BPK 432: Thermophysiology 750 Word Summary

Counter-Point Argument

Humans do not selectively cool their brains when they become hyperthermic

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The hypothesis & counter-point we are supporting:
Humans do not selectively cool their brains as a protective mechanism when hyperthermic

The hypothesis & point we are refuting:
Humans are able to selectively cool their brains as a protective mechanism when hyperthermic

Research Outcomes supporting our counter-point & hypothesis:
It has been documented that hyperthermia causes tissue damage. However, according to Burger et al. (1964), different tissue types experience damage at around the same degree Celsius, particularly above 42 °C. This suggests that cooling the brain might be just as important as cooling any other organ. In other words, the brain is not specifically vulnerable to heat.

Nybo et al. (2002) demonstrated that blood temperature exiting the internal jugular vein was higher than the arterial blood measured through esophageal and aortic arch temperatures. Findings were consistent under both normothermic and hyperthermic exercise trials suggesting that the brain must have been warmer than the body core. This also eliminates the possibility of counter-current exchange as that would require a cooler blood in the veins than in the arteries. No indications of selective brain cooling were evident even though the subjects reached rather high core body temperatures - the highest individual core temperature being 40.1 °C with a corresponding jugular venous temperature of 40.4 °C.

Many studies claiming human SBC occurs used tympanic temperature as a reflection of brain temperature. In order to investigate the correlation between the two, Shiraki et al. (1988) measured brain temperatures after the implantation of thermocouples into the lateral ventricle and white matter above the ventricle. They found that ventricular and esophageal temperatures were unaffected by fanning of the face and were held at relatively constant levels. Conversely, tympanic temperature decreased during the fanning and followed forehead skin temperature with a lag. This suggests tympanic temperature is influenced by the skin temperature of the head and
largely unrelated to the temperature of the brain. This study confirms that the skin deep in the external auditory meatus and the tympanum are influenced by the temperature of the face. Therefore, evidence for selective brain cooling based on measurements of the tympanic membrane cannot be accepted.

Moreover, they show that esophageal temperature did not rise during face fanning. Had selective brain cooling occurred, the thermosensory regions in the brain (the hypothalamus) would have cooled accordingly. This, in turn, would have activated heat conserving mechanisms such as the cessation of sweating. As the changes expected to happen with selective brain cooling did not take place, they conclude that selective brain cooling does not happen in humans.

Jessen & Kuhnen (1992) used interpeak latencies (IPLs) of the acoustically evoked brain stem potentials to detect temperature changes in the brain stem following hyperthermia while either restricting or allowing heat dissipation from the face. They found no difference in brain stem T during hyperthermia with regards to whether facial sweating was or was not allowed. Therefore, evaporative heat loss by the face did not result in the selective cooling of the brain.

**Research outcomes refuting the point & hypothesis:**

According to White et al. (1995), tympanic T remains lower than esophageal T during hot bath immersion, in correlation with increased blood flow to the nasal mucosa. This suggests that an increase in respiratory heat loss might be associated with human SBC. However, in this study the authors did not quantify heat loss. It could have revealed important details about human SBC, such as that the increase in nasal mucosal blood flow might not contribute too much to evaporative heat loss, meaning the trait might be vestigial. Also, the difference between the two Ts might have been due to a latency in conductance to the tympanic membrane. Therefore, in
order to claim human SBC due to increased respiratory heat loss one needs to do a quantitative analysis to see if the heat lost can be significant.

In a study by Mariak et al. (1999), they showed that respiratory heat loss can in fact cool parts of the brain. They inserted thermocouples into the subdural space ($T_{sd}$) or close to the cribriform ($T_{cr}$) plate during an aneurysm surgery. When they induced mild hyperthermia in the patients while bypassing the upper respiratory pathways (using an endotrachial tube), they observed that all the Ts they measured increased. Upon the removal of the tube they observed that the $T_{cr}$ dropped below $T_{es}$ in 3 out of 4 cases. Importantly though, $T_{sd}$ never seemed to follow the drop in $T_{cr}$, indicating that the cooling effect respiration might have on the brain is localized close to the upper respiratory tract and it was not enough to cool other parts of the brain. Not to mention the patient where cooling did not occur at all. In conclusion, while respiration might have a local cooling effect on the brain, it is not sufficient for cooling the whole brain.

**Conclusion:**

The evidence discussed supports our hypothesis that humans do not selectively cool their brains, the evidence brought forward by studies that support the point argument show flaws with association, assumption, and method.
References:


