Hyperthermia-Induced Vasoconstriction: A Thermodynamic Regulation Counter Mechanism

ABSTRACT

Hyperthermia-induced vasoconstriction is a paradoxical physiological phenomenon that has limited explanation and exploration in research, despite being observed in clinical case studies. The bodily response of vasoconstriction and vasodilation to increased heat loss and gain respectively work on the principle that heat transfer through the amount of vascular surface area exposed can be moderated in order to be minimised or maximised. Hyperthermia-induced vasoconstriction likely transitions from the break point of maximal limit of vasodilation to counter extreme heat loss beyond this point and to mitigate the potential detrimental effect in arterial pressure disturbance by extreme temperature condition. We also propose an additional explanation of this physiological counter mechanism based on the perspective of body core-peripheral steep temperature gradient reduction during hyperthermia or significant temperature increase, and a spontaneous tendency to avoid unstable blood pressure perturbation.

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INTRODUCTION

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). The bodily response of vasoconstriction and vasodilation to increased heat loss and gain, works on the principle that heat transfer through the amount of vascular surface area exposed can be moderated in order to be minimised or maximised through muscle activation, which in turn also results in volume changes.

Regulating Thermodynamic Temperature-Volume-Pressure Disturbance

Logic explanation would support persistent physiological response of vasodilation following hyperthermia, yet observations reported that the opposing response of vasoconstriction has taken place. We propose an explanation that under the condition of extreme and enduring hyperthermia, continuous vasodilation would be health-compromising on the system and a critical factor of hypotension or severe arterial pressure reduction would result should a break point of maximal limit on vasodilation be not set in place to counter further rapid heat loss beyond this point. Vasodilation inevitably has an effect on vascular pressure because smooth muscle relaxation and expansion of the volume of blood arteries and vessels increase space and reduce pressure, an interplay of thermodynamic equilibrium. Hyperthermia-induced vasoconstriction prevents the detrimental effect of continuity in progression of vasodilation in relation with vascular pressure, and functions to maintain internal pressure gradient's constancy and stability, as constricting blood vessels would elevate arterial pressure and perfusion of core body organs and system, such as the experimental rabbit's carotid artery in a previous research. In order to keep the arterial pressure constant and unperturbed, reduction in volume through vasoconstriction is needed to counter the hypotensive effect of sharp temperature increase and corresponding vascular volume expansion(dilation) of hyperthermia. In the case of the experimental rabbit's carotid artery, graded temperature increases with decreasing volume through vasoconstriction, internal arterial pressure would remain constant and be preserved to maintain thermodynamic equilibrium. In addition, heat loss to the extreme through exposed surface area is also regulated and moderated. In addition, we include another explanation of this physiological counter mechanism based on the perspective of body core-peripheral steep temperature gradient reduction during hyperthermia or extreme temperature condition and a spontaneous tendency to avoid unstable blood pressure perturbation. Such spontaneous physiological counteractive measure to minimise vascular surface area and volume of heat exposure through extreme vasodilation by inducing vasoconstriction to counter against persistent thermodynamic disturbance of internal organ(s) strategically restores and re-elevates potential significant arterial blood pressure reduction.

Moderation of Vascular Surface Exposure

Experimental 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 based on 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).

Body Core-Peripheral Temperature Gradient

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.

Preservation of Tissue Perfusion and Discussion

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 beyond the break point of hyperthermia-induced vasodilation transitioning into vasoconstriction. The three fundamental thermodynamic states of vascular temperature, pressure and volume work closely and persistently to maintain equilibrium and stable gradients within a system where the impact of changes on one induces a unitary response by all states as a counter mechanism.

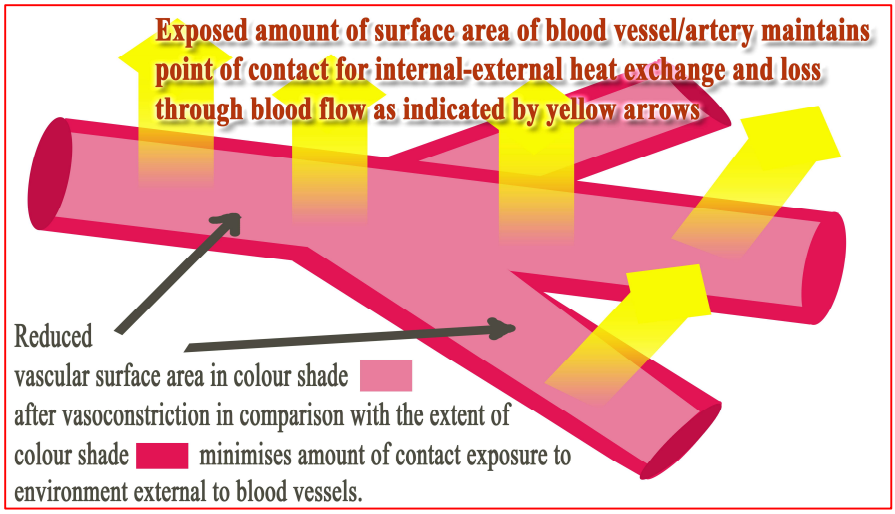

Figure 1: Vascular Surface Area Adaptability to Heat Flow Exchange Variability

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.

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