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Editorial

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Basic Research Sheds Light on the Aspect of Cardio-Ankle Vascular Index (CAVI) including Elastic and Muscular Arteries

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As an English physician, Thomas Sydenham (1624–168), claimed that a man is as old as his arteries, the association between age and atherosclerosis has been recognized. It is not an exaggeration even if it is said that the blood vessel determines the healthy longevity. Therefore, the noninvasive determination of atherosclerosis, including arterial stiffness, is important. Pulse wave velocity (PWV) has been used as a noninvasive index of arterial stiffness and atherosclerosis; further, it is useful as a predictor or surrogate endpoint marker for cardiovascular disease. The cardio-ankle vascular index (CAVI) by measuring PWV and blood pressure (BP) was developed and is adjusted for BP based on the stiffness parameter β[1]. Although CAVI and brachial-ankle PWV (baPWV) represent the arterial stiffness of elastic and muscular arteries, we reported that CAVI showed a weaker correlation with systolic BP than with baPWV and was not affected by changes in BP during measurement[2]. Thus, we believe that CAVI can accurately measure the arterial stiffness independent of BP at the time of measurement.

A recent review article published in the Journal of Atherosclerosis and Thrombosis summarized that CAVI is associated with the presence of atherosclerotic diseases including coronary artery disease, cerebral infarction, chronic kidney disease, and thickening of the carotid intima–media thickness, and coronary risk factors, such as hypertension, diabetes mellitus, uric acid disorders, sleep apnea syndrome, smoking, and obesity in several cross-sectional studies[3]. In addition, several prospective studies demonstrated that CAVI is a predictor of cardiovascular outcomes[3] including that CAVI is an independent long-term predictor of major adverse cardiovascular events, particularly cardiovascular death, in patients with acute coronary syndrome[4]. Moreover, high CAVI (cut-off point of ≥ 9) was reported to be independently associated with a rapid decline in glomerular filtration rate in patients at high risk of cardiovascular disease with or without chronic kidney disease, which suggested that systemic vascular stiffness predicted a decrease in renal function in this population[5]. Although many clinical evidences of CAVI have been reported, there are a few basic researches of CAVI[6-8].

In this issue of Journal of Atherosclerosis and Thrombosis, Katsuda *et al.* reported an interesting basic research about arterial stiffness using Beta defined according to the theory of CAVI in anesthetized rabbits[9]. As shown in **Table 1**, the infusion of phentolamine, a non-selective α adrenergic blocker, reduced BP and total peripheral vascular resistance (TPR) and increased heart rate (HR) and cardiac output (CO). In this systemic circulation, phentolamine decreased ifBeta, which is the arterial stiffness of the ilio-femoral artery (muscular artery), and increased aBeta, which is the arterial stiffness of the aorta (elastic artery). Following infusion of β1 adrenergic blocker atenolol, BP, HR, and CO decreased and TPR did not change, whereas aBeta and ifBeta remained unchanged. These results suggested that phentolamine stimulates arterial smooth muscle relaxation and decreases arterial resistance and ifBeta with a compensation of increased aBeta in the elastic artery. In contrast, although atenolol decreased BP, it did not affect the arterial smooth muscle and did not change TPR, aBeta, and ifBeta. Katsuda *et al.* concluded that the contradictory reactions of aBeta and ifBeta with phentolamine suggest that stiffnesses of the aorta and ilio-femoral artery are separately regulated during decreased BP induced by phentolamine, but not by atenolol. For understanding the clinical meaning of

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Table 1. Different reactions of phentolamine and atenolol on circulation and arterial stiffness

Drug	Phentolamine	Atenolol
Mechanism of drug action	non-selective α adrenergic blocker	β1 adrenergic blocker
Blood pressure	↓	↓
Heart rate	↑	↓
Cardiac output	↑	↓
TPR	↓	→
aBeta: elastic artery	↑	→
ifBeta: muscular artery	↓	→

TPR: total peripheral vascular resistance, aBeta: arterial stiffnesses of the aorta (elastic artery), ifBeta: arterial stiffnesses of the ilio-femoral artery (muscular artery).
Cited and modified from reference #9.

CAVI that represents the arterial stiffness both of the elastic and muscular artery, this basic research is important and meaningful. I hope that further basic research on CAVI is actively pursued and believe that it will help us understand elastic and muscular arteries, which is an important aspect of CAVI.

Conflict of Interests

The author declares no conflicts of interest.

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