Ventricular-arterial Dysfunction in Cardiovascular Disease

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1. Ventricular-vascular interaction: Introduction and hemodynamic significance

The interaction of the heart with the systemic vasculature, termed ventricular-vascular interaction, is a central determinant of net cardiovascular performance. Ventricular-vascular coupling was defined as the ratio of effective arterial elastance to left ventricular end-systolic elastance. Vascular stiffening of the large arteries is a common feature of aging and is exacerbated by many common disorders such as hypertension, diabetes, and renal disease. Many investigators studied the changes in ventricular-vascular coupling and implications on hemodynamics in normal and diseased hearts.

The major effects of ventricular-vascular stiffening:

1) *an enhanced lability of cardiovascular function*
2) *limited reserve capacity*
3) *greater pressure sensitivity to venous and arterial dilators or blood volume alterations*
4) *greater likelihood of poorly matched heart-artery systems with cardiac failure*
5) *compromised coronary flow and exacerbated responses to myocardial ischemia*

2. Ventricular-vascular interaction: a major pathophysiology in heart failure with preserved ejection fraction

Abnormal ventricular-vascular coupling due to stiffening of both systems may contribute to heart failure with preserved ejection fraction. Vascular stiffening of large arteries is a common feature and exertional dyspnea is a common symptom especially in elderly and/or hypertensive patients even if they have normal systolic function at rest. In healthy normal subjects, the effective arterial elastance is nearly one half of left ventricular elastance and the change of ventricular vascular interaction with exercise is characterized by a decreased in the ventricular-vascular coupling index, indicating an augmented of pump efficiency (i.e., a relative increase in ventricular contractility than arterial load). On resting status, ventricular vascular interaction can be maintained because the left ventricle is hypertrophied compensatory to overcome the increased arterial stiffness in elderly or hypertensive patients. During exercise, uncoupling of ventricular vascular interaction might develop in elderly and/or hypertensive patients with

limited effort tolerance and it would be associated with reduced LV functional reserve.

Left ventricular structure and functional reserve are likewise influenced by hypertension and it is known that the increased afterload in systemic hypertension may induce adaptive changes of left ventricular structure. A few studies have focused on ventricular-vascular coupling in hypertensive patients, but there was no difference in the index of ventricular-vascular coupling in hypertensives compared with normotensives. The resting Ea/Ees ratio in hypertensive patients was on average close to 0.50, similar to normal values, which indicate optimal ventricular-vascular coupling. However, exertional dyspnea is quite common in these patients and therefore it can be suggested that the change of ventricular-vascular coupling, a net cardiovascular performance could be abnormal in hypertensive patients. Najjar et al. demonstrated that age-associated differences in ventricular-vascular interaction in both genders during exercise. Resting ventricular-vascular coupling was not age related in men or women. In both genders, Ea/Ees decreased during exercise and declined to a lesser extent in older subject. In elderly women, arterial stiffening and ventricular-vascular uncoupling became prominent with exercise. So, ventricular-vascular uncoupling during exercise maybe a major pathophysiology of heart failure with a preserved ejection fraction predominantly affected in elderly women.

References

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