LETTERS
Michel Cabanac

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More Comments on “Keeping a Cool Head”

To the Editor: In his April 1986 article, “Keeping a Cool Head,” Dr. Cabanac proposes that the human cavernous sinus is an arteriovenous heat exchanger that, when receiving cool facial venous blood, can keep the brain 1°C or more below trunk core temperature (Tc). To support this concept he used tympanic temperature (Tty) as an index of intracranial temperature. This use of Tty requires it to be more closely linked to brain temperature than is any index of Tc.

The only study comparing Tty to brain temperature that Cabanac cites is inconclusive because in that report (1) Tty, rectal temperature, and hypothalamic temperature were virtually identical except when a cold drink produced a sharp drop in Tty and hypothalamic temperature, while rectal temperature, a lagging index of Tc, showed a blunted and delayed drop. Cabanac states further that a number of thermoregulatory responses correlate better with Tty than with esophageal temperature. However, these responses are influenced by skin temperature as well as by Tc, and many thermophysiology— as Cabanac notes—consider Tty to be contaminated by skin temperature. He also points out that only a few millimeters separate the tympanic membrane from the internal jugular vein. This distance, however, includes the middle ear, which normally is filled with still air, an excellent thermal insulator.

A privileged status for Tty as an index of brain temperature thus has yet to be established. In fact, since most of the tympanum’s blood supply (excepting that via the carotico-tympanic artery) comes through branches of the external carotid artery, there isn’t cooling of that blood supply a more plausible explanation than selective brain cooling to account for Tty lower than Tc? Indeed, simultaneously heating part of one side of the face while cooling the corresponding contralateral area will raise Tty on the heated side while lowering Tty on the cooled side (3).

Now let us assume that each internal carotid artery passes through the cavernous sinus for 2.8 cm of its length, with a diameter of 0.5 cm and a wall thickness of 0.1 cm. If these dimensions are estimated from full-size published photographs of dissections, let us take the thermal conductivity of the tissue as that of pig muscle, 0.0046 W/(cm. °C) (2). Such an apparatus can transfer 0.405 W for each °C of thermal gradient between arterial and venous blood. If the brain receives each minute 750 ml of blood that has a specific heat of 3.85 J/(ml. °C) and is cooled by 1°C, then heat must be removed from the arterial blood at a rate of 48.1 W. To accomplish this, the venous blood in the sinus—only part of which represents cool blood from outside the cranium—must be 119°C cooler than the arterial blood. Even allowing for some heat transfer elsewhere in the cranium, it seems impossible to approach the arteriovenous heat transfer that Cabanac’s concept requires.

There is persuasive evidence for selective brain cooling in many animals that rely primarily on panting for evaporative heat loss. With panting, the surface available for evaporation is relatively small compared with that available with efficient sweating. Might efficient sweating thus be so much more effective in limiting increases in Tc as virtually to eliminate any need for selective brain cooling?

References

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Reply

To the Editor: The first point raised by Bruce Wenger concerns the validity of tympanic temperature as an index of brain temperature. It should not call for much comment on my part because all this was already discussed in my article. However, there is a lesson in humility here, both for him and for me, because apparently the same papers convey contrary messages. Wenger finds “inconclusive” the evidence provided by Baker et al. that tympanic (Tty) and hypothalamic (Thy) temperatures follow identical courses, whereas rectal temperature is par-

More interesting is Wenger's second point, his calculation of the temperature gradient through the sinus cavernosus necessary for the cooling of the brain by 1°C. Wenger apparently did not read my reminder that the internal carotid is surrounded by venous pools from its entry into the carotid canal to its entry into the sinus cavernosus. These pools (Plexus venosus caroticus internus) receive venous blood from the surface and communicate with sinus cavernosus. The length of the interface can be multiplied by at least three. In addition, there is no reason why countercurrent heat exchange should not take place between jugular vein and carotid artery all the way down to the heart, provided that the venous blood is cooler than the arterial blood.

Wenger's calculation does not disprove selective brain cooling; it simply shows that not all the heat exchange takes place in the sinus cavernosus. This we already knew. We have just seen that the vein-artery exchange surface is larger than the sinus cavernosus. There are many other venous pathways through the cranium; the emissary veins are innumerable. A recent paper has shown a striking development of emissary foramina with cephalisation in hominids (D. Falk. Evolution of cranial blood drainage in hominids: enlarged occipital/marginal sinuses and emissary foramina. Am. J. Phys. Anthropol. 70: 311–324, 1986). In addition, there is no reason why some degree of direct conductive cooling should not take place through the external layers of the head. J. D. Whitby and L. J. Dunkin (Cerebral, oesophageal, and nasopharyngeal temperatures. Br. J. Anaesth. 43: 673–676, 1972) have shown that there exists a temperature gradient within the human brain: from 4 cm deep to 2 cm deep the temperature decreases by 0.4°C. As for the origin of this lower temperature in the outer layers of the brain, it is probably due to heat loss from the surface of the head. H. Brinnel (Proceedings of the World Conference on Heat Stress 1st Sydney 1987) measured intracranial temperature in a human subject. The tip of the thermocouple was placed below the parietal bone and external to the dura. The temperature recorded was 1°C below esophageal temperature ($T_{es}$) whereas, incidently, $T_{ey}$ was only 0.2°C lower than $T_{es}$. This intracranial temperature was influenced by face fanning of the patient and dropped by 0.5°C while $T_{es}$ and $T_{ey}$ dropped by only 0.2°C. It should be recalled that many arteries travel on the brain surface before they penetrate the brain itself.

Finally, and this question is my last response, if there is no selective brain cooling how is the brain cooled in a hyperthermic subject, when each °C increase in the carotid blood brings ~50 W to the brain (not to mention the brain's own heat production)? Opponents to the hypothesis of selective brain cooling in humans have yet to answer this question.

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Capillary Permeability

To the Editor: In his article entitled "When Capillary Permeability Increases," which appeared in the February 1987 issue of NIPS, Dr. Crone very succinctly describes the cellular events involved in modulation of capillary permeability. Also described are a number of mediators, such as histamine, serotonin, bradykinin, ATP, ADP, and AMP, which increase microvascular permeability.

One mediator of microvascular permeability that was not mentioned, which I feel should have been included, is platelet-activating factor or PAF. A number of preclinical observations in animals and humans accord this view. The rise from obscurity began in 1981 when it was found that a single intravenous injection of PAF (a mere 50 nmol/kg) produced a marked increase in microvascular permeability, evident by a rapid (within 20 min) and massive loss of plasma (extravasation), leading to over 42% hemocoencentration (9). Later, it was shown that PAF-induced hemocoencentration resulted from increased microvascular permeability, up to and including a complete loss of selective endothelial permeability, which leads to extravasation of plasma and elevated hematocrit levels.

The hemocoencentration effects of PAF are dose and route dependent but independent of platelets or neutrophils. To date, PAF is reported to increase cutaneous, systemic, and pulmonary microvascular permeabilities in a variety of species, including rats, guinea pigs, dogs, primates, and humans (1, 4, 7, 10). By intradermal injection, PAF is 10,000-fold (on a molar basis) more potent than histamine as a mediator of increased microvascular permeability (6). When given intravenously, PAF is over 17,000-fold more potent than histamine and 5-fold more active than leukotriene C$\_4$ as regards the hemocoencentration effects (3). Active at the picomolar range, PAF is considered as one of the most potent mediators of increased microvascular endothelial permeability identified to date.

Morphologically PAF can disrupt postcapillary endothelial junctional integrity (5), thus increasing permeability via the "small pore system." Also, PAF increases both the size and density of endothelial plasma-lamellar vesicles (8), which may contribute to increased permeability via the "large pore system."

Lastly, specific receptor antagonists to PAF (acting as receptor antagonists to platelet binding of PAF) can inhibit in a dose-dependent manner PAF-induced permeability increases and hemocoencentration effects in rats (7), guinea pigs (3, 4, 7), dogs (4), primates (4), and humans (2).

Based on these combined investigations, I would respectively submit that PAF be included as a mediator of increased vascular permeability.

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References


Reply

To the Editor: I quite agree with Dr. Handley. I should add that the list of substances that augment capillary permeability was never meant to be exhaustive.

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