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Editorial

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Further Role of Blood Pressure-Independent CAVI in Addition to a Predictor of Cardiovascular Events

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Recently, Kirigaya *et al.* have published important paper in Journal of Atherosclerosis and Thrombosis (JAT)[1]. They reported that arterial stiffness assessed using the cardio-ankle vascular index (CAVI) was a significant predictor of major adverse cardiovascular events (MACE) and death for 62 months in patients with acute coronary syndrome (ACS) after adjustment for the established coronary risk factor. The present report strengthened the significance of CAVI as a predictor for MACE and cardiovascular death.

The significance of the independency of CAVI from blood pressure is sometimes misunderstood. In most epidemiologic studies, CAVI is correlated with blood pressure. On the basis of this, some people sometimes claimed that CAVI is dependent on blood pressure. This claim is completely misunderstanding. The independency of CAVI from blood pressure is merely from blood pressure at the time of measurement. It is clear that hypertension for long term enhances arteriosclerosis and increases CAVI.

From the other aspect, a criticism regarding the independency of CAVI from blood pressure was raised by Sproncks *et al.*[2]. They claimed that CAVI was theoretically dependent on blood pressure, and they proposed a variant equation named CAVI$_0$. The editors of Journal of Hypertension supported them[3]. Sproncks *et al.*'s equation apparently appeared good on paper, but did not actually work. The refutation was published in JAT[4]. In short, using the VaSera system to measure pulse wave velocity from the origin of the aorta to the ankle, CAVI$_0$ brought unreasonable results, in which CAVI$_0$ of young hypertensive women was lower than that of normal women. The reason was high dependency of CAVI$_0$ on diastolic pressure. This argument was nearly settled. The independency of CAVI from blood pressure at the time of measurement became more certain after various discussions.

CAVI is composed of organic stiffness and functional stiffness[5], and the latter function is regulated by vasoactive compounds[6]. High CAVI was decreased during the improvement of congestive heart failure in a relatively short time, suggesting the involvement of CAVI in the formation of cardiac dysfunction as a vascular function[7]. A new field of vascular function cooperating with cardiac function could be developed using CAVI. Further studies are required to establish the meaning of CAVI as an index of vascular function in addition to a marker of arteriosclerosis.

Conflict of Interest

The author has no conflict of interest in this paper.

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