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The cardiovascular responses of the freshwater turtle *Trachemys scripta* to warming and cooling

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Summary

Seven freshwater turtles *Trachemys scripta* were instrumented with flow probes and cannulated for blood pressure measurements. The turtles were warmed from 24 to 34°C, and cooled down to 24°C, with and without atropine. Animals exhibited a hysteresis of heart rate and blood flow to both the pulmonary and systemic circulations, which was not cholinergically mediated. Blood pressure remained constant during both warming and cooling, while systemic resistance decreased during heating and increased during cooling, indicating a barostatic response. There was a large right-to-left (R–L) shunt during warming and cooling in untreated animals, which remained relatively constant. Atropinisation resulted in a large L–R shunt, which decreased during warming and increased during cooling. Nevertheless, heating rates were the same in untreated and atropinised animals, and cooling rates were significantly longer in atropinised animals, indicating that shunt patterns contribute little to heat exchange.

Key words: temperature, rate of heat exchange, turtle, *Trachemys scripta*, reptile, heart rate, blood flow, blood pressure, cardiac shunt, heart rate hysteresis.

Introduction

Many species of reptiles heat at a faster rate than they cool, indicating physiological regulation of heat transfer that can include evaporative cooling whilst panting (Templeton, 1960; Dawson and Templeton, 1966), changes in reflectance (Astatt, 1939; Norris, 1967), and even shivering thermogenesis (Harlow and Grigg, 1984; Slip and Shine, 1988). In addition, modification of heart rate and blood flow distribution may regulate rates of heat transfer between the surface and body core. For example, elevated cutaneous blood flow during warming may increase thermal conductance, while a decreased perfusion may allow for retention of heat when the animal is cooling (Baker and White, 1970). Cutaneous and carapace blood flows have been estimated using ^{133}Xe clearance in turtles (Weathers and White, 1971). When heat was applied locally to the skin, wash-out rates of the isotope increased, indicating an increased blood flow due to local peripheral vasodilation. Correspondingly, removal of the heat source caused a decrease in wash-out rates. Similar results have been obtained in other species of reptiles (Grigg and Alchin, 1976; Smith et al., 1978).

There is a close relationship between heart rate and peripheral blood flow, and heart rate has, accordingly, been taken as a sufficient indicator of changes in blood flow (e.g. Grigg and Seebacher, 1999; Seebacher, 2000). Studying the lizard *Amphibolurus barbatus*, Bartholomew and Tucker (1963) were the first to establish that heart rate, at any given body temperature, is higher during warming than cooling; this response is known as heart rate hysteresis. This phenomenon has subsequently been documented in many other lizards (e.g. Bartholomew and Lasiewski, 1965) and reviewed by Grigg et al. (1979), Seebacher (2000) and Seebacher and Grigg (2001), crocodilians (Smith, 1976; Franklin and Seebacher, 2003) and turtles (Weathers and White, 1971; Voigt, 1975). Heart rate hysteresis also occurs in free-ranging *Pogona barbata* (previously known as *A. barbatus*), and the corresponding changes in blood flow have been estimated to prolong significantly the time that body temperature remains within the preferred range (Grigg and Seebacher, 1999; Seebacher, 2000).

Heart rate hysteresis may be a simple consequence of a barostatic regulation that acts to alter heart rate in response to changes in peripheral resistance, but blood pressures have generally not been measured during changes in body temperature. However, heart rate hysteresis persists following total autonomic blockade in *Pogona barbata* (Seebacher and Franklin, 2001), and autonomic blockade does not affect subcutaneous blood flow in the iguana *Ctenosaura hemilopha* during heating (Weathers and Morgareidge, 1971).

Apart from regulated changes in the perfusion of the surface of the body, it has also been proposed that cardiac shunt patterns should influence the rate of temperature change in reptiles (Tucker, 1966; Baker and White, 1970; Hicks, 1998) as bypass of the pulmonary circulation could reduce heat loss

at the lung surface and promote heating. According to Tucker (1966), bypass of the pulmonary circulation reduces heat loss at the lung surface and promotes heating. In turtles, blood flow distribution among the systemic and pulmonary circulations is determined by their relative vascular resistances (Crossley et al., 1998; Hicks, 1998). It is possible, therefore, that changes in the net cardiac shunt patterns merely reflect a passive consequence of an altered balance between the vascular resistance in the systemic and pulmonary circulations (Hicks, 1998). Thus, when resistance in the systemic circulation decreases during heating, a net right-to-left (R–L) cardiac shunt develops, while the increased systemic resistance during cooling leads to a net L–R shunt developing. However, an increased R–L shunt during warming will lower arterial oxygen levels (Wang and Hicks, 1996), reducing oxygen supply even though demands for oxygen are rising. Consequently, the benefits conferred by increasing rates of heat transfer during warming may be countered by the need to maintain sufficient rate of oxygen delivery.

So far, no previous studies have provided a complete set of measurements of systemic and pulmonary blood flows during heating and cooling in reptiles, and blood pressures are rarely reported. Here we wish to establish whether blood pressure remains constant during heating and cooling, which would be indicative of a functional barostatic regulation. Secondly, having established normal blood flows and net shunt pattern during heating and cooling, we wish to manipulate pulmonary blood flow using atropine infusion to investigate whether the rates of heating and cooling are affected.

Materials and methods

Experimental animals

Studies were undertaken on seven freshwater turtles *Trachemys scripta* (Gray), obtained from Lemberger Inc. (Oshkosh, WI, USA), and transported to Aarhus University where they were maintained for more than 6 months before use. All animals were kept in a 1 m×1 m fibreglass tank containing water at 28°C (40 cm depth) and dry basking platforms that allowed access to heating lamps for behavioural thermoregulation. All experimental animals appeared healthy and had a body mass of 0.94–1.65 kg.

Surgery and instrumentation

For surgery, turtles were placed ventral side up, intubated with soft rubber tubing inserted through the glottis, and artificially ventilated using an HI 665 Harvard Apparatus Respirator (Cambridge, MA, USA), at approximately 15–25 breaths min^{-1}, and a tidal volume of 30–50 ml kg^{-1}. The ventilator was connected to an isoflurane vaporizer (Keighley, England) initially set at 4% isofluorane, which normally led to disappearance of the pedal withdrawal reflex within 15 min. The following surgery was performed at 1–1.5% isofluorane. A 4 cm×4 cm piece of the ventral plastron was removed above the heart, using a bone saw. The pectoral muscles were loosened from the excised piece of plastron and connective tissue surrounding the left pulmonary artery (LPA) and left aortic arch (LAo) was carefully separated, so that ultrasonic blood flow probes (Transonic System Inc., NY, USA) could be positioned around the vessels. Acoustical gel was placed around the flow probes to displace air bubbles that could disturb the signal. The left carotid artery was occlusively cannulated for blood pressure measurements with PE-50 catheter tubing, filled with heparinised saline, and previously treated with the anti-coagulate triodecylmethylammonium chloride (TD-Mac) heparin complex to reduce blood clotting. Occasionally the catheter was flushed with heparinised saline. Finally, a thermistor, enclosed in a soft rubber coating, was placed beside the heart for measurements of core body temperature. A small notch was cut into the excised piece of plastron, so the leads and cannula could exit the animal, and it was then replaced in the animal and glued in place using silicone adhesive and two-component epoxy glue. The animal recovered from anaesthesia by continuing the artificial ventilation with air for approximately 25 min. When it had regained reflexes and spontaneous ventilation, it was transferred to a holding chamber (30 cm×30 cm) overnight for recovery. When the experimental protocol was completed, turtles were killed by an overdose of pentobarbitol (vascular infusion of 200 mg kg^{-1}).

Experimental protocol

Turtles were allowed to recover for 16–48 h after surgery. On the first day each animal was heated and cooled, while heart rate (f_H), systemic blood pressure (P_{sys}), pulmonary blood flow ($\dot{Q}_{pul}$), systemic blood flow ($\dot{Q}_{sys}$), and body temperatures (T_b) were recorded. In addition, a temperature probe was placed on the top of the carapace for surface temperature (T_s) measurements. In order to warm the animal, turtles were held in air in a box 50 cm×40 cm×60 cm with an infrared (E27) 150 W heating lamp placed 10 cm above the box. When T_b had reached 34°C, the heat lamp was switched off and the animal cooled to approximately 23–24°C. During the heating phase the animal exhibited variable periods of activity, causing variations in heart rate. The animal was then left overnight at room temperature in the box with access to water. On the second day the heating and cooling was repeated using identical conditions following an intra-arterial injection of atropine (5 mg kg^{-1}), delivered through the catheter. The animal was left for a period of 1 h and then heated and allowed to cool, as previously described. During heating, the surface temperature probe recorded an increase in temperature from 23±0.4 to 31.4±0.6°C during the first 2.6±0.2 min. Following this, temperature rose to 38.9±0.6°C, at which point the heat lamp was switched off. The temperature then dropped to 30.3±0.5°C within the first 4.3±0.4 min, and further decreased to 24.0±0.2°C after a period of 309±18 min. All animals were unrestrained throughout all of the experiments. The data for relative rates of warming or cooling were mass specific.

Data recording

The arterial catheter was connected to a Baxter Edward

disposable pressure transducer (model PX600, Irvine, CA, USA), which was calibrated daily against a static water column. The signals from the pressure transducer were amplified using an in-house built preamplifier. The blood flow probes were connected to a dual-channel blood flow meter (Transonic T206). All signals, including the temperature measurements, were recorded using a Biopac MP100 at 100 Hz.

Data analysis and calculations

The data were analysed by taking an average of each physiological variable at each desired temperature interval (24–25, 25–26,……., 33–34) during warming and cooling.

Heart rate was obtained from the pulsatile pressure of the carotid artery. All blood flows were corrected for body mass, and following this $\dot{Q}_{sys}$ was calculated as $2.85\times\dot{Q}_{LAo}$ (Wang and Hicks, 1996), and likewise $\dot{Q}_{pul}$ was recorded as $2\times\dot{Q}_{LPA}$ under the assumption that both pulmonary arteries receive equal flows. Systemic resistance (R_{sys}) was calculated using the mean blood pressure and mean blood flow ($R_{sys}=P_{sys}/\dot{Q}_{sys}$). This calculation assumes that central venous blood pressures are zero, which may lead to an underestimation of R_{sys}. The net shunt flow ($\dot{Q}_{shunt}$) was calculated as $\dot{Q}_{pul}-\dot{Q}_{sys}$. A negative value would therefore indicate a net R–L shunt, and a positive value indicates a net L–R shunt. The proportion of pulmonary blood flow relative to systemic blood flow was expressed as $\dot{Q}_{pul}/\dot{Q}_{sys}$. Total stroke volume ($V_{Stot}$) was calculated by dividing total blood flow ($\dot{Q}_{sys}+\dot{Q}_{pul}$) by heart rate.

All haemodynamic variables were analysed using a general linear model 3-way analysis of variance (ANOVA) for repeated measures using a Minitab statistical package. Data were tested for differences among untreated and atropinised animals, and whether the animal was warming or cooling. The independent variables were body temperature (T_b), untreated or atropinised, and warming or cooling. Body temperature can be taken as an independent variable rather than time, because measurements of blood flow and other cardiovascular variables were taken continuously but sampled for analysis at a series of set temperatures. This rules out the need to compensate for different warming or cooling rates between animals and the need to make data mass specific, although blood flow data was of course corrected for body mass. Data from animals with a stable body temperature of 22°C, before heating commenced, on the first and second day were compared using a paired *t*-test. The effect of atropine on the second day was also assessed using a paired *t*-test. Rates of heating and cooling (min per degree temperature change) and the effect of atropine were analysed with a two-way analysis of variance for repeated measures (two-way RM ANOVA). Differences were considered significant at a 95% confidence levels, when $P<0.05$.

Results

Resting values

Table 1 shows haemodynamic variables of resting turtles with a body temperature of 22°C, before heating, for untreated animals on the first day, and values before and after infusion of atropine on the second day. f_H, $\dot{Q}_{pul}$, and $\dot{Q}_{sys}$ increased significantly with atropine, causing a significant L–R shunt, while there were small insignificant changes in V_{Stot}, P_{sys}, or R_{sys}. There were no significant differences between any haemodynamic variables on the first and second day.

Heating and cooling rates

Untreated turtles placed under the heating lamp on average heated four times faster than they cooled (Fig. 1A). This was also the case with atropinised turtles. However, while all animals heated at similar rates, the time taken per degree temperature change during cooling in atropinised animals was significantly longer at all temperatures than in the untreated animals (Fig. 1B), so that the time taken for cooling in these animals was prolonged by approximately 1 h in comparison to untreated individuals (Fig. 1A). This was more pronounced at temperatures below 27°C, which accounted for the extended cooling period seen in atropinised turtles.

Haemodynamic variables

Heart rate was significantly higher during warming than during cooling at all temperatures, with all animals exhibiting a classic hysteresis of heart rate (Fig. 2A). In untreated animals, this was particularly evident at body temperatures

Table 1. *Resting haemodynamic variables for untreated and atropinised turtles*

Treatment	f_H (beats min^{-1})	V_{Stot} (ml kg^{-1})	$\dot{Q}_{pul}$ (ml kg^{-1} min^{-1})	$\dot{Q}_{sys}$ (ml kg^{-1} min^{-1})	R_{sys} (kPa ml^{-1} min^{-1} kg^{-1})	P_{sys} (kPa)	$\dot{Q}_{shunt}$ (ml kg^{-1} min^{-1})	$\dot{Q}_{pul}/\dot{Q}_{sys}$
Day 1	20.7±2.1	2.7±0.2	29.6±3.8	27.7±3.2	0.13±0.01	3.76±0.34	1.68±3.11	1.10±0.12
Day 2 untreated	20.7±2.6	3.1±0.1	31.9±7.49	31.1±4.7	0.13±0.02	3.89±0.40	–0.23±10.4	1.14±0.33
Day 2 atropinised	34.6±1.4*	3.9±0.7	88.6±19.3*	38.1±5.1*	0.11±0.02	3.99±0.37	48.8±21.8*	2.49±0.58*

Resting undisturbed turtles had a body temperature of 22°C. Values are given for turtles before heating in untreated animals on the fiirst day, and before and after atropine infusion (5 mg kg^{-1}) on the second day.

Values are means ± S.E.M. (N=6 for all variables except f_H and $\dot{Q}_{sys}$, where N=7).

f_H, heart rate; V_{Stot}, total stroke volume; $\dot{Q}_{pul}$, pulmonary blood flow; $\dot{Q}_{sys}$, systemic blood flow; R_{sys}, systemic resistance; P_{sys}, mean s systemic blood flow; $\dot{Q}_{shunt}$, net shunt flow; $\dot{Q}_{pul}/\dot{Q}_{sys}$, ratio of pulmonary and systemic blood flow.

*Significant difference from untreated animals; ‡significant difference between untreated and pre-atropinised animals ($P<0.05$).

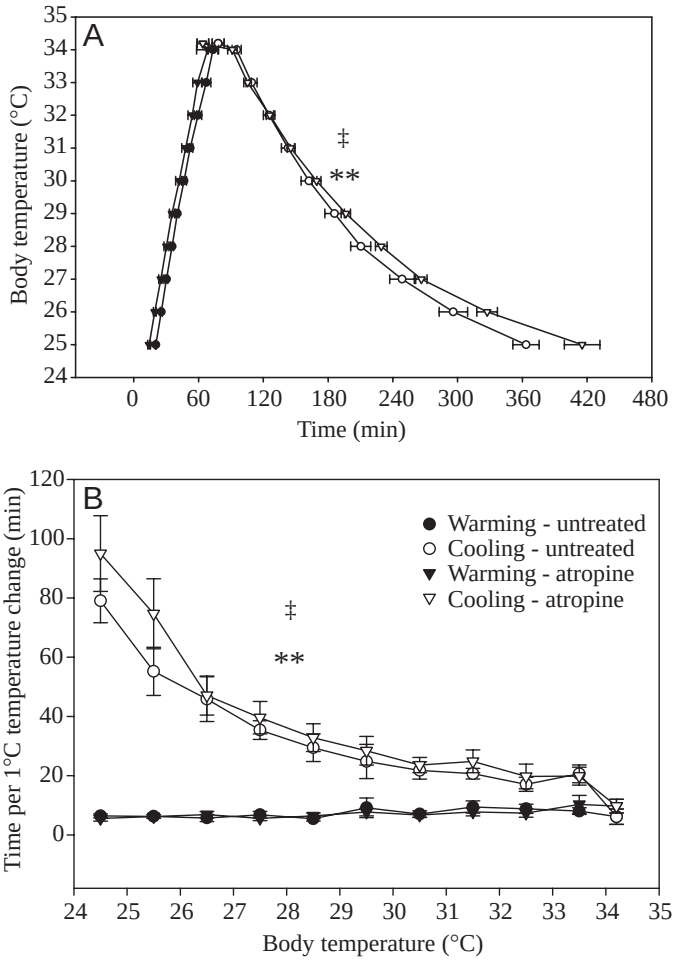

Fig. 1. Warming and cooling curves for untreated and atropinised turtles. (A) Heating and cooling curves. (B) The time taken per degree temperature change during warming and cooling. Values are means ± S.E.M. (N=7). **Significant difference between warming and cooling; ‡significant difference between atropinised and untreated animals.

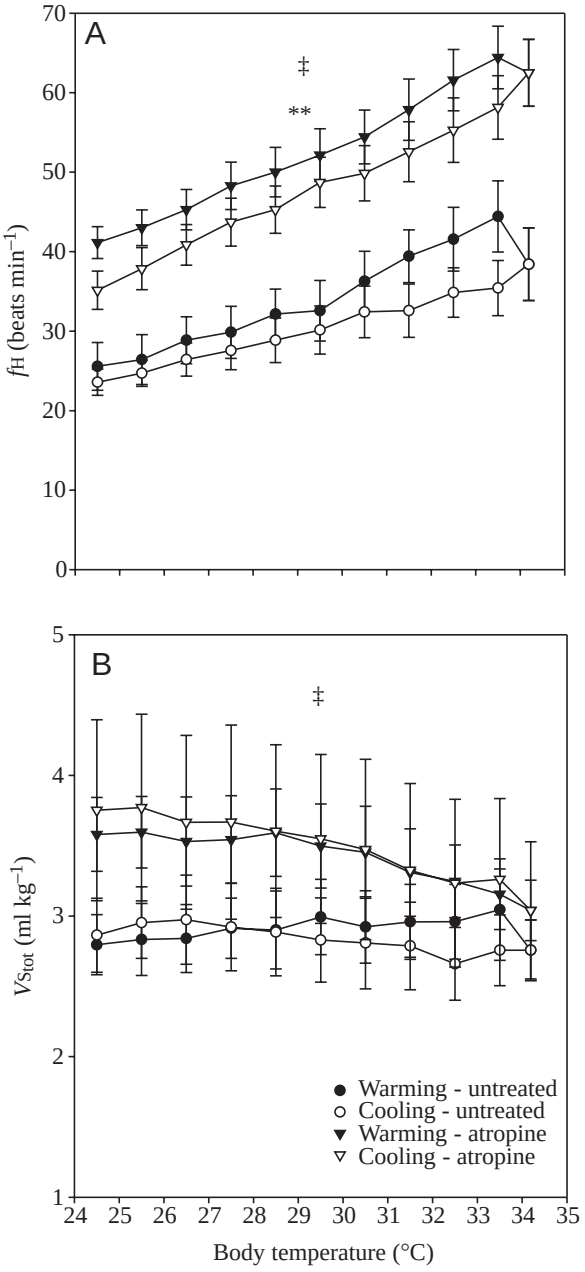

Fig. 2. Heart rate (f_H) (A) and total stroke volume (V_{Stot}) (B) during warming and cooling in untreated and atropinised animals. Values are means ± S.E.M. (N=6 and 7 in A and B, respectively). **Significant difference between warming and cooling; ‡significant difference between atropinised and untreated animals.

above 30°C. In atropinised turtles, heart rate was significantly higher, being almost double the untreated rate at all temperatures (Fig. 2A). There was no significant change in V_{Stot} during heating and cooling in untreated and atropinised animals (Fig. 2B). However, atropine significantly increased V_{Stot}.

In untreated animals, pulmonary blood flow increased twofold during warming, and decreased correspondingly during cooling to previous resting values (Fig. 3A). At body temperatures above 30°C, $\dot{Q}_{pul}$ was significantly lower during cooling than during warming. Overall, $\dot{Q}_{pul}$ was significantly higher during warming than cooling. In atropinised animals, $\dot{Q}_{pul}$ was significantly twofold higher than in untreated animals. $\dot{Q}_{sys}$ was considerably higher during heating than during cooling in both untreated and atropinised turtles (Fig. 3B). Atropinised animals had higher $\dot{Q}_{sys}$ than untreated animals during both heating and cooling, but this difference was not significant. Systemic blood pressure (P_{sys}) remained constant during both warming and cooling, in both untreated and atropinised animals (Fig. 4A). However, P_{sys} was significantly higher during warming than cooling. The reduced $\dot{Q}_{sys}$ during cooling was reflected by an increase in systemic vascular resistance (R_{sys}) (Fig. 4B). In untreated turtles, R_{sys} was higher during cooling at all temperatures, with the exception of 25°C, while in atropinised animals, R_{sys} was higher during cooling at all temperatures above 26°C (Fig. 4B). In addition, atropinised animals had significantly lower R_{sys} values during both

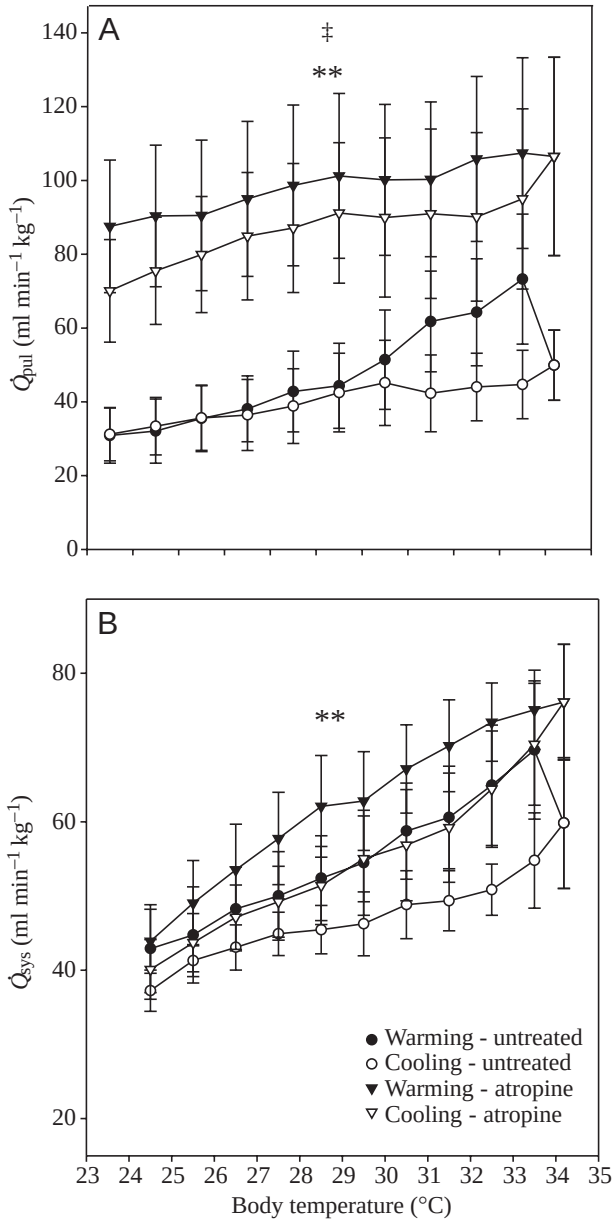

Fig. 3. Pulmonary ($\dot{Q}_{pul}$) (A) and systemic ($\dot{Q}_{sys}$) (B) blood flow during warming and cooling in untreated and atropinised animals. Values are means ± S.E.M. (N=7). **Significant difference between warming and cooling; ‡significant difference between atropinised and untreated animals.

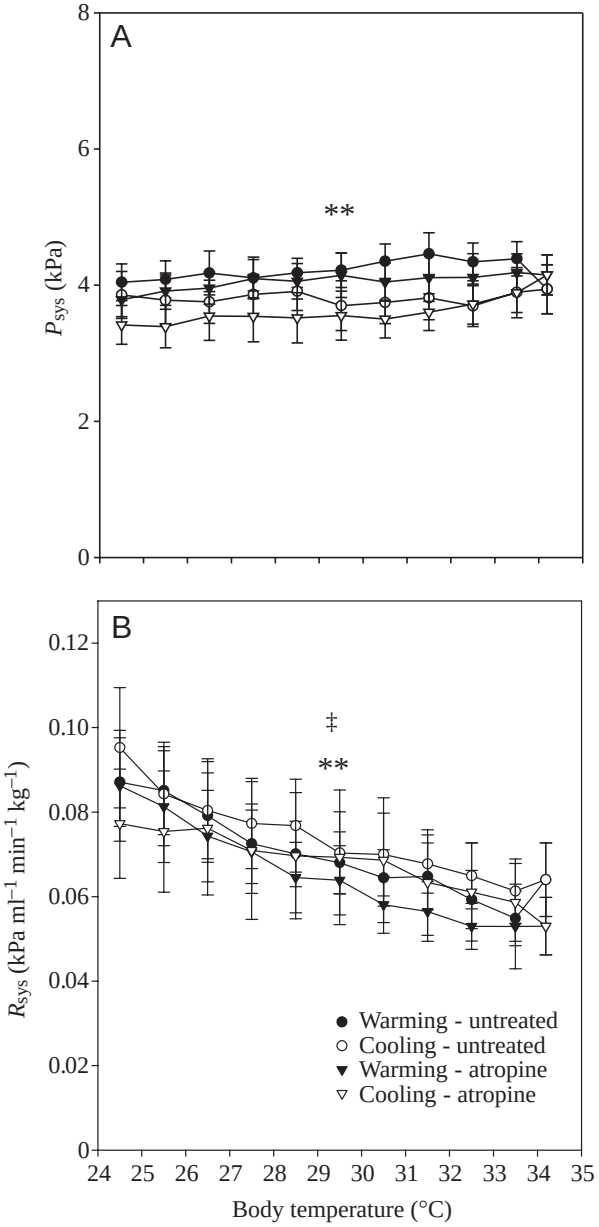

Fig. 4. Systemic pressure (P_{sys}) (A) and systemic resistance (R_{sys}) (B) during warming and cooling in untreated and atropinised animals. Values are means ± S.E.M. (N=6). **Significant difference between warming and cooling; ‡significant difference between atropinised and untreated animals.

warming and cooling, compared to those of the untreated turtles.

There was a net right-to-left shunt in untreated turtles at all temperatures regardless of whether the turtles were heating or cooling (Fig. 5A). However, the values remained relatively constant, until temperatures above 31°C, when there was a decrease in the right-to-left shunt during warming and an increase in the right to left shunt during cooling. However, there was no significant difference in $\dot{Q}_{pul}/\dot{Q}_{sys}$ between warming and cooling. In atropinised animals, a very large net left-to-right shunt prevailed during both heating and cooling, which was significantly different from untreated animals. However, as heating proceeded, this net left to right shunt progressively decreased, and then increased during cooling (Fig. 5A).

Discussion

Rates of heating and cooling

All animals heated at a faster rate than they cooled. This is

likely to be due to the heating and cooling regime they were exposed to, with the heating lamp generating a rapid rate of temperature rise, while cooling towards room temperature was a slow process. However, a faster rate of heating is consistent with previous studies on turtles, including *Trachemys*, and other species of reptiles (Bartholomew and Tucker, 1963; Spray and May, 1971; Weathers, 1971; Lucey, 1974; Voigt, 1975; Grigg et al., 1979; O'Connor, 1999; Seebacher and Franklin, 2001).

Hysteresis of heart rate and blood flows

Heart rate at any given body temperature was higher during heating than cooling. This 'heart rate hysteresis' has been described previously in turtles (Lucey, 1974; Voigt, 1975; Smith et al., 1981) and numerous other reptiles (Bartholomew and Tucker, 1963; Grigg et al., 1979; Grigg and Seebacher, 1999; Seebacher, 2000). The higher f_H values during heating were accompanied by an increased $\dot{Q}_{sys}$, but because systemic vascular resistance (R_{sys}) was reduced, blood pressure was not affected by temperature. Conversely, systemic blood flow was reduced during cooling and R_{sys} was increased so blood pressure was once again maintained unchanged (Fig. 4A,B). Furthermore, blood pressure does not change appreciably when reptiles are acclimated to different temperatures for several hours or days (e.g. Stinner, 1987; Lillywhite and Seymour, 1978; T. Wang, A. Neto, E. W. Taylor, P. Koldkjær, D. Andrade and A. S. Abe, manuscript submitted for publication). It is likely that hysteresis of heart rate reflects a barostatic regulation of blood pressure, where heart rate is increased in response to the lower R_{sys} during heating and *vice versa* during cooling. The effects of temperature on the cardiac limb of the barostatic response are not well characterised, but it remains functional over a wide range of temperatures in snakes and seems to contribute to maintenance of an almost temperature-independent blood pressure (T. Wang, A. Neto, E. W. Taylor, P. Koldkjær, D. Andrade and A. S. Abe, manuscript submitted for publication). In several species of reptiles, the heart rate responses to altered blood pressure can be fully blocked by double autonomic blockade (i.e. the combination of atropine with a beta blocker; e.g. Altimiras et al., 1998). However, the hysteresis of heart rate was not abolished by autonomic blockade in the lizard *Pogona barbata* (Seebacher and Franklin, 2001), and the hysteresis also persisted after atropinisation in our study (Fig. 2). Obviously, in order to fully investigate the suggestion that heart rate hysteresis is a consequence of a barostatic response, further investigation of the regulation of blood pressure during heating and cooling is required that should include adrenergic blockade and vagotomy to remove both the afferent and efferent arms of the barostatic response.

Our study provides the first complete set of measurements of systemic and pulmonary blood flows during heating and cooling in a reptile, but our measurements cannot reveal whether heating and cooling are associated with preferential perfusions of the vascular beds in the skin and carapace, where heat exchange is presumed to occur. Heart rate has previously been used as an indicator of blood flow (Grigg and Seebacher, 1999; Seebacher, 2000). In several species of reptiles, the ^{133}Xe isotope clearance method shows that cutaneous blood flows increase during warming, and decrease during cooling (Morgareidge and White, 1969; Weathers and White, 1971; Baker et al., 1972; Smith et al., 1978; Weinheimer et al., 1982). Furthermore, in *Iguana iguana*, using laser doppler, Dzialowski and O'Connor (2001) observed increased and decreased cutaneous blood flows with warming and cooling, respectively. When blood flow was plotted against body temperature, a strong hysteresis pattern was observed. This is consistent with our study, where *T. scripta* exhibited a hysteresis in changes in blood flow in both the systemic and the pulmonary circulation during warming and cooling.

Intracardiac shunt patterns

Although the physical conditions for heating and cooling were different, an identical protocol was used before and after the atropinisation period; hence, our experiment was designed to allow evaluation of the effects of net cardiac shunt patterns on heat transfer. It has been suggested that an increased R–L shunt during warming contributes to an increased rate of heating (Tucker, 1966), but a comprehensive description of $\dot{Q}_{pul}$ and $\dot{Q}_{sys}$ during heating and cooling has not previously been reported. Hicks (1998) argued that changes in cardiac shunt patterns when body temperature changes merely reflect differences in vascular resistances of the systemic and pulmonary circulations, rather than representing an actual regulation of the shunt patterns. In our study, the turtles exhibited a substantial R–L shunt that persisted during heating and subsequent cooling, although there was a tendency for a higher $\dot{Q}_{pul}/\dot{Q}_{sys}$ at the highest temperatures during heating (Fig. 5). This indicates that pulmonary vascular resistance is decreased progressively as R_{sys} was reduced during heating. After infusion of atropine, a large L–R shunt prevailed, but $\dot{Q}_{pul}/\dot{Q}_{sys}$ was progressively reduced with elevated temperature. Thus, when the turtles were unable to reduce pulmonary vascular resistance through the vagal innervation of smooth muscle surrounding the pulmonary artery, cardiac output was directed towards the systemic circulation as R_{sys} decreased in response to elevated temperature. During heating in untreated animals, progressive changes in the relative resistances in the pulmonary and systemic circuits may prevent the development of large R–L shunts as temperature rises. This response may reflect a need to maintain oxygen delivery as metabolism rises with temperature (e.g. Wang et al., 2001). Indeed, in rattlesnakes, varanid lizards and toads, the R–L shunt is reduced when acclimated to increased temperature (Ishimatsu et al., 1988; Wang et al., 1998; Gamperl et al., 1999).

The reversal of cardiac shunt patterns after atropine and the large increase in heart rate did not affect the rate of heating, even though $\dot{Q}_{sys}$ was elevated. Weathers and White (1971) also observed that atropine infusion failed to alter the rate of temperature change in a previous study on turtles, leading to the conclusion that the rate of temperature change is not obviously

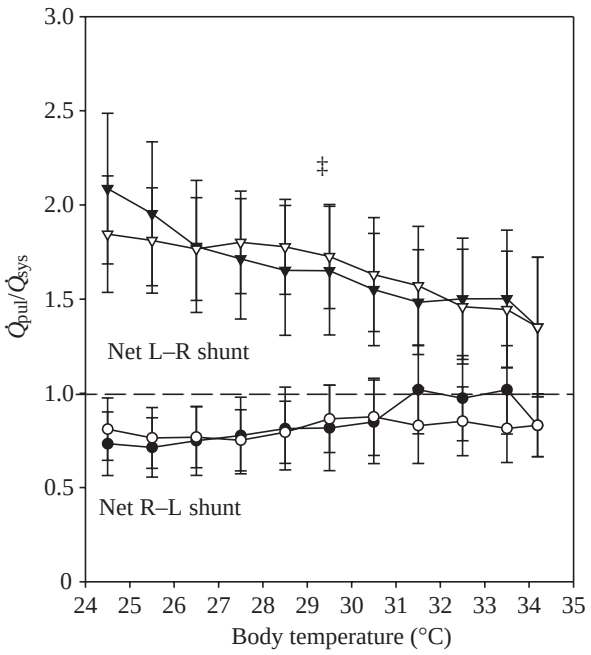

Fig. 5. Pulmonary blood flow divided by systemic blood flow ($\dot{Q}_{pul}/\dot{Q}_{sys}$) during warming and cooling in untreated and atropinised animals. Values are means ± S.E.M. (N=6). **Significant difference between warming and cooling; ‡significant difference between atropinised and untreated animals.

related to heart rate, systemic blood flow or the net cardiac shunt. In the lizard *Pogona barbata*, the rate of heating is, however, faster after atropine and slower after adrenergic blockade with sotalol (Seebacher and Franklin, 2001). It is possible that the higher $\dot{Q}_{pul}$ after atropine leads to an increased heat loss across the lungs, which could have offset an increased rate of heat uptake over the skin and carapace. However, if heat is lost at the lungs surface as a consequence of a large L–R shunt, then it would be expected that the rate of cooling in atropinised animals would be reduced. Conversely, in this study the rate of cooling was significantly faster in untreated turtles than in atropinised animals. It is therefore unlikely that increased pulmonary blood flow would cause heat to be lost across the surface of the lungs.

Concluding remarks

The maintenance of a constant blood pressure as body temperature changed indicates that the hysteresis of heart rate during warming and cooling of reptiles reflects barostatic regulation of blood pressure, but because this response persists in atropinised turtles, the efferent arm of this control remains to be understood. Future studies should include experimental manipulation of blood pressure, so that its effects on rates of warming and cooling can be ascertained. Major changes in the cardiac shunt pattern do not seem to affect the rates of temperature change, so that we have to conclude that changes in the net direction of blood flow, while critically involved in determining respiratory gas exchange, seem ineffective in determining heat exchange.

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