Mechanisms for the control of respiratory evaporative heat loss in panting animals

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Robertshaw, David. Mechanisms for the control of respiratory evaporative heat loss in panting animals. J Appl Physiol 101: 664–668, 2006. First published May 4, 2006; doi:10.1152/japplphysiol.01380.2005.—Panting is a controlled increase in respiratory frequency accompanied by a decrease in tidal volume, the purpose of which is to increase ventilation of the upper respiratory tract, preserve alveolar ventilation, and thereby elevate evaporative heat loss. The increased energy cost of panting is offset by reducing the metabolism of nonrespiratory muscles. The panting mechanism tends to be important in smaller mammalian species and in larger species is supplemented by sweating. At elevated respiratory frequencies and body temperatures alveolar hyperventilation begins to develop but is accompanied by a decline in the control of carbon dioxide partial pressure in arterial blood, probably through central chemoreceptors. Most heat exchange takes place at the nasal epithelial lining, and venous drainage can be directed to a special network of arteries at the base of the brain whereby countercurrent heat transfer can occur, which results in selective brain cooling. Such a phenomenon has also been suggested in nonpanting species, including humans, and although originally thought to be a mechanism for protecting the thermally vulnerable brain is now considered to be one of the thermoregulatory reflexes whereby respiratory evaporation can be closely controlled in the interests of thermal homeostasis.

heat exchange; body temperature; brain cooling

LOSS OF HEAT BY UTILIZATION of the latent heat of vaporization of water is an important component of the spectrum of physiological strategies available to species that control their body temperature in the face of variable thermal loads. The best methods known of increasing evaporative heat loss are panting, sweating and saliva spreading. This review will focus on panting as a thermolytic strategy. A lot of the research into panting occurred before 1975, with a revival of interest after 1995 that accompanied technical advances in both telemetry and data collection and analysis.

RESPIRATORY EVAPORATION

The function of the respiratory system is often viewed as being related primarily to gas exchange, and other functions such as acid-base control, phonation, and thermoregulation are often overlooked. The evolution and success of endothermy allowed the control of body temperature in cold environments to be independent of the external thermal environment but required the development of appropriate heat loss strategies in hot environments. Because respiratory gas exchange requires the humidification of inspired air, an increase in respiratory ventilation will also elevate respiratory evaporation as long as respiratory dehumidification and cooling does not occur (35). Thus evaporative heat loss by panting would be a relatively simple function to accommodate. In terms of evaporative heat loss of terrestrial endotherms it is possible that, from an evolutionary standpoint, either panting or saliva spreading may have been the first heat-loss mechanisms to emerge that utilized the latent heat of vaporization of water to increase heat loss. However, alveolar exchange of oxygen and carbon dioxide would also need to be controlled to preserve the requirements for gas exchange and pH homeostasis. Ideally the increase in ventilation should be confined therefore to the dead space where humidification takes place and should not compromise alveolar gas exchange, a requirement that can be achieved by an increase in respiratory frequency with a proportionate decrease in tidal volume (15), which, in fact, becomes a definition of panting (Fig. 1). If one reviews the extent to which panting is used as an evaporative heat-loss mechanism in the animal kingdom, it is found to occur in some reptiles, in birds, and in many mammalian species. There is a difficulty in determining from visual measurement of respiratory frequency alone that panting, as defined, truly exists. Only the technically difficult approach used in Fig. 1 whereby respiratory ventilation is partitioned into its alveolar and dead space components or direct measurement of respiratory water loss will truly satisfy the conclusion that panting is an evaporative heat-loss mechanism. However, careful visual observa-
tion as to the changes in the depth of respiration will often discriminate between increases associated with panting, exercise, or the hypermetabolism of hyperthermia.

The most notable nonpanting species in mammalian terrestrial species (marine mammals for obvious reasons relating to the physics of heat transfer in water need no such mechanism) are elephants and humans. The elephant has no identified evaporative heat-loss mechanisms that can be activated as part of thermal homeostasis; the elephant simply stores heat during the day and dissipates it at night (16). Primates, other than humans, demonstrate panting to a limited extent (17). In humans no respiratory response to heat exposure falls within the definition of panting.

When a comparison is made of the relative efficacy of the two main modes of evaporative heat loss, i.e., panting and sweating, it is often concluded that, although the movement of air across the moist surfaces of the turbinate bones in the nasal cavity assists in the evaporation of water in a way not generally available to the skin surface of sweating species, the heat loss of panting is limited by the increase in heat production of the muscles of respiration. However, the energy cost of panting, when measured as the change in total oxygen consumption between the thermonutral and thermolytic zones, is found to be zero (12, 14), truly an efficiency of 100%! The solution to this paradox was revealed by Hales (11), who compared the distribution of cardiac output before and during heat exposure and demonstrated that an increase in blood flow to the respiratory muscles during panting was compensated by a reduction in flow to some of the nonrespiratory muscles, leading to the conclusion that, if blood flow and oxygen consumption are matched, the metabolism of respiratory muscle may indeed be elevated during panting but that of other muscles would be equally depressed. An additional feature that contributes to the high energetic efficiency of panting is that the maximum panting frequency occurs at the resonant frequency of the respiratory system (5). Because the maximal panting frequency will therefore be inversely related to body size, this may explain the observation that in a range of bovid animals of different adult body size that use both panting and sweating, the larger species utilize sweating more than panting as a strategy for increasing evaporative heat loss (33). One may speculate, therefore, that if panting represents a primitive form of evaporative heat loss of the early mammals, which were small, the subsequent evolution of larger species necessitated the development of a supplementary form of evaporative heat loss, namely sweating. Evaporative heat loss of the kangaroo is unique in that all three strategies for increasing evaporation are used: saliva spreading and panting at rest and sweating during exercise (7).

THERMOLYSIS AND GAS EXCHANGE

The water and heat loss of respiration is dependent on both ambient humidity and minute ventilation. Under cold conditions, both convective and evaporative respiratory heat loss are largely uncontrolled and may need to be countered by an elevation of both metabolism and respiratory ventilation in the interests of thermal homeostasis. Thus high-altitude mountaineers in a hypoxic environment with an increased respiratory ventilation and inhaling air of low humidity are faced with the dual threats of significant water and heat loss, which may contribute to the risk of both dehydration and hypothermia. However, a reduction in minute ventilation upon exposure to cold temperatures will lead to hypercapnia and increased oxygen extraction from inspired air (37). A detailed study on the effects of cold exposure on the respiration of cattle demonstrated a reduction in total respiratory heat loss, expressed as a percentage of metabolic heat production, that was inversely proportional to ambient temperature and accompanied by arterial hypercapnia and hypoxemia (8). The respiratory system can be seen therefore to be responsive to, and be part of, a cascade of reactions to the thermal environment with a continuous extending from hypoventilation in the cold to hyperventilation in the heat.

Although the increases in ventilation in panting are largely confined to the dead space, there is inevitably a small but detectable increase in alveolar ventilation and a consequent hypocapnia; dead space ventilation cannot be physically separated from alveolar ventilation, and diffusive mixing of gases is bound to occur. If evaporative heat loss is inadequate and body temperature rises, there is a change in the pattern of respiration such that tidal volume increases and respiratory frequency decreases and panting changes from closed- to open-mouth respiration. The consequent alveolar hyperventilation leads to a progressive development of profound respiratory alkalosis (15). Thus two forms of panting have been described: one without any major change in alveolar ventilation and the other initiated as core temperature rises in which there is significant alveolar hyperventilation (Fig. 1). One explanation for the hyperthermic hyperventilation might be that the resistance to nasal airflow increases to such an extent that a change to a lower resistance mode of ventilation, i.e., through the mouth, would become necessary. The elegant model of respiratory air flow on the basis of anatomical measurements of the turbinates devised by Schroter and Watkins in 1989 (36) indicates that the Reynolds numbers in the gaps are low enough to predict that even at high ventilation rates air flow will always be laminar and airway resistance is unlikely to be a factor in the transfer to open-mouth panting.
Exercise can lead to hyperventilation in many species, and the question has been posed that, in addition to the anticipatory hyperventilation that occurs in humans before exercise, the respiratory stimulus for hyperventilation may be multifactorial, originating from exercising muscles themselves and thus be a function of exercise intensity or from a rise in blood lactate concentration, or be part of the thermoregulatory drive. In studies conducted on sheep, which is a panting species, Entin et al. in 1998 (10) identified body temperature as the only significant variable in exercise-induced hyperventilation. In both passive or exercise hyperpnea, thermoregulatory drive appears to be the common modality. However, the hyperthermic hypocapnia may suppress the chemoreceptor drive to respiration. This apparent conflict has been examined in three studies. Hales et al. (13) denervated the carotid chemoreceptors and found no evidence of any significant role for the peripheral chemoreceptors in the ventilatory response to heating in sheep. Maskrey et al. (28) enriched the inspired air of sheep with carbon dioxide to maintain normocapnia and found a reduction in panting frequency and an increase in tidal volume, which suggested that the threshold chemoreceptor response to carbon dioxide was lowered by hyperthermia. Entin et al. (9) also concluded that as body temperature rises there is a downward shift in the control level or “set point” of arterial PCO₂ that offsets any apparent homeostatic conflict between thermolysis and pH control. Such an adjustment of either respiratory threshold or sensitivity to PCO₂ may be significant in species that rely on panting as their main means of evaporative heat loss in that it removes any possible “braking” effect of hypocapnia on thermolysis.

STUDIES ON HUMANS

If it is accepted that there is little or no evidence for panting as a thermoregulatory mechanism in humans then the effects of either passive or exercise-induced hyperthermia on respiration should separate the uniquely respiratory consequences of hyperthermia from those related to temperature regulation. The work of Cabanac and White (4) and White and Cabanac (39) showed that, contrary to the situation in panting species where there is no clear threshold body temperature for the onset of hyperventilation (10), a change in respiration occurs only when core body temperature has risen to a threshold value; once a threshold temperature is exceeded, hyperventilation ensues. This threshold is significantly higher than the threshold temperatures for both the onset of sweating and increase in cutaneous blood flow, demonstrating that the respiratory response to hyperthermia cannot be analogous to the panting response and be part of the usual group of thermolytic reflexes (38). The hyperthermic hyperventilation observed in panting species, such as dog and sheep, may be analogous therefore to that observed in nonpanting humans and therefore be a fundamental property of the respiratory system of all species of bird and mammal whether or not they use panting as a heat-loss mechanism. The feature that is common to humans and nonpanting animals could be the relationship between respiration and selective brain cooling.

SELECTIVE BRAIN COOLING

Panting involves inhalation of air through the nose, and in most species the mouth is closed. Although it is common knowledge that the dog shows open-mouth panting, inspired air enters the nasal cavity and exits through the mouth (34). Humidification of the inspired air therefore occurs from fluid secreted onto the surface of the nasal epithelium (3). An increase in blood flow to the nasal mucosa provides the necessary heat for evaporation, and the venous blood draining the turbinates is thereby cooled. The subsequent distribution of the venous blood is variable; it may enter either the angularis occuli vein and then to the cranial cavernous sinuses before finally entering the jugular vein, or alternatively it may enter the facial vein and thence to the jugular vein. The pathway of direction of flow is under sympathetic neural control of the muscular coat of either vein, which act as sphincters and redirect the flow along one route or the other (21). The angularis occuli possesses α-adrenergic receptors, stimulation of which leads to vasoconstriction, whereas the receptors of the facial vein are venodilators of the β-adrenergic variety (21). Under generalized sympathetic stimulation, they are essentially antagonistic in their function. Blood entering the cranial cavernous sinuses surrounds the arterial blood supply to the base of the brain, which, in many panting species, consists of a network of vessels known as the carotid rete (6). Such an arrangement is an efficient countercurrent heat exchanger that allows the arterial supply to the brain to be cooled (2). Although the existence of brain cooling has been demonstrated in many species that possess a carotid rete, there is no such structure in humans, rabbits, and horses. However, the internal carotid artery of these species traverses the cranial cavernous sinus, and there is a potential for countercurrent cooling although the absence of a vascular network such as the carotid rete would be expected to minimize the efficacy of heat transfer. McConaghy et al. (29) studied selective brain cooling of the horse, a species that has no carotid rete and may not even be a panting animal. They clearly demonstrated the cooling of blood in the upper respiratory tract and selective brain cooling during both heat exposure and exercise. Although humans do not pant, sweating from the head is particularly well developed during both cutaneous and respiratory evaporative heat loss could contribute to selective brain cooling. Using tympanic membrane temperature as an index of brain temperature and esophageal temperature as an index of arterial temperature Cabanac and White (4) and White and Cabanac (39) have provided evidence of selective brain cooling in humans during both passive and exercise hyperthermia. The use of tympanic membrane temperature as an index of brain temperature has been questioned until some direct measurements of intracranial and subdural temperatures were made by Mariak and colleagues (27) on patients undergoing surgery for subarachnoid hemorrhage. They confirmed not only the relationship between intracranial temperature and tympanic membrane temperature (26) but also the link between upper respiratory cooling and selective brain cooling (27). They also concluded that, on the basis of the speed of response to respiratory evaporation, the transfer of heat from the nasal epithelium was by convective and not conductive mechanisms.

The identification of selective brain cooling in both panting and nonpanting animals as well as those species that do not possess a carotid rete was initially perceived as a mechanism for the protection of a thermally vulnerable organ. However, this conclusion may be an oversimplification, and studies on free-ranging animals demonstrate significant variability in the

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extent to which it occurs; to quote from Jessen and Kuhnen (19), there is a “...need for caution in assigning a specific function to the selective cooling mechanism.” However, it may be possible in all the apparent contradictory conclusions to generate a possible working hypothesis. The magnitude of selective brain cooling will depend on 1) the degree of nasal cooling, and in this context the skin cooling by sweating in humans may contribute if cutaneous venous drainage is allowed to enter the angularis occuli vein; 2) distribution not only of cool venous blood to the cranial cavernous sinus but also the routing of arterial blood to the heat exchangers; 3) the extent to which inhaled air is taken in through either the nose or mouth; and 4) the role of selective brain cooling as a component of thermal homeostasis. The work of Jessen and colleagues has been prominent in elucidating the thermal factors that determine the initiation of selective brain cooling. The hypothalamic temperature sensors themselves are cooled and selective brain cooling would, therefore, be part of a feedback loop with brain temperature being the regulated variable. When the thermal affecter systems are partitioned into those in the brain or trunk, Kuhnen and Jessen (23, 24) concluded that cranial thermosensitivity largely determined the temperature threshold for selective brain cooling, whereas trunk temperatures influence the slope of the response above the threshold. Such a control system integrates all the thermal inputs and dampens any oscillations in respiratory evaporative heat loss.

The feedback loop for selective brain cooling will suppress panting. It has been proposed that in dehydration the enhancement of panting and the use of selective brain cooling combined with the suppression of sweating in goats (18, 32) can be viewed as a water retention mechanism in that it will conserve ~35% of the water intake (22). Brain cooling thus becomes part of the cascade of water conservation responses to dehydration that are part the hyperthermia of dehydration. The significance of open-mouthed panting and the accompanying respiratory alkalosis that occurs during hyperthermia has been a mystery in terms of understanding its role in thermoregulation. The work of Aas-Hansen et al. (1) demonstrated that during open-mouth panting in reindeer, there is a redirection of inspired air flow away from the nasal cavity to the mouth. Consequently, there could be a reduced flow of venous blood into the cranial cavernous sinus and a reduction in selective brain cooling. Accordingly brain temperature rises and removes the inhibition of respiratory evaporative heat loss, thereby assisting in total body heat dissipation.

In general, the demonstration and replication of selective brain cooling is consistent under laboratory conditions. However, nonthermal factors such as the presence of an investigator in the experimental area will suppress selective brain cooling (25). Presumably these transient periods of abandonment of selective brain cooling are mediated by a redirection of venous blood flow away from the cavernous sinus. A similar event has been observed in studies of free-ranging animals in which it is obvious that tight thermal control is not always apparent because of nonthermal factors. These may include the fright of being chased observed in wildebeest (20) or bouts of spontaneous activity reported in free-ranging springbok (30). Exercise or generalized sympathetic stimulation may therefore activate the α-adrenergic receptors of the sphincter muscle of the angularis occuli vein and divert flow into the facial vein and lead to a loss of selective brain cooling.

Although there is evidence for selective brain cooling in humans, recent observations on total brain cooling during exercise with or without hyperthermia (31) appear to negate the idea of selective brain cooling. However, the general thesis of selective brain cooling has been that the cooled area of the brain is confined to the thermosensitive regions of the hypothalamus and that generalized brain cooling may not take place.

In summary, the evolution of research into respiratory and cutaneous evaporative heat loss from the head and its relationship to selective brain cooling has moved away from the concept of the brain as being uniquely vulnerable to hyperthermia to the notion that it represents part of the control mechanism for thermal homeostasis.

REFERENCES