The aim of this study was to evaluate the effects of the stimulation of central cholinergic synapses in the regulation of heat loss in untrained rats during exercise. The animals were separated into two groups (exercise or rest) and tail skin temperature (T\text{tail}), core temperature and blood pressure were measured after injection of 2 µL of 5 x 10^{-3} M physostigmine (Phy; n = 8) or 0.15 M NaCl solution (Sal; n = 8) into the lateral cerebral ventricle. Blood pressure was recorded by a catheter implanted into the abdominal aorta, T\text{tail} was measured using a thermistor taped to the tail and intraperitoneal temperature (T\text{b}) was recorded by telemetry. During exercise, Phy-treated rats had a higher increase in mean blood pressure (147 ± 4 mmHg Phy vs. 121 ± 3 mmHg Sal; P < 0.001) and higher T\text{tail} (26.4 ± 1.0° C Phy vs. 23.8 ± 0.5° C Sal; P < 0.05) that was closely related to the increase in systolic arterial pressure (r = 0.83; P < 0.001). In addition, Phy injection attenuated the exercise-induced increase in T\text{b} compared with controls without affecting running time. We conclude that the activation of central cholinergic synapses during exercise increases heat dissipation due to the higher increase in blood pressure.

**Key words:** thermoregulation; arterial pressure; central cholinergic synapses; fatigue

**INTRODUCTION**

Mammals maintain their body core temperature constant at ~37° C regardless of fluctuations in environmental temperature. Body temperature is a result of the balance between heat production and heat loss. Exercising rats increase their heat production in response to an increase in metabolic rate (1, 2) that is counteracted by activation of heat loss mechanisms in order to avoid high core temperatures that would limit exercise performance (3-7).
It has already been established that the hypothalamus is the main locus for body temperature regulation including control of heat dissipation mechanisms (8, 9). Hypothalamic areas modulate the rate of heat transfer from body core to the surface through sympathetic manipulation of skin blood flow. The rat tail dissipates an equivalent of 25% of resting heat production (10) and tail skin vasodilation is the primary mechanism of heat loss during exercise (11).

Cholinergic synapses in the brain have been reported to be involved in heat loss mechanisms during rest and exercise. In resting rats, central injection of cholinergic agonists produces hypothermia due to increased heat loss (12, 13). Furthermore, Rodrigues et al. (14) reported that cholinergic stimulation attenuated the exercise-induced increase in colonic temperature. Since the metabolic rate was not altered in Phy-treated rats, it is reasonable to assume that intracerebroventricular injection of Phy enhanced the heat loss mechanisms during exercise in these animals. However, there is no available data in the literature regarding the alterations in skin blood flow during exercise following central injection of Phy.

During exercise, mean arterial pressure and heart rate increase simultaneously to match tissue metabolic demands. This cardiovascular response, in addition to the thermoregulatory reflex drive, is also involved in cutaneous blood flow regulation (15). In fact, the tail receives a high percentage of the cardiac output of resting rats (16). Recently, Zhang et al. (17) reported the interaction of systemic arterial pressure regulation and heat loss mechanisms in anesthetized rats. The incremental changes in the carotid-sinus baroreceptor pressure increased the tail temperature, leading to reductions in body core temperature. Other authors (18, 19) have also demonstrated the regulation of skin blood flow by non-thermoregulatory stimulus.

There is evidence that the central cholinergic synapses also play an important role in the neural control of cardiovascular function. Central administration of cholinergic agonists, including injections into the lateral ventricles, raises mean arterial pressure in resting rats (20-23). This pressure response is mediated through an increase in sympathetic nervous system activity (20, 24, 25). However, there are no published reports of the effects of central cholinergic stimulation on cardiovascular response during dynamic exercise. Therefore, the purpose of the present study was to investigate whether intracerebroventricular injection of Phy increases tail heat loss in running rats and if this thermoregulatory response involves an increase in blood pressure.

MATERIAL AND METHODS

Animals

Adult male Wistar rats weighing 250-300 g (11-12 wk old) were used in all experiments. Rats were housed in individual cages under controlled light (05:00 – 19:00 h) and temperature (24 ± 1°
C) conditions, with water and rat chow provided ad libitum. All proposed experimental procedures were approved by the Ethical Committee of the Federal University of Minas Gerais for the Care and Use of Laboratory Animals.

The animals were anesthetized with 2.5% tribromoethanol (Aldrich, Milwaukee, WI) (300 mg/kg body wt ip) and fixed to a stereotaxic apparatus. A stainless steel guide cannula (22 gauge and 16.0 mm in length) was implanted into the right lateral cerebral ventricle according to a previously described technique (1, 7, 14, 26, 27). A pressure drop in the saline-filled manometer attached to the cannula indicated that the tip of the guide cannula had been correctly positioned in the ventricular space (28). The rats were given at least 3 days to recover from surgery and were then gradually introduced to exercise on a treadmill for small animals (Columbus Instruments, Columbus, OH) by running them at a constant speed of 18 m/min and 5% inclination for 5 min during 3 consecutive days.

Arterial cannulation and intraperitoneal implantation of thermal sensor

Following the last of these introductory exercise sessions, the rats were implanted with a catheter for measurement of systemic pulse pressure. Under 2.5% tribromoethanol anesthesia (300 mg/kg body wt ip), a piece of polyethylene tubing (PE10, Becton Dickinson, Franklin Lakes, NJ) filled with heparin in normal saline was inserted into the descending aorta via the left common carotid artery. The free ending of the polyethylene tubing was tunneled subcutaneously and exteriorized at the cervical dorsal area.

Temperature sensors (TR3000-XM-FM, Vital View Mini-Mitter, Sunriver, OR, USA) were implanted into the peritoneal cavity through a small incision in the linea alba. The rats were given two days to recover from this surgery. On the first day of experiments, the rats had already recovered the preoperative body weight (262 ± 4 g preoperative vs. 261 ± 5 g first experiment; \( P > 0.05 \)) (29). Furthermore, time to fatigue in the Sal experimental protocol was consistent with our previous study where the animals were allowed to recover for 5 days after intraperitoneal thermal sensor implantation (6).

Experimental protocol

On the day in which the experiments were carried out, the rats were placed in an environmental chamber (Russells Technical Products WMD 1150-5, Holland, MI), a thermocouple (YSI Inc., Dayton, OH) was fixed to the tail with tape and the arterial cannula was connected to a pressure transducer (Biopac, Santa Barbara, CA). Ambient temperature was maintained constant at 23°C and relative humidity at 60%. The rats were allowed to rest in the treadmill chamber for 30 minutes. After resting, a 30-gauge, 16.3 mm long injection needle connected to a Hamilton syringe was introduced via the guide cannula into the right lateral cerebral ventricle of the animals. The rats were randomly selected to receive an intracerebroventricular injection of 2.0 \( \mu \)L of 0.15 M NaCl solution (Sal) or 5 x 10^{-3}M physostigmine (Phy; Eserine, Sigma Chemical, St. Louis, MO). Immediately after the injections, the animals were placed inside the treadmill chamber and submitted to exercise at 24 m/min and 5% inclination until the onset of fatigue. This exercise intensity corresponds to approximately 80% \( VO_{2\text{max}} \) of untrained rats (2). Fatigue was determined by failure of the rats to keep pace with the treadmill (1, 7, 26, 27). The rats were submitted to both Phy and Sal experimental conditions with an interval of at least two days between each experiment. All experiments were performed between 9:00 AM and 4:00 PM.

Control experiments were carried out in resting rats. The animals were allowed to rest in their home cages for 60 min in the experimental room. After this stabilization period, the rats received
an intracerebroventricular injection of Phy or Sal and blood pressure and tail temperature were measured during additional 60 min. Ambient temperature was maintained at 26 ± 1° C.

**Measures**

Time to fatigue (TTF) was measured from the start of exercise until the onset of fatigue. Intraperitoneal temperature was established as core body temperature (T\textsubscript{b}) index and was measured by telemetry. Tail skin temperature (T\textsubscript{tail}), an index of heat loss responses (11), was measured on the lateral surface ~2 cm from the base of the tail (30) using a thermocouple. T\textsubscript{b} values were recorded every 15 seconds, while T\textsubscript{tail} and the temperature inside the treadmill chamber were measured every minute during rest or exercise. Cardiovascular measurements were continuously recorded throughout the experiments.

**Calculations**

Heat storage rate (HSR; J/min) was calculated (8) as HSR = (ΔT\textsubscript{b})⋅m⋅c / TTF, where ΔT\textsubscript{b} is the change in body temperature, m is body weight in grams and c is the specific heat of the body tissues (3.47 J⋅g\textsuperscript{-1}⋅° C\textsuperscript{-1}). Systolic arterial pressure (SAP), diastolic arterial pressure (DAP) and heart rate (HR) measurements were taken from systemic pulse pressure recordings. Mean arterial pressure (MAP) was calculated as DAP + 1/3⋅(SAP – DAP).

**Verification of the position of brain cannulas**

At the end of each experiment, rats were deeply anesthetized with i.p. pentobarbital sodium (100 mg/kg body wt) and were perfused with 0.9% NaCl followed by 10% formalin solution. The brain was removed and stored in formalin solution. After a few days, the brain tissues were frozen at -13° C and cut into 30 μm slices on a cryostat microtome (Microm, Riverstone, NSW). Brain slices were examined under a light microscope to check the injection site.

**Statistical analysis**

Data are expressed as means ± SE. Mean values from each experimental condition were evaluated by a subdivided parcel analysis of variance followed by the least significant difference test. Paired Student’s t-tests were used to compare total exercise time and heat storage rate between the two experiments. The integrated areas under the T\textsubscript{tail} and the SAP curves (AUC) were calculated by the trapezoidal rule. The correlations between AUC T\textsubscript{tail} and AUC SAP and between ΔDAP until fatigue and TTF were assessed using Pearson’s coefficient. Significance level was set at P < 0.05.

**RESULTS**

As illustrated in Fig. 1A, B and C, intracerebroventricular injection of Phy (n = 8) into resting animals induced an increase in MAP, SAP and DAP relative to those injected with Sal (n = 8). The effects of Phy injection on blood pressure of resting rats were already observed at 3 min (135 ± 4 mmHg Phy vs. 120 ± 3 mmHg Sal, MAP, P < 0.05; 152 ± 5 mmHg Phy vs. 130 ± 3 mmHg Sal, SAP, P < 0.05; 126 ± 3 mmHg Phy vs. 107 ± 5 mmHg Sal, DAP, P < 0.05) and peaked at 10 min, returning to the basal value within 20 min of injection. This increase in blood
Fig. 1. Temporal profiles of A: mean arterial pressure (MAP), B: systolic arterial pressure (SAP), C: diastolic arterial pressure (DAP), and D: heart rate (HR) before and after intracerebroventricular injection of physostigmine (Phy; 5 x 10^{-3} M, n = 8) or 0.15 M NaCl (Sal, n = 8) in resting rats. Values are means ± SE. *P < 0.05 compared with corresponding basal values (time 0). *P < 0.05 compared with control condition.
pressure was followed by a decrease in HR at 7 min (341 ± 12 bpm Phy vs. 382 ± 18 bpm Sal; P < 0.05), that persisted until 14 min after injection (Fig. 1D).

As illustrated in Fig. 2A and B, exercise induced a rapid increase in MAP and SAP both in Sal (n = 7) and Phy experimental conditions (n = 7). However, intracerebroventricular injection of Phy increased the exercise-induced pressure response when compared with the Sal trial. The differences between the two conditions were already observed at 1 min (141 ± 6 mmHg Phy vs. 113 ± 3 mmHg Sal, MAP, P < 0.05; 158 ± 6 mmHg Phy vs. 127 ± 4 mmHg Sal, SAP, P < 0.05) and these differences persisted until 17 min following initiation of exercise (139 ± 4 mmHg Phy vs. 122 ± 3 mmHg Sal, MAP, P < 0.05; 155 ± 5 mmHg Phy vs. 137 ± 5 mmHg Sal, SAP, P < 0.05). At the fatigue point, rats showed a higher MAP during the Phy than during the Sal experiment (135 ± 7 mmHg Phy vs. 121 ± 4 mmHg Sal; P < 0.05). There was no difference in SAP between the two test conditions at this point (149 ± 8 mmHg Phy vs. 136 ± 6 mmHg Sal). On the other hand, the exercise induced only a small and transient increase in DAP during the Sal trial (Fig. 2C). DAP was significantly higher in the Phy compared to the Sal experiment and there were also significant differences between the two trials from 1 min (126 ± 5 mmHg Phy vs. 101 ± 1 mmHg Sal; P < 0.05) until fatigue point (118 ± 5 mmHg Phy vs. 103 ± 4 mmHg Sal; P < 0.05).

As shown in Fig. 2D, treadmill exercise elevated HR throughout the experimental period in both Sal (n = 7) and Phy trials (n = 7). However, the Phy injection attenuated the exercise-induced increase in HR, and there were statistically significant differences between the two experimental conditions from 6 min (465 ± 14 bpm Phy vs. 517 ± 11 bpm Sal; P < 0.05) to 8 min following initiation of exercise (473 ± 19 bpm Phy vs. 518 ± 11 bpm Sal; P < 0.05).

In resting rats, Phy-induced pressure response was also followed by an increase in T_tail (n = 12; Fig. 3A). The differences between treatments were observed at 7 min (29.2 ± 0.5° C Phy vs. 27.5 ± 0.4° C Sal; P < 0.05) and remained different until 30 min. Exercise induced an increase in T_tail in both trials. However, at 5 min of exercise the Phy injection had already induced a significant increase in T_tail that was more intense than the increase achieved following the Sal injection, which was only achieved following 7 min of exercise. The differences in T_tail between the two trials were already present at 5 min (26.4 ± 1.0° C Phy vs. 23.8 ± 0.5° C Sal; P < 0.05) and persisted until 11 min (30.6 ± 0.3° C Phy vs. 28.5 ± 0.7° C Sal; P < 0.05). We also observed a close correlation between T_tail and SAP both in resting (r = 0.87, P < 0.001; Fig. 4A) and exercising rats (r = 0.83, P < 0.001; Fig. 4B).

As shown in Fig. 3C, T_b in the Sal trial (n = 8) was already significantly elevated 3 min after exercise had started (37.7 ± 0.2° C at 3 min vs. 37.5 ± 0.2° C resting value; P < 0.05). However, during the Phy trial (n = 8), this elevation in T_b was only observed following 15 min of exercise (37.8 ± 0.2° C at 15 min vs. 37.6 ± 0.2° C resting value; P < 0.05). From the 12th min after exercise had started until fatigue, T_b was significantly lower in the Phy trial than in the Sal trial. The highest difference between the two trials occurred at the fatigue point.
Fig. 2. Temporal profiles of A: mean arterial pressure (MAP), B: systolic arterial pressure (SAP), C: diastolic arterial pressure (DAP), and D: heart rate (HR) before and after intracerebroventricular injection of physostigmine (Phy; $5 \times 10^{-3}$ M, $n = 7$) or 0.15 M NaCl (Sal, $n = 7$) in exercising rats. Values are means ± SE. Time to fatigue is indicated by the horizontal bars at the bottom. $+ P < 0.05$ compared with corresponding basal values (time 0).

* $P < 0.05$ compared with control condition.
Heat storage rate was 61% lower following the Phy injection compared to that recorded following the Sal injection (15.4 ± 11.7 J/min Phy vs. 38.7 ± 12.1 J/min Sal; *P* < 0.05).

Intracerebroventricular injection of Phy (*n* = 8) failed to induce any difference in exercise performance compared with Sal treatment (*n* = 8) when performance was measured by time to fatigue (26.3 ± 3.8 min Phy vs. 27.8 ± 5.4 min Sal; *P* = 0.72). ΔDAP until fatigue was closely correlated to exercise performance after the Phy injection (*r* = -0.92; *P* < 0.01) and, in contrast, there was no significant correlation during the Sal trial (*r* = -0.08; *P* = 0.87; Fig. 5).

(37.9 ± 0.2°C Phy vs. 38.5 ± 0.4°C Sal; *P* < 0.05).
Fig. 4. Correlation between the integrated area under systolic arterial pressure curve (SAP AUC) and the integrated area tail skin temperature curve (T_{tail} AUC) during the first 30 min of resting (A; n = 7) and the first 20 min of running (B; n = 6) in rats after intracerebroventricular injection of physostigmine (Phy; 5 × 10^{-3} M; black circle) or 0.15 M NaCl (Sal; white circle).

Fig. 5. Correlation between the alterations on diastolic arterial pressure until fatigue (ΔDAP) and total time to fatigue (TTF) after intracerebroventricular injection of physostigmine (Phy; 5 × 10^{-3} M; n = 7; black circle) or 0.15 M NaCl (Sal; n = 7; white circle) in exercising rats.
DISCUSSION

The main finding of this study was that the increase in $T_{\text{tail}}$ was preceded by a rise in mean arterial pressure, suggesting that stimulation of cholinergic synapses modulates heat loss during exercise mainly through alterations in blood pressure that were unrelated to alterations in $T_b$. After the Phy injection, mean arterial pressure increased immediately after the onset of exercise, followed by an increase in $T_{\text{tail}}$ at 5 minutes, which in turn resulted in attenuation of the exercise-induced increase in $T_b$ at 12 minutes compared with control conditions. Despite the differences in cardiovascular and thermoregulatory adjustments, exercise performance was not altered by Phy injection.

Cardiovascular adjustments occurred immediately after the onset of exercise in both trials. In our controls rats, the rise in $T_b$ preceded the increase in $T_{\text{tail}}$. $T_b$ had already increased 3 min after the onset of exercise and this was followed by increases in $T_{\text{tail}}$ 7 min after exercise had started (Fig. 3, B and C). This result is in accordance with reports from other authors suggesting that body temperature is the main modulator of skin blood flow (31). On the other hand, during the Phy test, the rise in $T_{\text{tail}}$ preceded the increase in $T_b$. $T_{\text{tail}}$ increased 5 min after initiation of exercise, whereas $T_b$ only increased following 15 min of exercise (Fig. 3, B and C). These data suggest that the increase in tail blood flow after the Phy injection was not determined by the thermoregulatory reflex. During the Phy trial heat loss probably increased due to increases in exercise-induced pressure response. In fact, $T_{\text{tail}}$ was closely related to the increase in systolic blood pressure. Some experimental models using anesthetized or conscious resting rats demonstrated that skin and body temperature are modulated by the arterial baroreceptor reflex (17, 19). Zhang et al. (17) reported that incremental changes in carotid-sinus baroreceptor pressure resulted in a rise in tail temperature, which in turn led to reductions in systemic arterial pressure and body temperature. O’Leary and Johnson (19) also showed that an increase in mean arterial pressure as a result of phenylephrine injection caused vasodilation of the tail in normothermic rats. The rise in heat loss mechanisms after cholinergic central stimulation was reproduced in resting rats and it was related to cardiovascular adjustments. The maximal increase in MAP induced by Phy injection compared to Sal trial was not different between the resting and exercising rats (~30 mmHg in both groups). These data suggest that the effects of Phy injection on MAP are additive to those induced by the central command of exercise.

Intracerebroventricular injection of Phy increased the exercise-induced pressure response (Fig. 2A, B and C). These data provide evidence that cholinergic neurotransmission is involved in the central regulation of blood pressure in exercising rats. Intracerebroventricular injections of Phy and other cholinergic agonists, such as choline or neostigmine, have been shown to raise arterial blood pressure in conscious rats (21-23, 32). However, to our knowledge,
this is the first study to evaluate the effects of central cholinergic stimulation on the cardiovascular responses of rats to exercise.

Pharmacological and immunohistochemical studies have demonstrated that cholinergic neurons in the hypothalamus (21, 33) and ventrolateral medulla (34) mediate the pressure response to cholinesterase inhibitors in rats. In addition, since prior intracerebroventricular injection of atropine prevented the pressure response following injection of cholinergic agonists, it is likely that the response is mediated by central muscarinic receptor on neurons involved in cardiovascular function (20, 21, 23, 32, 33). Peripherally, the rise in arterial blood pressure is mediated via an increase in ganglionic sympathetic outflow (25), in cardiac sympathetic activity (24) and in sympathoadrenal activity (20). In agreement with these data, intravenous injection of the α-adrenergic receptor blocking agent phentolamine significantly attenuated the pressure response to central cholinergic stimulation (20, 32). Another mechanism involved in cardiovascular responses to intracerebroventricular injection of cholinergic agonists in resting rats is the increase in circulating arginine-vasopressin (AVP) (20, 23). An increase in cholinergic transmission to AVP-secreting neurons produces an elevation in plasma AVP concentrations greater than the values required to elicit an effect on pressure (35). It has also been reported that intravenous injection of AVP-specific vascular receptor antagonists attenuated the pressure response to centrally administered cholinomimetics (20, 32).

According to the literature, we can assume that Phy effects were centrally mediated, since autonomic and cardiovascular responses to intravenous injection of Phy were prevented by atropine sulfate but not by methylatropine, which is not able to cross the blood-brain barrier (25, 36). Moreover, in order to minimize any possible peripheral action, Phy was injected in a volume of 2.0 μL, which corresponds to approximately 5% of the average volume of the lateral ventricle (37).

Phy injection attenuated the exercise-induced elevation in HR (Fig. 2D). This response possibly occurred due to the higher blood pressure observed following the Phy injection. There is some evidence that during dynamic exercise the baroreflex is adjusted to operate at higher blood pressure levels, thereby allowing simultaneous increases in arterial pressure and HR (38, 39). However, if blood pressure increases above this limit, the rise is counteracted by baroreflex (40, 41). According to this concept, in the present experiments Phy injection attenuated the exercise-induced increases in HR 5 min after the onset of exercise, while the increases in arterial pressure occurred immediately after exercise began. Therefore, as observed in resting rats, the Phy-induced response of HR during exercise appears to have been brought about by a rise in blood pressure greater than those values observed during the Sal trial. These findings provide evidence that arterial baroreflex is maintained during exercise to oppose an exarcebatted rise in blood pressure.

The Phy-injected rats presented lower $T_b$ during exercise compared with the rats submitted to the Sal trial (Fig. 3C). Central administration of cholinergic
agonists has been shown to decrease body temperature in resting rats through an increased cutaneous blood flow (12, 13) and these data suggest that this may also occur during exercise. Rodrigues et al. (14) observed attenuation in the exercise-induced increase in colonic temperature after central cholinergic stimulation. Since heat production evaluated by the metabolic rate remained unaltered following the Phy injection, it is reasonable to assume that activation of the central cholinergic synapses decreased body temperature through an increase in heat loss mechanisms (14). Our data show that the tail skin temperature was higher in those animals submitted to Phy treatment between 5 minutes and 11 minutes following the onset of exercise (Fig. 2B). Increases in tail temperature are related to higher cutaneous blood flow (30, 31, 42), which represents the major heat loss mechanism during exercise (11), since rats are unable to spread saliva on their body while running.

It is generally accepted that the exercise-induced increase in skin blood flow is caused by a dilatation of local vessels (11). However, our data suggest that the rise in skin temperature probably occurred irrespective of cutaneous vasodilation, as reflected by the elevated values of diastolic arterial pressure. Central cholinergic agonists activate the sympathetic nervous system (20, 24, 25) and elevated plasma catecholamines concentrations (20) cause tail skin vasoconstriction through $\alpha$-adrenoceptors (42). We suggest that the higher heat loss following the Phy injection reflects the heart’s ability to increase cardiac output despite the increase in peripheral resistance, leading to an increase in skin blood flow.

During exercise, elevated body temperature is associated with cessation of exercise in rodents (3-7, 43) and in humans (44, 45). Other authors have suggested that the drive to exercise may be diminished by hyperthermia (46). A previous study carried out in our laboratory showed that the duration of exercise in running rats was best correlated with the heat storage rate (6). Furthermore, there is evidence of the involvement of central cholinergic synapses on the development of fatigue since intracerebroventricular injection of atropine drastically reduces exercise tolerance in running rats (26, 27). However, we showed that a lower heat storage rate in our experimental protocol was not associated with any improvement in time to fatigue in Phy-injected rats. Our present results involving cholinergic stimulation are in agreement with a previous report. Rodrigues et al. (14) have also reported that intracerebroventricular injection of $5 \times 10^{-3}$ M Phy, in the same concentration used in the present experiment, did not alter exercise performance at 20 m/min and 5% inclination. It has been suggested that fatigue is a protection mechanism that involves the integration of different physiological systems, control of body temperature being just one of these systems (47). However, despite the protective effect that lower body heat should provide against thermal damage, time to fatigue was not extended in Phy-treated animals. The exercise-induced increase in diastolic blood pressure provides a considerable challenge to cardiovascular homeostasis and it
might be associated to exercise intolerance (48). This hypothesis is in agreement with our observation that the increase in diastolic blood pressure in Phy-treated rats was inversely related to time to fatigue (Fig. 5).

We conclude that stimulation of the central cholinergic synapses during dynamic exercise increased mean arterial pressure, which increased tail cutaneous blood flow, leading to heat loss without affecting running performance.

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