Hemodynamic heterogeneity of inadequate cardiac output increase identified by 2-dimensional volumetric exercise echocardiography: slow, stiff or weak heart?

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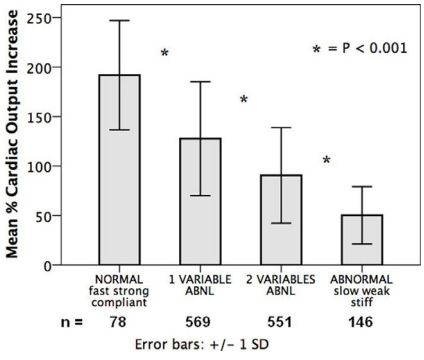
Background: Two-dimensional (2-D) volumetric exercise stress echocardiography (ESE) provides an integrated view of preload reserve through end-diastolic volume (EDV) and left ventricular contractile reserve (LVCR) through end-systolic volume (ESV) changes.

Purpose: To assess the dependence of stroke volume (SV) and cardiac output (CO) upon LVCR EDV changes and heart rate (HR) during ESE.

Methods: We prospectively performed semi-supine bicycle or treadmill ESE in 1,344 patients (age 59.8±11.4 years; 550 female; ejection fraction = 62.5±8%) referred for known or suspected coronary artery disease in 20 quality controlled laboratories of 16 countries from 2016 to 2019. SV was calculated at rest and peak stress from raw measurement of LV EDV and ESV by biplane Simpson rule, 2-D echo. LVCR was the stress-rest ratio of force (systolic blood pressure by cuff sphygmomanometer/ESV, abnormal values <2.0 identify a "weak" heart). Preload reserve was defined by an increase in LV EDV. Abnormal values (lack of EDV increase, peak EDV $\leq$ rest EDV) identify a "stiff" heart. Cardiac output was calculated as SV * HR (measured with standard EKG). HR reserve (stress/rest ratio) <1.85 identifies a "slow" heart with chronotropic incompetence.

Results: By selection, all patients had negative SE by wall motion criteria. Of the 1,344 patients included in the study, 448 belonged to the lowest tertile of CO increase. Of them 326 (73%) achieved HR reserve <1.85; 220 (49%) had a blunted LVCR and 374 (83%) a reduction of preload reserve, with 348 patients (78%) showing ≥ 2 abnormalities. The more the abnormal criteria, the worse the CO response, which was lowest in slow, stiff and weak hearts: see figure.

Conclusion: Patients with normal CO reserve during exercise usually have a fast, compliant and strong heart. Abnormal CO reserve is associated with heterogeneous hemodynamic responses, with slow, stiff and/or weak hearts. The clarification of underlying hemodynamic heterogeneity is the prerequisite for a personalized treatment, and can be easily extracted from a standard 2-D volumetric SE. Hearts with normal CO are all alike; every heart with abnormal CO is abnormal in its own way.



CO % changes in subsets (*p<0.001)