1. **Dronedarone in high-risk permanent atrial fibrillation.** Connolly SJ, Camm AJ, Halperin JL, et al, PALLAS Investigators. N Engl J Med 2011;365:2268–76. doi: 10.1016/S0019-4832(12)60031-5

Dronedarone is a new anti-arrhythmic drug developed for the management of atrial fibrillation (AF) and in major trials like Euridis/Adonis and Athena has been shown to reduce AF recurrence, slow the ventricular rate and reduce cardiovascular outcomes and deaths in patients with paroxysmal and persistent AF. In the background of these studies, PALLAS trial was designed with the hypothesis that patients with high-risk permanent AF would similarly benefit from dronedarone. This trial included patients aged ≥65 years with permanent AF of ≥6 months duration and at least one risk factor. Patients were randomised to receive dronedarone (400 mg twice daily) or placebo and both the groups were well matched with clinical characteristics and risk factors which included coronary artery disease (CAD) (40.8% vs 41.2%), previous stroke or transient ischaemic attack (26.9% vs 28.3%), symptomatic heart failure (14.4% vs 14.8%), a left ventricular ejection fraction of 40% or less (21.3% vs 20.7%), peripheral arterial disease (PAD) (11.6% vs 13.2%); or the combination of an age of ≥75 years, hypertension, and diabetes (18.2% vs 17.1%). CHADS2 score of ≥2 was seen in 88.1% in the drug arm and 89.3% in the placebo arm. The first co primary outcome was stroke, myocardial infarction, systemic embolism, or death from cardiovascular causes. The second co primary outcome was unplanned hospitalisation for a cardiovascular cause or death. The trial was prematurely terminated for safety reasons after a year of enrolment when 3236 patients had undergone randomisation. The first co primary outcome occurred in 43 patients receiving dronedarone and 19 receiving placebo (hazard ratio, 2.29; 95% confidence interval [CI], 1.34–3.94; $P=0.002$). There were 21 deaths from cardiovascular causes in the dronedarone group and 10 in the placebo group (hazard ratio, 2.11; 95% CI, 1.00–4.49; $P=0.046$), including death from arrhythmia in 13 patients and 4 patients, respectively (hazard ratio, 3.26; 95% CI, 1.06–10.00; $P=0.03$). Stroke occurred in 23 patients in the dronedarone group and 10 in the placebo group (hazard ratio, 2.32; 95% CI, 1.11–4.88; $P=0.02$). Hospitalisation for heart failure occurred in 43 patients in the dronedarone group and 24 in the placebo group (hazard ratio, 1.81; 95% CI, 1.10–2.99; $P=0.02$). The occurrence of co primary end point doubled in the dronedarone arm predominantly due to increase in rates of stroke and death from cardiovascular causes. ANDROMEDA trial had earlier shown increased cardiovascular mortality with this drug in patients with severe LV systolic dysfunction. PALLAS reproduced this trend and conclusively showed that dronedarone increased rates of heart failure, stroke, and death from cardiovascular causes in permanent AF patients with high-risk for major vascular events and should not be used in this group of patients.

2. **Prevention of ventricular fibrillation episodes in Brugada syndrome by catheter ablation over the anterior right ventricular outflow tract epicardium.** Nademanee K, Veerakul G, Chandanamattha P, et al. Circulation 2011;123:1270–9. doi: 10.1016/S0019-4832(12)60032-7

Brugada syndrome is an autosomally transmitted channellopathy and one of the important causes of sudden death in the young with apparently normal hearts. The electrophysiological mechanisms resulting in the abnormal electrocardiogram (ECG) pattern in this syndrome are unclear. Implantable cardioverter defibrillator (ICD) is the only effective treatment to prevent sudden death in these patients but is difficult solution in the very young, in low-income communities and in those with frequent ventricular fibrillation (VF) episodes necessitating frequent ICD discharges. Haissaguerre et al. earlier showed the efficacy of radiofrequency ablation in these patients by targeting PVC's originating from right ventricular outflow tract (RVOT). This paper describes endocardial and epicardial electroanatomic mapping in 9 patients with type I Brugada syndrome having recurrent VF episodes necessitating ICD discharges. It demonstrates the presence of abnormal arrythmogenic substrate exclusively in the anterior RVOT epicardium in the form of low amplitude (<1 mv) fractionated electrograms (>130 ms duration) and polyphasic late potentials (>100 ms) after QRS complex on surface ECG) which cause delayed depolarization to cause arrhythmias. Such abnormal electrograms were not seen in the endocardial surface of the same region of RVOT. Ablation at these epicardial sites rendered ventricular tachycardia (VT)/VF non-inducible (7 of 9 patients [78%]; 95% confidence interval [CI], 0.40–0.97, $P=0.015$) and normalisation of the Brugada ECG pattern in 89% (95% CI, 0.52–0.99; $P=0.008$). Long-term follow-up (20±6 months) showed no recurrent VT/VF in all patients off medication (except 1 patient on amiodarone). This paper effectively proves the existence of a localised electrical substrate in patients with Brugada syndrome and presents the potential feasibility of radiofrequency ablation in these patients having recurrent ICD shocks or storms of ventricular arrhythmias.

3. **Endocardial radiofrequency ablation for hypertrophic obstructive cardiomyopathy acute results and 6 months' follow-up in 19 patients.** Lawrenz T, Borchert B, Leuner C, et al. J Am Coll Cardiol 2011;57:572–6. doi: 10.1016/S0019-4832(12)60033-9

Endocardial radiofrequency (RF) ablation of septal hypertrophy (ERASH) is a new approach to reduce left ventricular outflow tract (LVOT) gradient in hypertrophic obstructive cardiomyopathy (HOCM) by inducing a discrete septal contraction disorder. The purpose of this study was to study the safety and efficacy of this procedure for LVOT gradient reduction in HOCM. Nineteen severely symptomatic patients despite medications with LVOT gradients of ≥50 mmHg at rest or after provocation were enrolled.