Hypertension Research (2023) 46:1705–1706
https://doi.org/10.1038/s41440-023-01290-2

COMMENT

# Effects of hot spring bathing on cardiac and vascular function

Satoko Ojima[1] · Mitsuru Ohishi[1]

**Keywords** Hot spring bathing · Hydrostatic pressure · Vasodilation · Hemodynamic

Received: 26 March 2023 / Accepted: 5 April 2023 / Published online: 25 April 2023


Hot spring bathing has been a global practice dating back to ancient times as a means of preventing various diseases and promoting health. Despite the widely discussed psychological benefits associated with hot spring bathing, it has not remained easy to prove the physical benefits scientifically. Nonetheless, recent studies on the effects of hot spring bathing on cardiovascular function have been reported, particularly changes in heart rate (HR), cardiac output (CO), and blood pressure (BP) before and after hot spring bathing. Not only healthy individuals but also patients with hypertension after bathing have reported increase in the level of HR or CO and decrease in the level of systolic or diastolic BP compared with before bathing [1–3]. These findings are also supported by the study conducted by Wang et al. [4].

These observed effects can be explained from two perspectives: hydrostatic pressure and vasodilation. Hydrostatic pressure resulting from submerging the water impacts circulatory dynamics as the arteries and veins are externally compressed by the hydrostatic pressure of the water, depending on depth. When entering the water up the chest level, the hydrostatic pressure of the blood vessels is optimally balanced by the hydrostatic pressure of the outside water, resulting in the same hemodynamic state as in the spinal position. As a result, venous return increases during bathing, leading to elevated HR and CO compared with levels before bathing. These effects persist for a while, even after bathing. Furthermore, the relationship between HR, CO, and BP can be explained by the definition of CO as a function of HR and stroke volume (SV), and BP is defined by CO and peripheral vascular resistance (PVR) (Fig. 1).

✉ Satoko Ojima
satoko9cats@gmail.com

[1] Department of Cardiovascular Medicine and Hypertension, Graduate School of Medical and Dental Sciences, Kagoshima University, Kagoshima, Japan

Vasodilation involves various factors such as thermal effects, nitric oxide (NO), the autonomic nervous system, and natriuretic peptides, all of which are known to decrease BP [5, 6]. Bathing in a hot spring leads to an increase in HR and CO, while the vasodilating effect of heat from the springs causes a decrease in PVR that surpasses the effect of the HR and CO increase, resulting in a decrease in BP. Moreover, elevated HR after bathing increases CO, leading to an increased blood flow and induction of shear stress on the vascular endothelium. This, in turn, stimulates NO production via the endothelial NO synthase, activated by shear stress, using the amino acid L-arginine as a substrate. Furthermore, NO activates guanylate cyclase, an enzyme that generates cyclic guanosine monophosphate (cGMP) from guanosine triphosphate in the vascular smooth muscle. The resulting cGMP acts on the vascular smooth muscle to dilate blood vessels, thereby decreasing PVR and finally causing a decrease in BP.

At a water temperature of approximately 40 °C, the baroreceptor responds to the heat-induced increase in CO and activates the parasympathetic nervous system, leading to vasodilation. This results in a decrease in BP. Conversely, bathing in hot water exceeding 42 °C activates the sympathetic nervous system, causing vasoconstriction, which results in an increase in both BP and HR, which is the opposite of previous results [7]. The study by Wang et al. was conducted at a water temperature of 38–40 °C [4].

It is known that when bathing, the heart can be overloaded with hydrostatic pressure and increased CO, leading to the production of atrial natriuretic peptide (ANP) from the atria, and the brain natriuretic peptide (BNP) from the ventricles. ANP has been used as a therapeutic agent for patients with heart failure in Japan [8]. BNP is also widely applied in diagnosing and evaluating heart failure diseases and their severity, and it serves as a prognostic factor [9]. The natriuretic and vasodilating properties of ANP and BNP reduce circulating blood volume, resulting in decreased BP.

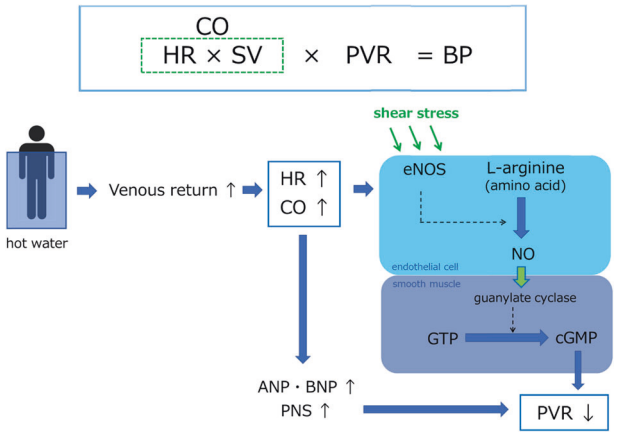


**Fig. 1** Correlation between hot spring bathing and BP decrease. CO cardiac output, HR heart rate, SV stroke volume, PVR peripheral vascular resistance, BP blood pressure, eNOS endothelial nitric oxide synthase, NO nitric oxide, GTP guanosine triphosphate, cGMP cyclic guanosine monophosphate, ANP atrial natriuretic peptide, BNP cerebral natriuretic peptide, PNS parasympathetic nervous system

Thus, it is evident that the impact of hot spring bathing on BP is composed of various factors. Since the effects of hot baths can vary depending on the disease, it is important to provide guidance based on thorough comprehension of the physical effects of hot spring bathing to ensure the safe enjoyment of the individuals.

## Compliance with ethical standards

**Conflict of interest** The authors declare no competing interests.

**Publisher's note** Springer Nature remains neutral with regard to jurisdictional claims in published maps and institutional affiliations.

## References

1. Ishikawa J, Yoshino Y, Watanabe S, Harada K. Reduction in central blood pressure after bathing in hot water. Blood Press Monit. 2016;21:80–6.
2. Kataoka Y, Yoshida F. The change of hemodynamics and heart rate variability on bathing by the gap of water temperature. Biomed Pharmacother. 2005;59:92–9.
3. Tei C. Waon therapy: soothing warmth therapy. J Cardiol. 2007;49:301–4.
4. Wang PC, Song QC, Chen CY, Su TC. Cardiovascular physiological effects of balneotherapy: focused on seasonal differences. Hypertens Res. 2023. https://doi.org/10.1038/s41440-023-01248-4.
5. Tei C, Horikiri Y, Park JC, Jeong JW, Chang KS, Toyama Y, et al. Acute hemodynamic improvement by thermal vasodilation in congestive heart failure. Circulation. 1995;91:2582–90.
6. Sartori C, Lepori M, Scherrer U. Interaction between nitric oxide and the cholinergic and sympathetic nervous system in cardiovascular control in humans. Pharm Ther. 2005;106:209–20.
7. Eimantas N, Ivanove S, Solianik R, Brazaitis M. Exposure to acute noxious heat evokes a cardiorespiratory shock response in humans. Int J Hyperth. 2022;39:134–43.
8. Kobayashi D, Yamaguchi N, Takahashi O, Deshpande GA, Fukui T. Human atrial natriuretic peptide treatment for acute heart failure: a systematic review of efficacy and mortality. Can J Cardiol. 2012;28:102–9.
9. Ibrahim NE, Burnett JC Jr, Butler J, Camacho A, Felker GM, Fiuzat M, et al. Natriuretic peptides as inclusion criteria in clinical trials: a JACC: Heart Failure Position Paper. JACC Heart Fail. 2022;8:347–58.