Introduction: Background The most common mechanism of ventricular tachycardia (VT) in patients with structural heart disease (SHD) is scar-related re-entry. Focal VT is typically seen in patients without SHD and is rarely seen as a dominant mechanism in patients with established or acute ischaemic heart disease (IHD). There is a paucity of data characterising focal VTs remote or adjacent from regions of dense scar in patients with SHD. In this study, we report the frequency, procedural characteristics and subsequent clinical outcomes in a series of patients with SHD shown to have a focal mechanism despite the presence of scar and the typical electrophysiological milieu for re-entrant VT. Aim To summarise the procedural characteristics and outcomes of patients with structural heart disease (SHD) who have focal VT.

Methods: Consecutive patients with SHD undergoing VT ablations over the previous 2-year period were included. Patients were included with SHD (ischaemic or non-ischaemic) who were referred for catheter ablation for medically refractory VT. In addition to using programmed electrical stimulation (PES) during induction, we included burst RV pacing and an isoprenaline protocol pre- and post ablation.

Result: Nineteen of 112 patients with SHD (17%) undergoing VT ablation over 2 years had a focal VT mechanism elucidated (mean age, 67±13 years; ejection fraction, 46±14%; non-ischemic cardiomyopathy 10). Repetitive failure of termination with anti-tachycardia pacing (69% of patients) or defibrillator shocks (56%) was a common feature of focal VTs. A median of 3 VTs/patient were inducible (28 focal, 34 re-entrant VTs; 53% of patients with both focal and re-entrant VT mechanism). Focal VTs originated from the right ventricle (RV: 68%) than the left ventricle (LV: 32%) (Figure). In the RV, the RV outflow tract was the most common (37% of all focal VTs), RV moderator band (21%), apical septal RV (1 patient; 5%) and lateral tricuspid annulus (1 patient; 5%). The lateral LV (non-Purkinje) was the most common LV focal VT site (16%) followed by the papillary muscles (15%). After a median follow-up of 276 days, 79% of patients remained arrhythmia-free; no patients had recurrence of focal VT at repeat procedure. In those with recurrence, defibrillator therapies were significantly reduced from a median of 53 anti-tachycardia pacing (ATP) episodes pre-ablation to 10 ATP episodes post ablation. During follow-up, 2 patients (11%) underwent repeat VT ablation; none had recurrence of
focal VT.

**Conclusion**: Focal VTs are common in patients with SHD often coexisting with re-entrant forms of VT. High rates of failure of defibrillator therapies was a common feature of focal VT mechanism. Uncovering and abolishment of focal VT may result in further improvement in outcomes of catheter ablation in SHD.