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Left atrial diastolic and systolic functions modulate the response to the standardised Valsalva maneuver in normal subjects

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Background. Although the Valsalva maneuver (VM) is being advocated by current guidelines to identify with echocardiography patients with increased left ventricular (LV) filling pressures using a decrease in mitral E/A velocity > 0.5 as cutoff, there are limited published data for both patients and the normal response to the maneuver in healthy subjects.

Purpose. To assess LV and left atrial (LA) physiology during a standardized VM (VMs) in normal subjects.

Methods. The VMs was performed in 50 healthy subjects (M:F 38:12; age 40 ± 12 y.; HR 70 ± 11 bpm; BSA 1.81 ± 11 m2), instructed to forcefully exhale for 20 seconds without an initial deep breath into a tube connected to a sphygmomanometer, maintaining a 25-35 mmHg pressure. The VM was repeated 2 times at 5 minute intervals to record sequentially in the apical 4-chamber view: 1. LV and LA volumes; 2. Transmitral flow velocities. LA diastolic reservoir function (LAres) was calculated as: (maximum – minimum volume) / minimum volume x 100. Results. During the VMs, in all subjects LV indexed end-diastolic (-14 \pm 7 ml/m2, -31 \pm 15 %) and end-systolic (-6 \pm 4 ml/m2, -31 \pm 18 %) volumes, and stroke volume index (-9 \pm 5 ml/m2, -30 \pm 15 %) decreased similarly with unchanged LV ejection fraction %, and LA maximum and minimum volume indices both decreased (respectively -8 \pm 6 ml/m2, -3 \pm 3 ml/m2; -32 \pm 25 %) with high variability. Mitral peak E velocity also decreased (-22 \pm 13 cm/s, -27 \pm 14 %) in all subjects, whereas peak A velocity change varied, such that a "pseudo-abnormal" decrease of E/A > 0.5 was seen in 18 subjects (37 %). At baseline, this subgroup had lower heart rate (66 \pm 11 vs 73 \pm 10 bpm, p= .026), higher LAres (193 \pm 67 vs 145 \pm 47 %, p= .006), lower peak A velocity (50 \pm 12 vs 58 \pm 12 cm/s, p= .04) and higher E/A (1.8 \pm .6 vs 1.4 \pm .3, p= .004). During VMs, LV and LA volumes decreased similarly in all subjects, but increase in heart rate was higher (12 \pm 8 vs 6 \pm 5 bpm, p= .023), and peak A wave increased instead of decreasing (20 \pm 20 % vs -8 \pm 18 %, p< .001) in the subjects with "pseudo-abnormal" decrease of E/A. During VMs, decrease in E/A was mainly determined (regression analysis, r: .76, p= .029) by baseline LAres (B= -.71) and change in LAres during VMs (B= -.47), whereas an increase in peak A velocity (r: .46, p= .031) was mainly determined by degree of HR increase (B= .41) and baseline LV EF (B= .3).

Conclusions. During VMs, a "pseudo-abnormal" decrease of the E/A velocity ratio is present in almost 40 % of normal subjects, and is determined by the interplay of the baseline diastolic compliance and the increase in systolic function of the LA during VM. These results may influence the accuracy of the VMs in the detection of increased LV filling pressures in patients.

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