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Comparative Biochemistry and Physiology, Part A 148 (2007) S4–S6
CBP
www.elsevier.com/locate/cbpa

Seventh International Congress of Comparative Physiology and Biochemistry Salvador, Bahia, Brazil, August 12–16, 2007

1. Cardiovascular responses to heat

1.1. How important are changes in blood flow distribution for thermoregulation in a reptile (*Crocodylus porosus*)?

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Changes in blood flow are a principal mechanism of thermoregulation in vertebrates. Changes in heart rate will alter blood flow, although multiple demands for limited cardiac output may compromise effective thermoregulation. We tested the hypothesis that regional differences in blood flow during heating and cooling can occur independently from changes in heart rate. We measured heart rate and blood pressure concurrently with blood flow in an ectothermic vertebrate (*Crocodylus porosus*). Blood flow was measured by laser Doppler flowmetry, and by injecting coloured microspheres. All measurements were made under different heat loads, with and without blocking cholinergic and α-adrenergic receptors (autonomic blockade). Heart rates during heating were significantly faster than during cooling in the control, but not when autonomic receptors were blocked. There were no significant differences in blood flow distribution between control treatments, and autonomic blockade treatments. In both treatments, blood flow was directed to the dorsal skin and muscle and away from the tail and duodenum during heating. When the heat source was switched off, there was a shunt of blood from the dorsal surface to the duodenum. Blood flow to the leg skin and muscle, and to the liver did not change significantly with thermal state. Blood pressure was significantly higher during the autonomic blockade than during the control. Thermal time constants of heating and cooling were unaffected by the blockade of autonomic receptors and we concluded that animals partially compensated for lack of differential heart rates during heating and cooling by redistributing blood within the body, and by increasing blood pressure to increase flow.

doi:10.1016/j.cbpa.2007.06.008

doi:10.1016/j.cbpa.2007.06.007

1.2. Cardiovascular responses to temperature and their effect on oxygen delivery in reptiles

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The respiratory system is designed so that no step in the cascade is limiting to oxygen delivery. Changes in body temperature also effect other control systems. For example, the respiratory system can be used to improve thermolysis whereas the cardiovascular system can influence the distribution of heat throughout the body affecting the balance between thermolysis and heat gain. To this extent, it is generally accepted that ectotherms display a hysteresis in heart rate and hence peripheral flow to enhance the uptake of heat during warming and/or to slow the loss of heat during cooling. It is plausible, therefore, that conflicts can arise between thermoregulatory requirements, the need for adequate oxygen delivery to meet demand, and the need to maintain appropriate blood pH. This paper examines the effects of cooling and rewarming in a goanna (*Varanus rosenbergi*) and the estuarine crocodile (*Crocodylus porosus*) in terms of the relationships between ventilation, oxygen delivery by the cardiovascular system, metabolic rate and acid base state.

doi:10.1016/j.cbpa.2007.06.009

1.3. Thermal acclimation confers no tolerance to acute temperature change in cardiac myocytes from the bluefin tuna

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Whole cell voltage clamp was used to investigate cellular Ca cycling in bluefin tuna (*Thunnus orientalis*) atrial and

ventricular cardiac myocytes. Tuna were acclimated to either 15 °C or 23 °C for a minimum of 3 weeks before experimentation. Experiments were carried out at test temperatures of 10, 15 and 23 °C. Thermal acclimation had little effect on the ability of the SR to accumulate Ca. However, L-type Ca current (ICa) density was significantly reduced in cold acclimated fish compared with warm acclimated fish when tested at 23 °C. Acute reductions in temperature significantly decreased both ICa and SR Ca content in atrial and ventricular myocytes from both acclimation temperatures. Results indicate that acute reductions in temperature reduce Ca flux both across the sarcolemmal membrane and through the SR. This will place a significant burden on the heart of the bluefin tuna and may explain the known reduction in heart rate and myocardial output at decreased temperatures. Surprisingly, cold acclimation conferred no tolerance to acute drops in temperature.

doi:10.1016/j.cbpa.2007.06.010

1.4. Non-thermal modulation of the cardiovascular response to a heat stress: a potential role for the AV3V region

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Elevations in core temperature stimulate a redistribution of cardiac output from the viscera to the periphery, thereby allowing greater heat dissipation. However, this heat-induced redistribution of blood flow is also strongly influenced by non-thermal factors such as hydration status. Dehydration both delays the onset and reduces the magnitude of cutaneous dilation resulting in hyperthermia and potentially heat illness. While it is clear that thermal and non-thermal information are integrated centrally, where in the brain this occurs remains unknown. One potential integrative site is the periventricular tissue that surrounds the anteroventral third ventricle (AV3V). The AV3V encompasses the organum vasculosum of the lamina terminalis, the ventral median and periventricular preoptic nuclei and has been functionally implicated in behavioural, hormonal and neural effector mechanisms related to body fluid and cardiovascular regulation. In a series of recent studies we have demonstrated that the AV3V also has an important thermoregulatory role. Lesions of the AV3V reduce thermal tolerance by disrupting various physiological and behavioural thermoregulatory mechanisms. In particular, AV3V lesions attenuate the cardiovascular response to heat stress, reducing the increase in heart rate, mesenteric resistance and mean arterial pressure normally associated with hyperthermia. Furthermore, the hyperosmotic modulation of two thermoregulatory mechanisms was also eliminated by disruption of the AV3V. These data indicate that this anatomically small region has a large role to play in thermoregulation.

doi:10.1016/j.cbpa.2007.06.011

1.5. Cardiac survival during temperature change from fish to mammals: a role for altered calcium homeostasis

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Maintenance of heart function during temperature change is important for the survival of most vertebrates. Contraction and relaxation of the whole heart is driven by contraction and relaxation of individual cardiac myocytes. At the level of the myocyte, contractility is controlled by the cellular cycling of calcium. This talk will discuss the temperature sensitivity of the many protein pumps and ion channels that cycle calcium in the heart and how their regulation during temperature change is important for calcium homeostasis. In particular the role of the sarcoplasmic reticulum in maintaining cellular calcium homeostasis during temperature change will be addressed. The discussion will explore how the role of the sarcoplasmic reticulum differs in eurythermal fish and hibernation mammalian cardiomyocytes and the implications this has for surviving temperature change.

doi:10.1016/j.cbpa.2007.06.012

1.P1. Axolotls as a potential model to study the miocardial restoration

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The heart of axolotls, like in other amphibians, beats spontaneously in vitro for long periods of time at room temperature (1), which allows the evaluation of the myocardial contractile activity without the need of field stimulation (2). Using the isolated organ assays we found that in heart of *Ambystoma dumerilii* the heart rate (HR) increased and the tension (T) decreased when temperature was augmented. One day after in vivo heart damage with isoproterenol (Iso), the HR (43–24%) and T (50–25%) between 8 and 24 °C decreased with respect to control. Five, 30 and 90 days after Iso, HR showed a gradual recovery with similar effect when the temperature was changed;