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COMMENTARY

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Impact of baseline arterial elasticity (stiffness) on left ventricular functions in healthy subjects exposed to short-term extreme cold

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In the current issue of *The Journal of Clinical Hypertension*, Chen et al.[1] assessed the association of baseline blood pressure variability (BPV) with the changes in left heart functions after short-term extreme cold exposure. A total of 70 healthy participants were exposed to the cold outside (Mohe City, Heilongjiang Province, China, the average temperature of each month was below 0°C for 8 months, and the temperature interval during the study was −17 to −34°C) for 1 day, and were monitored by a 24-h ambulatory blood pressure monitoring (ABPM) and underwent transthoracic echocardiography before and after extreme cold exposure. All participants performed their daily activities during the daytime and rested in tents at nighttime. The forehead skin and respiratory tract alone were exposed to cold mainly because the participants wore winter clothes. Among 70 subjects in the study, 41 participants (58.6%) revealed an increase in left ventricular ejection fraction (LVEF), and the remaining 29 participants (41.4%) showed a decrease in LVEF after cold exposure. Baseline coefficients of variation (CV) in BP (particularly daytime) and average real variability (ARV) as parameters of BP variability (BPV) were lower in participants with LVEF increase compared to the LVEF decrease group. In multivariable regression analysis, CV and ARV were reported as significant predictors of LVEF change after short-term extreme cold exposure. Beyond focusing on the LVEF change alone, end-diastolic volume (EDV), end-systolic volume (ESV), E/A, E/e', and ventricular-arterial coupling (VAC) were significantly reduced, however global longitudinal/circumferential strain (GLS/GCS), torsion, untwisting rate, effective arterial elastance (Ea), and end-systolic elastance (Ees) were significantly increased after short-term extreme cold exposure in all participants. Although the EDV was reduced in participants with both LVEF increased and decreased after an extreme cold exposure, the ESV was only reduced in participants with LVEF increase. Furthermore, the Ees was increased and the VAC was reduced in participants with LVEF increase after an extreme cold exposure. Besides changes in LV functions, there was also an increase in mean 24-h heart rate (particularly night-time heart rate) and systolic BP (particularly daytime systolic BP) in participants with LVEF increase after extreme cold exposure. As mentioned in the study, there was no data about the body temperatures and sympathetic or parasympathetic hormone levels in response to short-term extreme cold exposure.

The human body has adaptive mechanisms to the acute and chronic changes in the ambient temperatures.[2,3] The central and autonomic nervous system plays a critical role in the management of those adaptive changes that directly act on cardiac and vascular functions.[3–6] An alteration in the central and autonomic nervous system affects both ventricular and arterial functions, thus ventricular-arterial coupling (VAC). Blood pressure is also a reflection of VAC and is calculated by using heart rate, stroke volume, and total peripheral arterial resistance. However, BP is a dynamic parameter, and BPV is a well-known indicator of BP fluctuations over time that is affected by changes in the arterial stiffness/elasticity and autonomic nervous system.[7–9] As mentioned in the study by Chen et al.,[1] extreme cold exposure causes an alteration in autonomic nervous system activity. This might be obvious from an increase in both 24-h heart rate and systolic BP in participants with an increased LVEF. Increases in HR, BP, and afterload are well-known acute physiological effects of increased adrenergic activity after cold exposure.[4,5] However, the autonomic nervous system does not work with the all or not principle. After an initial defense response with an increased adrenergic tone in subjects exposed to cold, the autonomic tone is balanced between sympathetic and parasympathetic systems

and shifts toward the parasympathetic predominance in long-term exposure to cold.[2,4–6,10] Several confounding factors (both genetic and acquired) may be responsible for those differences in changes or adaptations.[2] Despite an overt difference in baseline BPV parameters (CV and ARV were greater in participants with LVEF decrease after short-term extreme cold exposure) between increased and decreased LVEF groups in the study by Chen et al.,[1] there was no significant change in BPV between LVEF groups after extreme cold exposure. Thus, the baseline determinants of BPV [including basal autonomic nervous system tonus (probably on the side of an increased sympathetic tone) and arterial stiffness/elasticity in participants with a baseline higher BPV and LVEF decrease after extreme cold exposure] in study participants are critical in the prediction of changes in left ventricular functions.[9,11] Previous studies demonstrated that the BPV is closely associated with the arterial stiffness and vice versa.[9,12,13] Baseline arterial stiffness in patients with LVEF decrease following short-term extreme cold exposure may have resulted in a higher baseline BPV, increased afterload, VAC dyssynchrony, and subsequent suppression of left ventricular function. Hintsala et al.[14] demonstrated that moderate whole-body cold exposure (−10°C, wind 3 m/s, 15 min, winter clothes, standing) increased BP and cardiac workload more among those with higher systolic home BPV. They proposed that an elevated home BPV may be a marker of augmented sympathetic tone-driven vascular reactivity for cold exposure. On the other hand, in those with increased LVEF after exposure to extreme cold, a significant decrease in ESV is one of the main changes that should be taken into consideration, as it is used in the calculation of all hemodynamic parameters. The ESV is determined by ventricular contractility, afterload, and eccentric remodeling, but not by preload.[15] Therefore, a higher baseline BPV as an indicator of arterial stiffness in the LVEF decrease group may have negatively affected the autonomic nervous system-mediated adaptation of both ventricular and arterial functions (coupling) to the short-term extreme cold exposure. Additionally, we do not know whether the results would have been similar if the researchers had conducted this study with participants living in regions with different ambient temperatures instead of local participants or residents of the region. The mode and severity of adaptive changes (autonomic modulation) in response to short-term extreme cold exposure outside may be different for residents versus non-residents of cold regions. Furthermore, patients with different baseline characteristics (race, body habitus, body surface area, basal metabolism rate, physical fitness) or diseases like hypertension, vascular disease (e.g., coronary artery disease, peripheral artery disease), heart failure, and autonomic dysfunction (e.g., elderly, diabetes, neurological diseases) may have different, less or no adaptive mechanisms to the extreme ambient temperatures as mentioned by Chen et al.[1,16] Therefore, the findings of the current study should be tested in healthy non-resident participants and diseased resident/non-resident participants at different time intervals of extreme cold exposure for generalization to the different populations. This study's findings may be especially important for athletes and travelers who will be visiting extremely cold regions around the world and staying in cold environments. A medical examination (including 24-h ABPM and echocardiography) to evaluate adaptation mechanisms to extreme cold exposure before traveling to these regions may be useful in providing preventive medicine approaches and necessary recommendations to concerned individuals.

CONFLICT OF INTEREST STATEMENT

U.C.: Proctoring for Conduction System Pacing at Biotronik & Medtronic.

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