A pro-thrombotic left atrial haemodynamic profile is present in older individuals with cardiovascular risk factors even without atrial fibrillation

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Background: Atrial fibrillation (AF), by inducing left atrial (LA) stasis and thrombus formation, is a major cause of ischaemic embolic stroke. However, up to 25% of embolic strokes occur in the absence of AF or any other identifiable mechanism. We hypothesised that exposure to risk factors leads to a prothrombotic LA haemodynamic profile, with a consequent risk of thrombus formation and embolic stroke even in sinus rhythm (SR).
Purpose: To quantify the relative effects of AF and risk factors on LA function and haemodynamics in three groups: (1) patients with persistent AF before and after cardioversion, (2) patients in SR with no history of AF who were matched to AF patients for stroke risk, and (3) healthy controls.
Methods: Ninety-five participants were recruited: 37 AF patients scheduled for cardioversion [median CHA2DS2-VASc = 2 (1.5–3.5); median age 69 years], 35 matched non-AF patients [median CHA2DS2-VASc 3 (2.0–4.0); median age 69 years], and 23 healthy subjects [median CHA2DS2-VASc 0 (0–0); median age 33 years]. Advanced cardiovascular MRI was used to assess LA function and 4D flow.
Results: Patients with persistent AF displayed significantly lower LA emptying fraction (LAEF), reservoir strain, and peak blood flow velocities compared to both control groups in SR, as well as profound alterations in atrial blood flow patterns and vortex volume ratio (all $p<0.001$ vs both control groups). In the 65% of patients who were in SR at $\geq$4 weeks post cardioversion, all LA parameters were significantly improved (all paired $p<0.001$ vs baseline scan in AF; red vs blue in Figure 1), but LA haemodynamics were now no longer different from those in matched non-AF patients (all p=ns; blue vs green in Figure 1 C-D). However, both AF patients cardioverted to SR and matched non-AF patients had significantly impaired LA function (LA EF and LA reservoir strain) and LA haemodynamics (lower peak velocity and higher vortex volume ratio), compared to heathy controls (all $p<0.01$; blue/green vs grey in Figure 1).
Conclusions: We found evidence of a gradient of LA haemodynamic dysfunction, where the most marked phenotype was seen in patients scanned in AF, but with significant abnormalities remaining after restoration of SR in AF patients, and intriguingly, also present to a similar degree in matched non-AF patients. Advanced atrial imaging offers the potential to select patients with an adverse pathophysiological substrate directly predisposing to LA thrombus formation for future clinical trials of anticoagulation in SR.

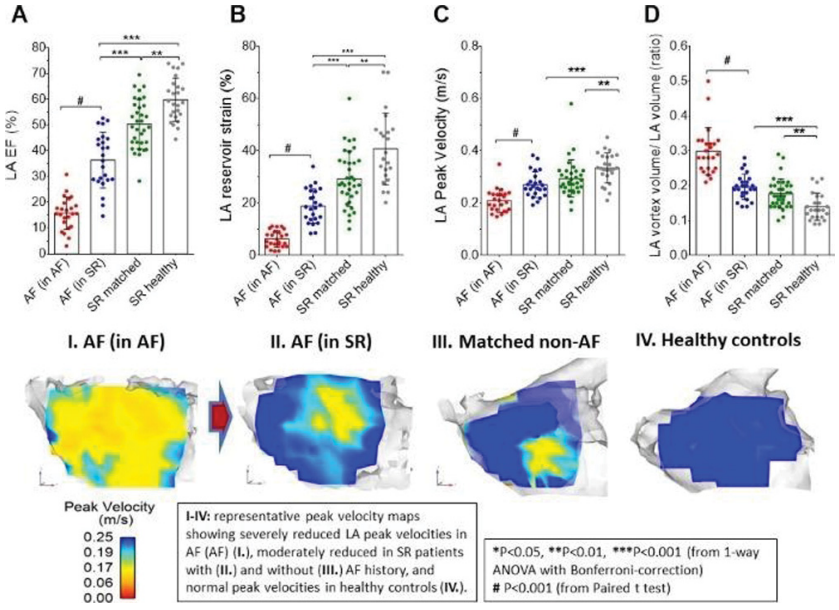

Figure 1