Artery Research (2018) 21, 53–57

Available online at www.sciencedirect.com

ScienceDirect

journal homepage: www.elsevier.com/locate/artres

Impact of leg heating on central hemodynamics in postmenopausal women

Keisei Kosaki [a], Jun Sugawara [b], Asako Zempo-Miyaki [c], Seiji Maeda [d,*]

[a] Graduate School of Comprehensive Human Sciences, University of Tsukuba, Tsukuba, Ibaraki, Japan
[b] Human Informatics Research Institute National Institute of Advanced Industrial Science and Technology (AIST), Tsukuba, Ibaraki, Japan
[c] Faculty of Sport and Health Sciences, Ryutsu Keizai University, Ibaraki, Japan
[d] Faculty of Health and Sport Sciences, University of Tsukuba, Tsukuba, Ibaraki, Japan

Received 12 October 2017; received in revised form 7 January 2018; accepted 11 January 2018

KEYWORDS
Central pressure augmentation;
Wave reflection;
Pulse wave velocity;
Central-to-leg arterial stiffness gradient

Abstract *Background:* The determinants responsible for increasing central pressure augmentation, a strong risk factor for cardiovascular disease, remain highly controversial. The aim of this study was to determine the contribution of the impedance mismatch between central and leg arteries on central pressure augmentation. Thus, we investigated whether central pressure augmentation is influenced by manipulation of central-to-leg arterial stiffness mismatch by an acute leg heating.
Methods: Nineteen postmenopausal women underwent the warmth stimulation on both lower legs (20 min of exposure to far infra-red radiation at 43–45 °C followed by retaining warmth by a blanket for 30 min). Central (aortic) hemodynamic measures were obtained from applanation tonometrically-radial arterial pressure waveforms via the validated general transfer function.
Results: The leg heating decreased only the leg pulse wave velocity. And thus, the central-to-leg arterial stiffness was changed from positive to negative; however, any central (aortic) hemodynamic measures including reflected wave amplitude, aortic blood pressure, and augmentation index, were not changed significantly.
Conclusions: Our results suggest that the acute bout of leg heating induces the less of central-to-leg arterial stiffness mismatch but not influence central pressure augmentation in postmenopausal women.

* Corresponding author. Faculty of Health and Sport Sciences, University of Tsukuba, 1-1-1 Tennodai, Tsukuba, Ibaraki, 305-8574, Japan. Fax: +81 29 853 2986.
E-mail addresses: kuromaru0319@gmail.com (K. Kosaki), jun.sugawara@aist.go.jp (J. Sugawara), a.miyaki.velma08@gmail.com (A. Zempo-Miyaki), maeda.seiji.gn@u.tsukuba.ac.jp (S. Maeda).

https://doi.org/10.1016/j.artres.2018.01.001

Introduction

Enhanced central pressure augmentation, evaluated by augmented pressure (AP) and augmentation index (AIx), could be a strong risk for cardiovascular disease (CVD).[1] Postmenopausal women exhibit greater central pressure augmentation compared with premenopausal women and age-matched men.[2]

There is a widely held notion that the central arterial pressure waveform is synthesized by the overlapped reflection waves returning from the periphery (mainly lower body) on the incident wave in phase.[3] AP and AIx are associated with the extent and timing of reflected wave that arises from the periphery.[3] In the arterial tree, a traveling wave will be reflected to some extent wherever there is a discontinuity such as branching points (e.g., branches of renal arteries, aortic bifurcations), areas of alteration in arterial stiffness (e.g., from the elastic artery to the muscular artery), and high-resistance arterioles.[3] Therefore, alterations of peripheral vascular tone and arterial stiffness may influence central pressure augmentation.

Recently, however, this theory has been argued. Growing evidences suggest that major determinants of central BP waveform may be the incident waves arising from left ventricular ejection and proximal aortic compliance rather than the wave reflection.[4–10] For example, a community-based cross-sectional investigation revealed that aortic-brachial artery stiffness mismatch was not associated with the central AP and AIx.[11] An interventional experiment supported this result that the central hemodynamics were unaltered even if the brachial vascular tone was changed using an arm heating.[12] On the other hand, it remains unclear whether the impedance mismatch between central and leg arteries influences central pressure augmentation.

From ancient times, a leg thermal therapy with comfortable temperature (≈40 °C) such as "footbath" has been widely used for the purpose of improving the whole-body hemodynamics and keeping warm. In particular, the lower leg heating has an advantage that it can be carried out everyone easily and safely without the need for undressing. It is well known that the warmth stimulation elicits local various vasodilatory substances typified by nitric oxide.[13,14] In fact, it has been reported that an isolated leg heat stress increases leg blood flow and vascular conductance.[15] They may induce not only dilatation in local (lower leg) vascular bed but also upstream hyperemia (e.g., femoral arterial blood flow) and consequent dilation of conduit arteries because of the increases in downstream vascular conductance and inflow. If it is so, leg arterial stiffness could be decreased, and hence, arterial stiffness mismatch between central and leg arteries is attenuated. Taken together, the lower leg heating may change wave reflection and central pressure augmentation via decreased vascular resistance and less arterial stiffness mismatch even though acutely.

With this information as background, in the present study, we determined the effect of an acute bout of lower leg heating on central pressure augmentation in postmenopausal women.

Methods

Subjects

We studied 19 postmenopausal women (age, 64 ± 3 years; height, 154 ± 6 cm; weight, 53 ± 7 kg; mean ± SD). All the subjects had no apparent cardiovascular disease as assessed by medical history and did not take any medications. None of the participants had a history of smoking or regular physical exercise in addition to normal living. This study was reviewed and approved by the Institutional Review Board. Additionally, all procedures conform to the ethical guidelines of Helsinki Declaration. All potential risks and procedures of the study were explained to the subjects, and they gave their written informed consent to participate in the study.

Experimental protocol

The subjects abstained from alcohol, caffeine intake, and vigorous exercise for at least 12 h before the experiments. The experiment which consisted of 50-min intervention (leg heating or control) and pre- and post-intervention hemodynamics measurements was conducted in a temperature-controlled room (temperature ≈ 25 °C). Subjects remained supine position on a bed throughout the entire experimental protocol including at least 30 min of quiet rest before the start of pre-intervention measurement. Heat stimulation on both lower legs (under knees) was 20 min of exposure to far infra-red radiation at 43—45 °C (Leghot, Fujika Co., Ltd., Tokyo, Japan) followed by retaining warmth by a blanket for 30 min.[16] In the resting control condition, subjects remained supine resting without the leg heating intervention. These trials were performed on separate days within a week using a crossover design (Fig. 1).

Measurements

Heart rate, local (brachial and ankle) blood pressure (BP), and central (carotid-femoral) and leg (femoral-ankle) pulse wave velocities (PWV) were measured by a semi-automated vascular testing device (Form PWV/ABI: Model BP-203RPEII and TU-100; Colin Medical Technology, Komaki, Japan), as previously described.[17] The central to leg arterial stiffness gradient was evaluated by subtracting central PWV from leg PWV. Central (aortic) hemodynamic variables were synthesized from applanation tonometrically-radial arterial pressure waveforms with the validated general transfer function-based pulse wave analysis software (SphygmoCor version 8.0, AtCor Medical, Sydney, Australia), as we previously reported.[18] Aortic AP was defined as the difference between the first systolic peak (or shoulder) and second systolic peak pressures. Aortic AIx was calculated as the percent ratio of aortic AP to aortic pulse pressure, and standardized for a heart rate of 75 bpm. The timing of reflected wave return (TR) was calculated as the time from the beginning upstroke of the central pressure wave to the systolic upstroke of the reflected wave (inflection point). Synthesized aortic pressure waveform was decomposed