Hyperthermia-Induced Vasoconstriction: A Physiological Counter Mechanism

ARTICLE

The paradoxical phenomena of hyperthermia-induced vasoconstriction, hyperthermia-induced hypothermia, and hyperthermia induced arterial vasoconstriction have been observed in animal heatstroke clinical case studies (Romanovsky & Blatteis, 1996). We attempt to explain the basic mechanism behind such findings in terms of the principle of body heat conservation and natural countermeasure against heat exposure through reduction in the extent of vascular surface area exposed. One study which examined the outcome of heating a rabbit's carotid artery produced graded vasoconstriction which is proportional to temperature increase (Mustafa, Thulesius, & Ismael, 2004).These interesting results pointed out a spontaneous physiological counteractive measure to minimise organ and/or arterial surface area heat exposure through vasoconstriction which might otherwise prompt excessive heat gain/loss from the ambient environment, and/or vasodilation, should it occur. In the event of vascular surface exposure, there may be a bi-directional heat flow exchange that can be lost or gained on the basis of inter-surface concentration gradient through the blood vessel barrier. When a blood vessel or artery constricts through smooth muscle activation, the amount of vascular surface area being exposed, whether to heat that is external from or internal within the body, is reduced. Since such vasoconstriction is proportional to temperature increase, the speed or rate at which this constriction responds and therefore, the extent of vascular surface area being reduced, may be drastic with extreme means of bodily cooling under a heat wave environment, such as whole-body cold-water bath or shower. The reason being extreme vasoconstriction of both cerebral and peripheral blood vessels, which may result in increased intravascular pressure due to intravascular spatial volume decrease and as an above study concluded, cerebral blood flow decrease, and ischemia brain damage. On the other hand, cold-induced vasodilation is the dilation of peripheral arteries under exposure to extravascular cold temperature (Daanen, 2003; Flouris, Westwood, Mekjavic, & Cheung, 2008). It is counterintuitive but the build-up of a heat concentration gradient with greater intravascular heat flow than the external may prompt active heat loss and exchange across the vascular surface into the peripheral environment. Another factor to consider is the body’s spontaneous counter-reaction and opposition against cold-induced increase of intravascular pressure to occur under extreme

low ambient temperature, which then triggers the vasodilatory effect as a result to prevent excessive internal pressure rise within the arteries. It is no wonder that cold-induced vasodilation helps to reduce cold injuries, which may be attributed to its counter mechanism strategy to decrease severe intravascular pressure increase under extreme cold conditions (Kingma, Hofman, & Daanen, 2019).

Although cold-induced cutaneous vasoconstriction in humans bears functional significance in heat and blood flow reduction to external environment through contact surface area exposure of the skin blood vessels, hyperthermia-induced vasoconstriction of the animal's carotid artery is better viewed from the perspective of core body temperature (Alba, Castellani, & Charkoudian, 2019). In the event of hyperthermia-induced vasodilation of cutaneous vessels, a temperature gradient with the internal arteries of the body core is induced. In order to counter the increasing temperature gradient and maintain homeostatic equilibrium, cutaneous heat loss needs to be matched with core heat gain. In addition to the presence of a protective blood-brain barrier, the carotid artery has to preserve a strict boundary for the brain from extended cutaneous heat loss and the attempt of core body heat loss to lessen the temperature gradient with peripheral blood vessels.

A large body core-peripheral temperature gradient is related to poor tissue perfusion and decreased vascular function as a narrow range of core-peripheral difference of 4° C or less is indicative of normal circulation (Schey, Williams, & Bucknall, 2010). Hence, paradoxical, yet protective counter mechanism of vasoconstriction of the carotid artery may be necessary to preserve vulnerable brain components from probable associative heat loss during the event of hyperthermia and significant cutaneous vasodilation at the cost of extended core-peripheral temperature gradient. Therefore, the body's heat conservation strategy remains in effect and as previously noted, reduced vascular surface area exposure helps maintain a fairly optimal body core-peripheral temperature gradient to prevent unsteady blood pressure perturbation.

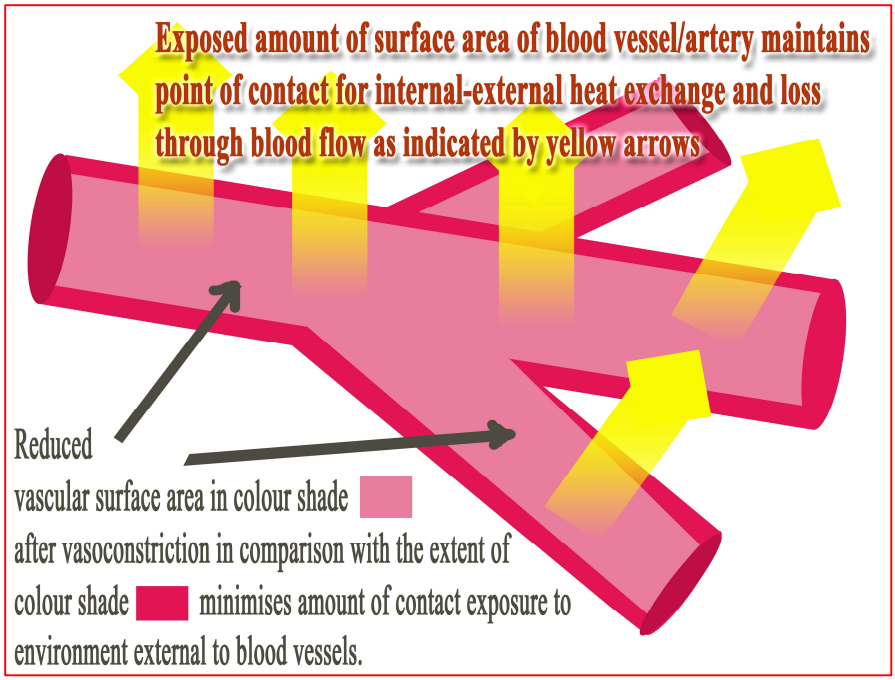

Figure 1: Vascular Surface Area Adaptability to Heat Flow Exchange Variability

Nevertheless, this does not preclude a likelihood of dysfunction as the body adapts to extreme environmental temperature disturbance(s) that alter physiological balance and homeostatic stability. Measures to cool the ambient environment and atmosphere may be necessary before direct and/or invasive means of patient treatment to minimise progression to extreme stages of signs and symptoms.

References

Alba, B. K., Castellani, J. W., & Charkoudian, N. (2019). Cold-induced cutaneous vasoconstriction in humans: Function, dysfunction and the distinctly counterproductive. *Experimental physiology*, *104*(8), 1202-1214.

Daanen, H. A. (2003). Finger cold-induced vasodilation: a review. *European journal of applied physiology*, *89*(5), 411-426.

Flouris, A. D., Westwood, D. A., Mekjavic, I. B., & Cheung, S. S. (2008). Effect of body temperature on cold induced vasodilation. *European journal of applied physiology*, *104*, 491-499.

Kingma, C. F., Hofman, I. I., & Daanen, H. A. M. (2019). Relation between finger cold-induced vasodilation and rewarming speed after cold exposure. *European Journal of Applied Physiology*, *119*, 171-180.

Mustafa, S., Thulesius, O., & Ismael, H. N. (2004). Hyperthermia-induced vasoconstriction of the carotid artery, a possible causative factor of heatstroke. *Journal of Applied Physiology*, *96*(5), 1875-1878.

Romanovsky, A. A., & Blatteis, C. M. (1996). Heat stroke: opioid-mediated mechanisms. *Journal of applied physiology*, *81*(6), 2565-2570.

Schey, B. M., Williams, D. Y., & Bucknall, T. (2010). Skin temperature and core-peripheral temperature gradient as markers of hemodynamic status in critically ill patients: a review. *Heart & Lung*, *39*(1), 27-40.

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Conflict of Interest

The authors have no conflicts of interest to declare.