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Ventricular-Arterial coupling during dipyridamole stress

T. Bombardini[1], S. Gherardi[2], F. Rigo[3], L. Pratali[2], E. Pasanisi[2], E. Picano[4]
[1]Cnr, Institute Of Clinical Physiology, Pisa, Italy; [2]Cesena Hospital, Cesena, Italy; [3]Mestre Hospital, Mestre, Italy; [4]Cnr, Institute Of Clinical Physiology, Pisa, Italy

Background: The interaction of the heart with the systemic vasculature, termed ventricular-arterial coupling, is a central determinant of net cardiovascular performance in normal and pathological conditions. Ventricular and arterial elastance can be easily assessed by echocardiography, both at rest and during stress.

Aim: To assess noninvasively left ventricular-arterial coupling in healthy and diseased subjects at rest and during dipyridamole (DIP) stress.

Materials and methods: We enrolled 365 patients (63±16 years; 231 males) referred to stress echo lab: 131 \"normals\" (Nl); 86 patients with coronary artery disease, 68 with negative (CAD, SE -) and 18 with positive (CAD, SE+) stress echo; 148 with idiopathic dilated cardiomyopathy (DCM). In all, ventricular-arterial coupling was indexed by the ratio of ventricular force (Systolic Pressure/End-Systolic Volume index) to arterial elastance (EaI, ratio of end-systolic pressure by stroke volume). 2D echo (for ESV and stroke volume) and cuff sphygmomanometer (systolic pressure, multiplied x 0.90 to obtain end-systolic pressure) provided the raw measurements.

Results: At rest, EaI was profoundly increased in DCM (6.3±4.4; p<.001 *vs.* all other groups: Nl=4±1.1; CAD, SE-=3.8±1; CAD SE+=4.2±1.3). DIP maximized ventricular-arterial coupling in normals. Residual vasodilatation and contractile reserve slightly increased cardiac efficiency in DCM and in CAD SE- pts. The CAD SE+ pts showed negative contractile reserve and the worse stress ventricular arterial coupling (see figure).

Conclusions: Ventricular-arterial coupling was optimized by DIP in normals, and disrupted in CAD patients with stress induced ischemia. Effective arterial elastance is dramatically increased in DCM at rest and weakly responds to vasodilator stress.