With increasing age, the aorta and arterial system experience various changes. The representative features are arterial wall stiffness and luminal dilatation. The main underlying pathologies of medial wall degeneration are breakdown of matrix protein, loss of smooth muscle, and accumulation of mucoid material. In addition to these pathologies, endothelial dysfunction and atherosclerosis occur. A typical clinical phenotype of these pathologic processes is aortic aneurysm. However, even before such an apparent stage, cellular and tissue pathologies are occurring. Therefore, each component of vascular aging could serve as a surrogate marker of clinical cardiovascular disease. Thus, one of the most convenient tools for such detection is brachial-ankle pulse wave velocity (baPWV).

In this issue of Journal of Cardiovascular Imaging, Ki et al. analyzed 263 study patients (mean age 63 ± 11 years, 71% male) who were free of overt cardiovascular disease. The main results of this study were: 1) the female group presented larger aortic root diameters (indexed by body surface area [BSA]) and a higher value of baPWV compared with the male group and 2) baPWV presented a correlation with sinotubular aortic diameter/BSA in the multivariate analysis (in which age, blood pressure, HbA1c, total cholesterol, glomerular filtration rate, and anti-hypertensive medication use were adjusted). Interestingly, that correlation was significant only in the female group ($\beta = 0.407$, $p < 0.001$). Echocardiography is one of the most available tools in routine clinical practice, and baPWV is increasingly applied due to its convenience. The noted study has strength in that the researchers combined these widely available modalities based on the ‘clinical concept of vascular ageing.’ But, the authors did not present a plausible underlying mechanism for the sex difference in the relationship between baPWV and aortic dilatation.

The sex difference in cardiovascular diseases is a current research topic. A number of studies have addressed sex differences in clinical manifestations, pathophysiology, and prognosis of hypertension, heart failure, coronary artery disease, and valvular heart disease. Simply, the action (or withdrawal) of estrogen could be a main determinant for the sex-dependent pathophysiology of cardiovascular disease. However, the sex difference in clinical medicine is multi-factorial with various modifiers. For example, there are many sex-dependent cardiovascular factors such as ventricular size and compliance, pattern of ventricular hypertrophy, microvascular circulation, myocardial oxygen consumption, arterial elasticity,
and pulmonary vascular reactivity. Also, other non-cardiovascular factors such as the immune system, pregnancy, and childbirth potentially play a role in the sex-specific clinical phenotypes of cardiovascular disease.

The significance of the noted study is limited to hypothesis generation. As arterial stiffness is a net effect of aging and various systemic factors, the pathogenesis can vary from person to person and has even been shown to differ according to the aortic level in a single person. Accordingly, the use of baPWV as a surrogate marker may differ depending on patient characteristics. The sex-dependent difference can be reasonably understood in this heterogeneous background. Finally, the authors deserve to be commended for their meaningful contribution. Hopefully, there will be further studies to identify the biologic mechanisms of sex-specific differences in arterial stiffness.

REFERENCES