Results: E/E' did not change significantly during exercise (20.6±8 vs 20.5±12 at rest and exercise, respectively). The increase in the IVRT was associated with a decrease in stroke volume (3.2±17 vs 15±15, p<0.0005), an increase in MR severity (in ERO 3±2 vs 5±9 mm², p<0.001), in transmural pressure (25±12 vs 16±13 mmHg, p<0.012), and in EF peak (45±26 vs 26±22 cm/s, p<0.006). Patients who stopped for dyspnea presented a larger rise in transmural pressure and gradient, in MR severity and in E/E' at peak test. With multivariate analysis, dynamic increase in MR severity at exercise remained the sole predictor of exercise-induced dyspnea.

Conclusion: In patients with systolic heart failure, changes in E/E' during exercise are highly variable from patient to patient and are related to both the dynamic component of MR and reduced LV compliance.

273 Systolic and diastolic function in patients with acute pulmonary edema and hypertension

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Background: Patients (pts) hospitalized with heart failure may later be noted to have normal systolic function, as evidenced by a normal left ventricular ejection fraction (LVEF). In this perspective, the main hypothesis has been presumed to be due to isolated diastolic dysfunction. Pts with acute pulmonary edema (APE) often have marked systolic hypofunction but, after reduction of the blood pressure, have a normal left ventricular EF (<50%). However, the left ventricular (LV) EF is usually evaluated after the clinical status has resolved. Thus, it is possible that APE was not the result of diastolic dysfunction but instead, was due to transient systolic dysfunction, acute mitral regurgitation (MR) or both.

Aim: To test the hypothesis that many pts with hypersensitive APE have diastolic dysfunction.

Methods: We studied 48 pts (26 men and 22 women; mean age 66±14 years) with APE and a systolic blood pressure >160 mmHg. Two-dimensional transesophageal echocardiography with color Doppler imaging was performed in each patient as therapy was being initiated, a second echocardiogram was obtained 24–48 hours after clinical stabilization had occurred, so that the patient was normotensive and pulmonary congestion resolved. We evaluated the EF (Simpson), the presence and severity of any MR, the wall motion score index (WMSI), the segment model.

Results: The mean systolic blood pressure was 197±10 mmHg during the initial transesophageal echocardiogram and was reduced to 132±16 mmHg (p<0.05) at the time of follow-up examination. The EF was similar during the acute episode (52±18%) and after treatment (51±15%). The EF after treatment correlated directly with the EF during the acute episode (r=0.85, p<0.01). The left ventricular WMSI was also the same during the acute episode (1.5±0.5) and after treatment (1.5±0.5). The WMSI at follow up correlated directly with the index at presentation (r=0.95, p<0.01). No patient had severe MR during the acute episode. 26 pts (54.2%) had normal EF (>50%) after treatment. In 20 (80.4%) of these 26 pts the EF was ≥50% during the acute episode.

Conclusions: In pts with hypertensive APE, the EF during the acute episode is similar to that after treatment, whereas, when the blood pressure has been restored and pulmonary congestion resolved. A normal EF after the treatment of a patient with hypertensive APE indicates a high probability that pulmonary congestion was due to isolated, transient diastolic dysfunction, since transient systolic dysfunction and/or severe MR are infrequent during acute episodes in these pts.