Introduction: Sinoatrial dysfunction and atrial arrhythmias frequently coexist in presence of structural heart disease (SHD). While atrial remodelling is frequently implicated, there is paucity of data on electrical activation from an endocardial-epicardial perspective. We tested the hypotheses that electrical dissociation and signal fractionation will be present in the sinus node region with overdrive suppression with SHD. We also assessed activation and conduction dynamics of the sinus node region from an endocardial-epicardial perspective.

Methods: Simultaneous intra-operative mapping of the endo- and epicardial aspects of sinus node region was performed during sinus rhythm at baseline (SRb) and after overdrive pacing (SRd) using two Advisor® HD Grid catheters (Abbott, 16 electrodes, 3mm inter-electrode spacing). Unipolar EGM’s and 3-D electrode locations were exported into custom-made software for phase mapping, activation times and for assessing signal complexities and conduction velocities (CV). Difference ≥20ms between paired endo- and epicardial electrodes defined dyssynchrony. Unipolar electrograms with ≥5 deflections occupying were classified as fractionated.

Result: Sixteen patients (mean age: 60.5±4.1years, 18.7% history of AF) with SHD (43% ischemia, 57% valvular disease) were included. 4862 EGM’s analysed. With SRb, 90%±0.2% of activations were synchronous, but this significantly reduced to 85.1%±0.2% (p<0.0001) with SRd with a tendency towards epicardial exits (46.3% vs 40.3%). Overall, 44.4% of the unipolar EGM’s in the sinus region showed fractionation. Although the proportion of complex signals were similar on epicardial and endocardial surfaces (49.5% vs 50%), fractionated signals were more frequent during sinus rhythm post over drive pacing than at baseline (48% vs 39.5%, p<0.0001). Mean CV’s did not differ between endo- and epicardial surfaces during SRd (53.9 vs 51.7 cm/s, p = 0.545)

Conclusion: Significant endocardial-epicardial electrical dissociation and electrogram fractionation is evident in the sinus node region post overdrive suppression in patients with SHD with a tendency towards
epicardial exit. Such complex 3D interaction in electrical activation provides further mechanistic insights for arrhythmogenesis in the presence of SHD.