperature increases in snakes (Greenwald 1971; Dmi'el and Borut 1972), lizards (Bennett 1972), and *Sphenodon* (Wilson and Lee 1970). Consequently, this aspect of oxygen delivery decreases as the metabolic demand for oxygen increases. In fact, at high body temperatures when $VO_{2\,max}$ is greatest, the factorial increment in OP during exercise may be significantly reduced (Bennett 1972; Dmi'el and Borut 1972; Wilson 1971). Decreasing maximal OP with increases in body temperature requires that either or both

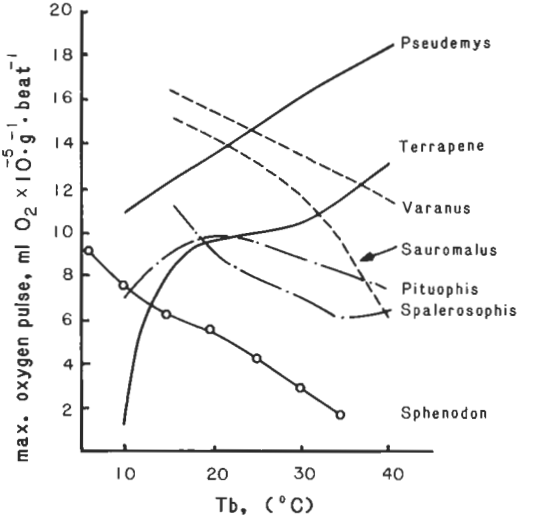

Fig. 6. Changes in maximum oxygen pulse as a function of body temperature in reptiles. Resting values of OP for all species ranged between 1 and 4 ml $O_2 \times 10^{-5}$ g^{-1} $beat^{-1}$. Data for turtles (*solid lines*) from Gatten 1974b; lizards (*dashed lines*) from Bennett 1972; snakes (*broken lines*) from Dmi'el and Borut 1972 and Greenwald 1971; *Sphenodon* from Wilson and Lee 1970

maximum oxygen extraction and stroke volume must decrease. Consequently, the three major components of cardiovascular oxygen transport, HR, SV, and $Ca\text{-}\tilde{v}_{O_2}$, must change in their relative contributions to incrementing O_2 delivery during exercise at different temperatures. What these changes are or how they are regulated have not been investigated. Since OP_{max} decreases as body temperature increases, HR increments must be responsible for a greater share of oxygen delivery increments at higher temperatures, but the changes that occur in SV or $Ca\text{-}\tilde{v}_{O_2}$ are now only conjectural. Known effects of temperature on cardiac muscle and on blood oxygen binding do not provide explanations for these changes in OP_{max}. High temperature may reduce SV by reducing cardiac tension generation or rate of tension generation. Both high and low temperatures decrease ventricular tension generation in a number of reptiles, however (Dawson and Bartholomew 1958; Licht et al. 1969), and this pattern of thermal sensitivity is not consistent as that for oxygen pulse. Maximum arterial-venous oxygen difference might also decrease with increased body temperature. Pough (1976) has shown that the oxygen capacity of reptilian blood decreases as temperature exceeds preferred body temperatures (PBT) in many species. Yet oxygen capacity also decreases at temperatures below PBT, a range of temperatures over which OP_{max} is increasing rather than decreasing. A third possibility is that R–L shunts during exercise increase as body temperatures increase. The increase in shunt fraction with temperature may occur as increased maximum heart rates or maximum flow rates through the ventricle disturb the functional separation of oxygenated and deoxygenated blood.

In contrast to the previous pattern, oxygen pulse in turtles has a different thermal dependence than that of other reptiles. Oxygen pulse at rest is more thermally sensitive in *Pseudemys* and *Terrapene* than in other reptiles (Gatten 1974b). Additionally, OP_{max} increases as body temperature increases in these species, contrary to the pattern found in lizards and snakes (Fig. 6). The influence of exercise on cardiovascular function in reptiles is complex, and the influence of temperature on the primary components of oxygen delivery is also complex and differs among orders of reptiles.

4 Areas of Future Research

The data on which this review is based are limited both in quantity and in their representation of the phylogenetic breadth of the class Reptilia. Varanid lizards are the only group for which we can say we have a fair knowledge of their ventilatory and cardiovascular responses to exercise. There is a clear need for more comparative studies of reptilian exercise physiology that take advantage of the wide array of anatomical and behavioral diversity within the group. For example, the evolutionary change in body morphology to the slender form of snakes necessitated the reduction of one lung in most snakes, while the other is greatly elongated. What effect might this change have had on oxygen delivery? A study of ventilatory responses of snakes to exercise could also address the effect of lung reduction on Pa_{CO_2} and Pa_{O_2} regulation. Cardiac morphology is also diverse among reptiles, ranging from forms where right-left ventricular shunts are large to forms with complete anatomical separation of ventricular flow. Study of crocodilian cardiovascular function during exercise would help us understand the consequences of incomplete ventricular flow separation in other reptilian groups.

Changes in body temperature have a major influence on the energy metabolism and exercise capacity of reptiles, yet the influence of body temperature on most aspects of activity physiology mentioned here is unknown. Body temperatures in excess of preferred body temperatures in many reptiles have a depressing effect on $VO_{2\,max}$ and consequently on maximal aerobic running speed. Does this effect have a pulmonary or, more probably, cardiovascular basis? Acid-base balance during exercise at different body temperatures is another area that has not been investigated.

Other subjects worthy of additional study include:

1. Control of ventilation during exercise. The presence of intrapulmonary chemoreceptors in lizards (Fedde et al. 1977; Scheid et al. 1977) and the relative ease of unidirectionally ventilating the reptile lung make these animals well suited for studies of mechanisms for acid-base and blood gas regulation.
2. Cardiac shunting during terrestrial exercise. Simultaneous measurements of pulmonary venous, mixed arterial, and mixed systemic venous blood O_2 content during exercise would allow evaluation of the importance of R–L shunts to gas exchange. Measurement of pulmonary versus systemic blood flow might also address this question. Do changes in shunt fraction explain the decline in arterial O_2 saturation seen during exercise in turtle blood?
3. Influence of temperature on the components of oxygen pulse. Do changes in shunt fraction, stroke volume, or oxygen extraction explain the changes in OP that occur as body temperature changes? What is the basis behind opposite temperature effects on OP seen in turtles compared to other reptiles?
4. Blood flow distribution during exercise. Although we assume that systemic blood flow increases in all reptiles during exercise, we have no knowledge of how this flow is distributed within or among organ systems. Existing techniques involving microsphere injections seen applicable to this question.

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