and RAA while T cell receptor sequencing confirmed the same T cell clones to be present in the RAA as the EAT.

**Conclusions and Implications** AF carries a unique EAT-resident T cell signature which correlates with the production of the pro-inflammatory cytokines interferon-γ and interleukin-17. Single-cell RNA-sequencing analysis confirms EAT to be the immune reservoir of the heart and EAT sampling can provide an accurate readout of the immune landscape of the underlying cardiac tissue. Targeting this local resident T cell population may unlock a novel angle in the management of the inflammatory and fibrotic components of AF genesis.

**BS7 NEUROHUMORAL RESPONSES IN TAKOTSUBO SYNDROME**

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**Background** We investigate if renin-angiotensin and endothelin-1 response pathways follow the same pattern of recovery as left ventricular ejection fraction in patients with takotsubo syndrome.

**Methods** Ninety takotsubo syndrome patients [n=30 in each of ‘acute’, ‘convalescent’ (3–5 months) and ‘recovered’ (> 1 year) groups] who were on minimal or no medication and were free of any significant cardiac/metabolic co-morbidities, and 30 healthy controls were studied. Serum concentrations of renin, angiotensin converting enzyme, angiotensin II, big endothelin-1, endothelin-1 were measured using commercially available ELISA, and BNP was measured using an immunoassay.

**Results** Left ventricular ejection fraction was 38 ± 1.6 % in acute, 63 ± 2.0 % in convalescent and 64 ± 2.6 % in recovered takotsubo syndrome patients. As shown in the Figure, serum renin concentrations are persistently elevated after a takotsubo episode (p=0.03 vs controls). Angiotensin converting enzyme levels are significantly depressed during the acute phase compared to convalescent (p=0.004), recovered takotsubo (p=0.02) or controls (p=0.03). Angiotensin II is increased in takotsubo patients (p<0.001 vs controls) remaining persistently elevated long-term in the recovered group (p=0.03 vs controls). B-type natriuretic peptide concentrations remain elevated after a takotsubo episode compared to controls (p=0.003). Big endothelin-1 levels are unchanged, but endothelin-1 is significantly lower after takotsubo syndrome compared to controls (p=0.03).

**Conclusions** Despite ‘normalisation’ of the left ventricular ejection fraction, there is long-term maladaptive activation of renin-angiotensin system in takotsubo syndrome patients. This suggests therapy aimed at modulating this pathway may be beneficial in the long-term.