EFFECTS OF HYPEROSMOLALITY ON BODY TEMPERATURE AND ARTERIAL PRESSURE REGULATIONS DURING EXERCISE IN THE HUMAN

Hiroshi Nose, Yoshi-ichiro Kamijo, Tadashi Okumoto, Yoshiaki Takeno, Kazunobu Okazaki and Shizue Masuki, Department of Sports Medicine, Shinshu University School of Medicine, 3-1-1 Asahi Matsumoto 390-8621, Japan.

Hyperosmolality by dehydration has been reported to suppress cutaneous vasodilation and sweating in hyperthermia (Takamata *et al.*, 1997). However, the physiological role of the suppression has not been elucidated yet. Recently, Nakajima *et al.* (1998) reported in awake and passively heated rats that mean arterial pressure (MAP) decreased with a rise in tail skin blood flow (SkBF) in a hypovolemic and isosmotic condition, whereas in a hypovolemic and hyperosmotic condition MAP remained unchanged with the suppression of tail skin vasodilation. They concluded that plasma hyperosmolality stimulates pressor responses in the hypovolemic condition that subsequently contribute to arterial pressure regulation during heat stress. Takamata *et al.* (1995) reported in resting and passively heated humans that the hyperosmolality-induced suppression of sweating was released by oropharyngeal reflexes by drinking such a small amount of water as altered neither blood volume nor plasma osmolality. However, they did not find any significant changes in SkBF and MAP after the drinking. However, during exercise in heat and also in the hypovolemic condition, where SkBF competes with muscle blood flow, drinking would release the hyperosmolality-induced suppression of cutaneous vasodilation as well as sweating, resulting in a fall in MAP due to increased total peripheral conductance. To examine the hypotheses, 6 male subjects underwent 4 hydration states; 1) normal plasma volume (PV) and isosmolality trial, 2) low PV (ΔPV = -10%) and isosmolality trial, 3) normal PV and hyperosmolality (ΔPosm = 10 mOsm) trial, and 4) low PV and hyperosmolality trial. The hydration states were attained in separate experimental days in each subject by prior administration of diuretics, intravenous hypertonic NaCl solution and/or 24-hr water restriction. After the treatments, subjects exercised with a cycle ergometer at 60% of maximal aerobic power for 50 min in a hot environment (atmospheric temperature of 30°C and relative humidity of 50%). After esophageal temperature, forearm SkBF (by venous occlusion plethysmography), and sweat rate (with humidity sensor placed on chest surface) reached a plateau by 15 -20 min of exercise, subjects drank the small amount of water of 37°C (100ml). Immediately after the drinking, SkBF and sweat rate in the hyperosmolaity trials increased by 5-10% above the base line, accompanied by the reduction in MAP by 5-10 mmHg. However, we did not find any significant changes in these variables after the drinking in the isosmolality trials. There were no significant reductions in plasma norepinephrine and vasopressin concentrations after the drinking in every trial. These results suggest that the hyperosmolality-induced suppression of cutaneous vasodilation contribute to arterial pressure regulation regardless of blood volume during exercise in a hot environment in humans. Moreover, the suppression may be caused by attenuated active vasodilator system but not by enhanced active vasoconstrictor system such as by increased sympathetic nervous activity and/or vasopressin release. Finally, the suppression may be released by drinking via oropharyngeal reflexes.

Nakajima, Y. *et al.* (1998) Plasma hyperosmolality and arterial pressure regulation during heating in dehydrated and awake rats. *Am. J. Physiol.* 275, R1703-R1711.

Takamata, A. *et al.* (1995) Osmoregulatory modulation of thermal sweating in humans: reflex effects of drinking. *Am. J. Physiol.* 268, R414-R422.

Takamata, A. *et al.* (1997) Osmoregulatory inhibition of thermally induced cutaneous vasodilation in passively heated humans. *Am. J. Physiol.* 273, R197-R204.

nosehir@sch.md.shinshu-u.ac.jp