Atrial fibrillation impairs dynamic right ventricular-pulmonary artery coupling and increases lung congestion during exercise in heart failure and preserved ejection fraction

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Background: Atrial fibrillation (AF) is common in patients with heart failure and preserved ejection fraction (HFpEF) and associated with left atrial (LA) myopathy, reduced exercise capacity and poor outcomes. However, the mechanisms underlying exercise intolerance in HFpEF with AF are not well-characterized.

Purpose: To test hypotheses that patients with HFpEF and AF (HFpEF-AF) would display greater acute impairments in right heart-pulmonary vascular coupling during exercise, leading to greater elevations in left and right heart filling pressures and increased lung congestion as compared to patients with HFpEF and no AF (HFpEF-no-AF) and control subjects free of heart failure and AF. As corollary hypotheses, we also posited that these changes would lead to greater ventricular interdependence during exercise in HFpEF-AF, and that pulmonary hypertension in HFpEF-AF was not simply ascribable to the LA dysfunction that accompanies AF, which would provide greater impetus to pursue restoration of sinus rhythm.

Methods: Subjects with HFpEF-AF (n=35), HFpEF-no-AF (n=85), and controls free of heart failure or AF (n=28) underwent cardiopulmonary exercise testing with invasive hemodynamic assessment and simultaneous echocardiography and lung ultrasound in a prospective study.

Results: As compared with controls and HFpEF-no-AF, subjects with HFpEF-AF displayed poorer left ventricular (LV) longitudinal strain, lower LA reservoir strain with greater LA volume at rest. With exercise, subjects with HFpEF-AF displayed more severe exercise-induced pulmonary hypertension, higher right atrial pressure (RAP) and more pronounced right ventricular-pulmonary vascular uncoupling. Peak oxygen consumption was lowest in patients with HFpEF-AF, coupled with greater limitations in cardiac output reserve and more severe increases in lung congestion and pulmonary vascular resistance (Figure 1). Dynamic ventricular interaction was greatest in HFpEF-AF, evidenced by greater increases in LV eccentricity index and RAP/PCWP (Figure 2) and less increase in LV transmural pressure compared with HFpEF-no-AF. In a sensitivity analysis, patients with HFpEF-AF displayed more severe pulmonary hypertension and pulmonary vascular disease compared to HFpEF-no-AF even after matching for the severity of LA remodeling and dysfunction.

Conclusions: Patients with AF and HFpEF display more severe abnormalities in pulmonary vascular reserve with exercise leading to greater lung congestion and enhanced exertional ventricular interdependence compared to patients with HFpEF-no-AF. While many of these differences appear attributable to LA dysfunction, the presence of AF is associated with more severe pulmonary vascular disease independent of LA myopathy, suggesting potential benefit from restoration of sinus rhythm even in the setting of LA myopathy.

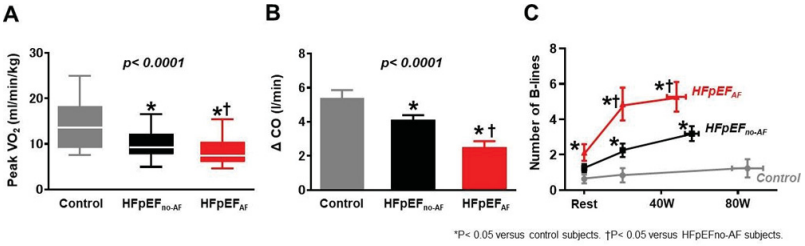

Figure 1

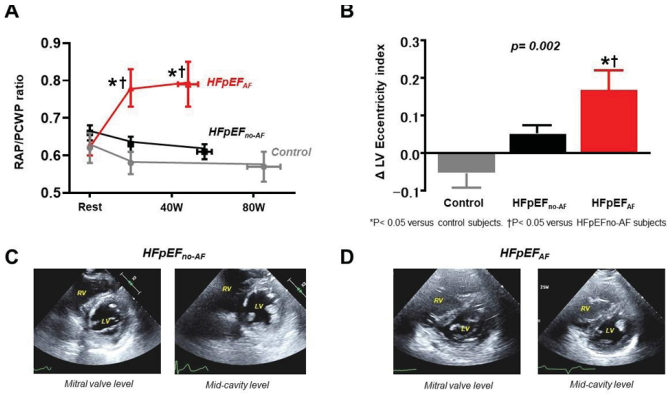

Figure 2