SELECTIVE BRAIN COOLING IN HUMANS: FANCY OR FACT?

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A mechanism that selectively cools the brain during hyperthermia is a well accepted fact in animals (1). Selective brain cooling (SBC) during hyperthermia has also been proposed in humans (5, 7, 16). However, this proposal has met with considerable debate. Several authors have rejected the idea of human SBC (2, 3, 12, 13, 19) for the following reasons:

a) SBC is illogical since this mechanism removes the error signal activating the defence against hyperthermia;

b) contrary to animals humans do not pant and thus do not possess a powerful heat sink at short distance from the brain;

c) humans do not have a carotid rete, the countercurrent heat exchanger between the arterial and venous bloods flowing in and out of the brain;

d) the high and constant arterial blood flow of the brain is sufficient to cool the brain during all conditions;

e) finally the low tympanic temperature recorded in hyperthermic humans is not a sign of SBC but, rather, is the sign of contamination of tympanic temperature by a low skin temperature.

All these arguments may rejected:

a) because the argument should also apply to animals;

b) because heat loss from the head skin during exercise with moderate convection can amount to more than 100 W. The evaporative heat loss from the upper airways adds another 100 W to the cephalic heat loss (17), and that heat loss from the upper airways contributes to the cooling of the brain in the human species (20, 21);

c) because other species without rete have been shown to possess SBC. In addition SBC in humans is certainly not limited to countercurrent heat exchange between carotid and jugular, but heat-loss takes place through the calvaria;

d) because arterial cooling is certainly present at rest, but during hyperthermia each °C increase of arterial blood brings *ca.* 45 W into the brain. Rectal temperatures as high as 41,9°C in long distance runner, and 47°C in patients who survived, have been recorded, while 40,5 is considered the critical temperature for the brain.

e) because the following thermoregulatory responses correlated with tympanic temperature as well as, or better than, with esophageal temperature: skin vasomotor, sweating, heart rate, V_{O2}, behavior, acclimatization, and because a lowering of tympanic temperature could be obtained without changing the temple and skin temperature (e.g. by cooling the other side of the head, 6), thus excluding any contamination of tympanic temperature by skin temperature.

Finally, the conclusions of some recent experimental articles which apparently contradict the existence of human selective brain cooling (8, 11, 18) may be refuted: 8) because the ultrasonic recording of *angularis occuli* blood flow confirmed the inward flowing of cool blood during hyperthermia; 11) because the sensitivity of estimating brain stem temperature from changes in the speed of conduction of auditory evoked potentials was on the threshold of reliability; and 18) because several artefacts may explain the poor correlation of tympanic temperature with brain temperature obtained in a child patient.

In conclusion, there is an overwhelming evidence in favor of the existence of human selective brain cooling. This conclusion entails some

important consequences both theoretical (9) and for the following applications: prevention and treatment of heat shock, whole body hyperthermia of cancer therapy (15), and improvement of endurance performance.

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