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REBUTTAL FROM WHITE, GREINER, AND MCDONALD

Below are our responses to two of the three main arguments presented by Nybo and Secher (7). Their third argument is addressed in our Point contribution, where it was already given that several animals demonstrate SBC without a carotid rete and consequently this countercurrent heat exchanger is not a prerequisite for SBC (1).

Comparison of cranial temperatures in anesthetized humans demonstrated an excellent relationship exists between tympanic ($T_{ty}$) and directly measured brain temperatures (3). Mariak and colleagues (3) concluded that of the externally accessible body temperatures, “$T_{ty}$ gives the best approximation of average cerebral temperature,” and this result was also supported by a separate group with direct measures of brain temperatures (6). With face fanning of normo- or hyperthermic, nonanaesthetized patients, evidence showed intracranial subdural temperature changes on the convexity of the brain were highly and significantly correlated ($r = 0.91, P < 0.05$) with $T_{ty}$ changes (5). The facts clearly illustrate $T_{ty}$ is a valid index of human intracranial temperature. It follows we agree with Nybo and Secher’s (7) view that $T_{ty}$ does not follow the external jugular temperature. This is expected for any human extracranial/thoracic temperature that remains above cranial temperatures during hyperthermia (1).

With direct measures of cribiform plate temperatures, human SBC was indisputably demonstrated with only small increases in rate and depth of breathing (4). Recent evidence (reviewed in Ref. 2) with cooling caps, enhanced upper airway cooling, and continuous ventilation of the upper airway each confirm the existence of SBC in humans.

Nybo and colleagues (7, 8) describe cranial heat balance using a black box approach. The heat balance equation they employ (10) includes variables for cranial heat production, cranial blood velocity/flow, and aortic arch to external jugular vein temperature gradients. Although this model appears to support cranial heat balance, at rest it is dependent mainly on heat loss by cranial perfusion (10); during passive or active hyperthermia it is a flagrant oversimplification. During hyperthermia, cranial thermoregulatory heat loss responses are initiated and these include eccrine sweating, cutaneous vasodilatation, and thermal hyperpnea. These thermoregulatory responses elevate cranial surface evaporative (9), convective/conductive (1), and respiratory heat loss (9). These thermoregulatory responses contribute to the centrifugal decreases of intracranial temperature from the brain ventricles to the subdural space (6). Together the evidence strongly supports that there is significant heat loss from cranial surface that needs to be considered in any cranial heat balance model and in the description of the mechanisms of SBC in hyperthermic humans.

REFERENCES


REBUTTAL FROM NYBO AND SECHER

White et al. mention three mechanisms for selective brain cooling that all represent potential routes for cerebral heat dissipation. However, heat removal through the skull is very limited and both at rest and during exercise with and without hyperthermia the main part of the metabolic heat produced in the brain is released via the venous blood leaving the brain (4, 7, 8). Therefore, the brain temperature is mainly dictated by the cerebral metabolic rate, the cerebral blood flow, and the temperature of the arterial blood. As discussed in the Counterpoint in this debate, ventilation-induced precooling of the carotid blood may increase from rest to exercise, but it should be considered that heat removed via this mechanism is restricted to $-0.1 \text{J} \cdot \text{g}^{-1} \cdot \text{min}^{-1}$ compared with a total cerebral heat production of $-0.6 \text{J} \cdot \text{g}^{-1} \cdot \text{min}^{-1}$. Furthermore, our previous evaluation of the cerebral heat balance reveals that <5% of the heat produced by the brain may be removed through the skull (7). White et al. question this assessment of the cerebral heat balance—specifically the reduction in cerebral blood flow during exercise-induced hyperthermia. Yet it has to be acknowledged that the reduction in the perfusion of the brain during exercise with heat stress has been verified by independent techniques (5, 6) and it is well known that cerebral blood flow declines when hyperventilation reduces the arterial $\text{PCO}_2$ as observed during passive and exercise-induced hyperthermia (3, 9).

When special cooling devices are applied (1) it is possible to create significant cooling of the brain, but in “natural settings,” i.e., resting or freely moving humans the rate of heat loss through the aforementioned mechanisms is limited. It would require unphysiological flow rates and/or temperature gradients.
if heat loss through the skull or via the emissary veins should have a significant effect on the brain temperature (2, 4).

So, are humans capable of demonstrating selective brain cooling? Yes—but humans can also fly in the sky. However, both situations require specialized technical support.

REFERENCES