Panting During Hyperthermia in Humans

The hypothesis & point we are supporting:

When humans become hyperthermic they exhibit panting as a thermoregulatory response.

The hypothesis of the counter-point that we are refuting:

When humans become hyperthermic they do not pant as a thermoregulatory response.

Research outcomes that support our point argument & hypothesis:

In a study by White & Cabanac (1995) human subjects were immersed in a 41°C bath inspired minute ventilation significantly increased from 10.0 L/min to 18.6 L/min after 5.5 minutes, at the 24th minute mark it peaked at 19.3 L/min. Moreover, they showed that the respiratory response in humans have a threshold temperature above which it occurs, around 38 °C. By interpreting the data we infer that there is a clear respiratory response to the hyperthermic conditions, the body is trying to maintain homeostasis by evaporative heat loss. Tidal volume decreased within 10 minutes of immersing the subjects into the hot water bath. This corresponds with the description of thermal tachypnea. Tidal Volume then increased linearly over time showing a transition to the second stage of the response. Similarly, there was an increase in respiratory frequency which began at 13 breaths/min and then increased significantly to 21 breaths/min. Eventually the frequency of breathing declined in a linear manner which is described by the second phase of panting.

Gaudio et al. (1968) investigated the response of human subjects when exposed to 54 °C dry bulb and 40 °C wet bulb temperatures at rest. Mean minute ventilation and tidal volume rose steadily during the exposure, while the frequency of breathing did not change significantly. The
rate of change for minute ventilation is around 1 l/min for each 1 °C rise in core body temperature. Mean Pco₂ decreased from 44 ± 3 to 33 ± 4 mm Hg, whereas pH rose from 7.383 ± .017 to 7.461 ± .037. These results suggest respiratory alkalosis, and thus this response can be best described as a thermal hyperpnea or second-phase panting.

Hanson et al. (1974) performed experiments where core body temperatures were at controlled levels, and subjects breathed air at three different temperatures (10 °C, 20 °C, 30 °C). The researchers measured the respiratory heat loss due to both convection and evaporation from the upper airways. They found that the ambient temperature did not affect evaporative heat loss, while it showed a strong positive correlation with increasing core body temperature. This could be attributed to the increase in minute ventilation. On the other hand, convective heat loss increased with decreasing ambient temperature or increasing body temperature.

House & Holmes (1992) did a research where three subjects exhibited a linear increase in ventilation along with a linear decrease in end tidal pressure of carbon dioxide (PetCO₂) when immersed in a warm bath of 39.5 °C. Thermal hyperpnea, or the second phase of panting is apparent when the ratio of PetCO₂/P arterialCO₂ is reduced, which they observed in their study.

White et al. (1995) showed in another study that there is a significant increase in blood flow to the nasal mucosa during hyperthermia. This is suggestive of a conserved mechanism for heat loss among animal species.

Critique of research outcomes that refute our point argument & hypothesis:

Forster et al. (1952) investigated the onset of panting in unanesthetized cat. They found that in majority of cases the onset of panting was clearly correlated with hypothalamic temperatures. This panting response included a breathing rate > 200 / min with an open mouse
and protruded tongue. Thus, it could be argued that humans do not have a true panting response as this form of panting never occurs. However, while it is true that humans do not showcase a similar panting response, they might lose the same amount of heat from the head relative to heat production as do cats with their panting response. Quantifying heat production/heat loss in this paper could have compared it with similar studies in humans (Rasch et al., 1991), and showed that the human response is equivalent in terms of heat loss.

Wilsmore et al. (2006) Investigated ventilatory changes in heat-stressed humans with spinal cord injury. They described that the ventilatory response could not be described as rapid and shallow breathing, thus it was unlikely to be a true panting response. However, they did not directly measure tidal volume of breathing, only breathing frequency was quantified and they came to a conclusion upon this fact. They did not administer any tests or quantify changes in tidal volume, PetCO2/ParterialCO2, or ventilation volumes which correspond to the phases of thermal tachypnea and hyperpnea.

Conclusion: When humans are exposed to a hyperthermic environment evidence supports that ventilatory changes are evident, specifically changes in minute ventilation, tidal volume, PetCO2 levels, and redistribution of blood to the nasal cavity. The change in variables mentioned to support our point validate that a constant internal core temperature needs to be maintained by respiratory evaporative heat loss, while panting is evident in humans as a thermoregulatory mechanism to dissipate heat.
References:


