Aortic Compliance and Left Ventricular Diastolic Function
– Do Endovascular Repairs for Aortic Aneurysm Alter Aortic Stiffness? –

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Endovascular aortic repair or endovascular aneurysm repair (EVAR) is a type of endovascular surgical treatment for abdominal aortic aneurysm (AAA) or thoracic aortic aneurysm (TAA). The procedure for treating TAA is specifically termed TEVAR (thoracic endovascular aortic aneurysm repair). Ruptured AAA has a mortality rate of approximately 90%; however, elective AAA repair is associated with a mortality rate of <5%.1 Aortic aneurysm is a life-threatening condition that necessitates consideration of repair, and patients at high risk for open surgery are EVAR candidates, making preoperative risk stratification important.2 Takeda et al demonstrated that EVAR increases vascular stiffness and induces left ventricular (LV) diastolic dysfunction, concluding that EVAR can worsen exercise tolerance, especially in patients with low LV distensibility.3

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The aorta is an elastic artery with regular elastic laminae between layers of smooth muscle cells in the tunica media. By acting as an elastic buffering chamber behind the heart, the aorta stores approximately 50% of the LV stroke volume during systole. In diastole, the elastic forces of the aorta propel this volume to the peripheral circulation, thereby converting the pulsatile arterial blood flow into a nearly continuous peripheral blood flow.4 The interaction between the ejected blood volume and the compliance of large elastic arteries is referred to as the Windkessel effect (Figure). The Windkessel function of the aorta influences not only on the peripheral circulation, but also heart. The extension of the aortic wall reduces LV afterload, and the blood volume stored within the distended aortic wall augments coronary perfusion.4

The stiffness of the aorta, which resists its systolic distension, increases with aging, hypertension, diabetes, abdominal visceral fat, and smoking.5 Aging plays a major role in the process of aortic degenerative disease. Pathologically, fragmentation and loss of elastic fibers of the aortic wall cause dilatation of the vessel. Subsequent repair involving replacement fibrosis with stiffer collagen increases wall stiffness. Concomitant hypertension and accelerated atherosclerosis may also contribute to both development of aortic aneurysm and decreased aortic compliance.5 Hypertension and arterial stiffness interact with each other in a bidirectional manner and are closely associated with age. Reduced aortic compliance reportedly induces LV hyper-

Figure. Windkessel is a German word meaning air chamber. When water is pumped into the chamber, the water both compresses the air in the enclosed room and pushes water back out of the chamber. The effect is to transform the pulsatile water output of pump into an almost continuous flow. The compressibility of the air in the chamber simulates the extensibility and elasticity of the aorta.
trophy.7 The exact etiology of aortic aneurysm is unclear, but smoking, atherosclerosis, hypertension and aging are thought to be risk factors, which are almost common features of patients with diastolic heart failure. LV hypertrophy and arterial stiffening could influence LV diastole by increasing the systolic load to prolong relaxation, compromise filling, and increase end-diastolic pressure.8

Aortic pulse wave velocity (PWV) provides information regarding the time taken for a pressure wave to travel a known distance. A higher aortic PWV denotes increased arterial stiffness. PWV is calculated on the basis of the Moens–Korteweg formula under the assumption that there are no significant changes in the vessel cross-sectional area or wall thickness along the arterial segment.9 In patients with aortic aneurysm, PWV values generally increase10–13 after the repair, but interpretation of this finding requires caution. PWV is defined as: $\sqrt{E} = \frac{2\pi R}{\rho h}$, where $E$ is Young’s modulus of the arterial wall, $h$ is wall thickness, $R$ is arterial radius at the end of diastole, and $\rho$ is blood density. In cases of aortic aneurysm, however, dilatation of the aortic radius obviously violates the assumption. Thus, to begin with, PWV may not represent the true degree of aortic stiffness in such cases. The restored radius of the aorta by aortic repair may influence PWV. And, because the aortic arch contributes approximately 50% of total arterial compliance,7 EVAR for AAA may differ from TEAVR regarding the increase in aortic stiffness. In contrast, there is also a report that brachial–ankle PWV (baPWV) is less influenced by the presence of AAA. The baPWV can be conveniently measured using pressure cuffs on the 4 extremities and is a measurement of aortic mechanical properties between the brachial artery and ankle artery. But the length of an AAA is less influenced by the presence of AAA. The baPWV can be a convenient measure of aortic mechanical properties.

However that may be, the Windkessel effect could be attenuated by EVAR, because the elastic properties of the aortic media are absent in the arterial segment that is replaced, especially if noncompliant stent-grafts are extensively used. Normalcy of LV systolic function in 50% of patients with heart failure implicates diastolic dysfunction as the probable etiology, whereas most patients with isolated LV diastolic dysfunction do not have the clinical features of heart failure.15 Decreased aortic compliance following EVAR may result in increased LV afterload and wall stress, predisposing to LV hypertrophy. And, LV diastolic filling and subendocardial blood flow may be deteriorated. Consequently, especially in patients who have LV diastolic dysfunction as well as aortic aneurysm, repair may aggravate or even cause the clinical syndrome of heart failure in the long term.

References