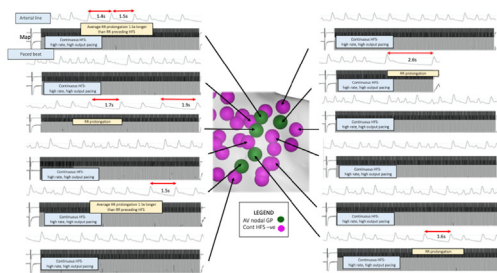


**Methods:** In 31 patients undergoing clinically-indicated AF ablation, standard mapping catheters (Navistar™, TactiCath™) were used to deliver HFS to the left atrial endocardium from either the Tau20 or Grass S88 with SIU5. AV dissociation (AVD-GP) and atrial ectopy (ET-GP) in response to HFS were tagged on 3D electroanatomic mapping systems (CARTO™ & Precision™) to assess current output threshold for GP detection, reproducibility of the Tau20, creation of high density GP maps and efficacy of RF ablation in abolishing the GP effect.

**Results:** Mean output to identify GP was 29 mA (n=12). Reproducibility comparing the two stimulators was 100% for AVD-GP (n=6) (Kappa=1, SE of kappa=0, 95% CI 1 to 1) and 100% for ET-GP (n=16) (Kappa=1, SE of kappa =0, 95% CI 1 to 1). Reproducibility using the Tau20 was 95% for AVD-GP (n=38) (Kappa = 0.64, SE of kappa =0.23, 95% CI 0.19 to 1) and 100% for ET-GP (n=13) (Kappa = 1, SE of kappa =0, 95% CI 1 to 1). Figure 1 shows a high-density GP map. Ablation abolished autonomic effects at 166 AVD-GP and 118 ET-GP (n=11).

**Conclusion:** A novel current-controlled high frequency stimulator appears to be a reproducible method for locating GP sites through physiological effect within the human left atrium.

Figure 1. A high-density GP map



The left atrial anatomy (grey) as visualised with the CARTO™ electroanatomic mapping system. Electrogram signals from the EP recording system (LabSystem Pro, Boston Scientific) are shown adjacent. GP sites green. HFS negative sites pink. Red arrows with duration above denote prolonged RR interval indicating a GP site.

### CE-521-03

#### LONG TERM OUTCOMES OF LEFT CARDIAC SYMPATHETIC DENERVATION FOR LONG QT SYNDROME AND CATHECHOLAMINERGIC POLYMORPHIC VENTRICULAR TACHYCARDIA

Johan Martijn Bos MD, PhD; Stephanie F. Polites MD; Christopher Moir MD and Michael John Ackerman MD, PhD

**Background:** Left cardiac sympathetic denervation (LCSD) is an effective anti-fibrillatory, minimally invasive therapy for patients with cardiac channelopathies such as long QT syndrome (LQTS) and catecholaminergic polymorphic ventricular tachycardia (CPVT). While performed for a few decades, only now are cohorts getting large enough to evaluate long term outcomes.

**Objective:** To examine the frequency of breakthrough cardiac events (BCEs) after LCSD in patients with either LQTS or CPVT.

**Methods:** We performed a retrospective review of all patients with either LQTS or CPVT who underwent LCSD at our institution since 2005. Clinical data was abstracted for each patient, including cardiac event rates before and after LCSD. BCEs were defined as arrhythmogenic syncope, seizure, appropriate ventricular fibrillation (VF)-terminating therapies, and/or sudden cardiac death (SCD).

**Results:** Overall, 301 patients were included [173 female (57%); mean age at LCSD  $19 \pm 13$  years] of which 235 (78%) had LQTS [121 (51%) LQT1, 56 (24%) LQT2, 13 (5%) LQT3 and 23 (10%)  $\geq 1$  LQTS mutation (LQTM)] and 66 (22%) had CPVT. Overall, 180 (60%) patients were symptomatic prior to LCSD (53% for LQTS; 85% for CPVT). Mean follow-up for the cohort was  $6.5 \pm 4.1$

years with a total follow-up of 1963 patient-years. In total, 50 (17%) patients [42/235 (18%) with LQTS and 8/66 (12%) with CPVT] have experienced  $\geq 1$  non-lethal BCE post-LCSD. Among the main LQTS genotypes, BCE rates were significantly higher in those with LQT3 (82%) or LQTM (35%) compared to LQT1 (12%) or LQT2 (11%;  $p < 0.001$ ). Additionally, among the 180 previously symptomatic patients, their post-LCSD BCE rate was significantly higher (24%) compared to 5% ( $p < 0.001$ ) for the 121 who were asymptomatic before their LCSD.

**Conclusion:** Long term follow-up continues to demonstrate that LCSD is a safe intervention with high therapeutic efficacy. However, LCSD is not a curative therapeutic option as one out of four previously symptomatic patients have experienced a channelopathy-associated BCE, albeit no deaths, following their LCSD with nearly 2000 patient-years of observation.

### CE-521-04

#### VASOVAGAL SYNCOPE WITH ISOLATED ATRIOVENTRICULAR BLOCK FOLLOWING CARDIONEURAL ABLATION DEMONSTRATING DISTINCT INNERVATION OF THE SINUS AND ATRIOVENTRICULAR NODES

Timothy Richard Maher MD; Andrew H. Locke MD; Andre d'Avila MD, PhD and Alexei Shvilkin MD, PhD

**Background:** Radiofrequency ablation (RFA) of cardiac parasympathetic ganglionated plexi (GPs), termed cardioneural ablation (CNA), has been shown to successfully treat vasovagal syncope (VVS). Sinus (SN) and atrioventricular (AVN) nodes are innervated by distinct GPs in the epicardial fat pads of both atria. We present a case of CNA of SN-specific GPs for VVS eliminating sinus arrest, but recurrent syncope with AV block.

**Objective:** To demonstrate the potential need to target both SN and AVN GPs in cardioinhibitory VVS.

**Methods:** NA

**Results:** A 36 year-old man with frequent VVS with sinus arrest documented on implantable loop recorder (ILR) was referred for CNA. Intervals at baseline: PR 208 ms, AH 137 ms, HV 50 ms. Bi-atrial mapping was followed by RFA at the anatomic sites of the Marshall Tract, left superior, right superior, and Aortic-SVC GPs. Vagal responses (sinus arrest and AV block) were seen during ablation of the left-sided GPs while his sinus rate increase observed during ablation of the right sided GPs. The posteromedial GP and right inferior GP were not ablated. Follow up ILR recordings showed reduced heart rate variability and increased sinus rates. Five months post-ablation another syncopal event occurred. ILR revealed 11 seconds of ventricular asystole due to complete AV block at unchanged sinus rate. Repeat CNA with RFA of the right inferior and posteromedial GPs resulted in decrease in PR from 213 to 176ms, AVN Wenckebach cycle length from 500 ms to 450 ms, AVN effective refractory period from 600/450 ms to 600/370 ms, and progressive decrease in AH interval with decreasing pacing cycle length (700 ms: from 101-> 93 ms, 600 ms: 142-> 120ms, 550 ms: 163->129 ms, and 500 ms: 193-> 142 ms) with no further change after atropine challenge. The patient has had no further syncopal events.

**Conclusion:** CNA can reduce VVS burden. Isolated ablation of the GPs providing SN innervation does not prevent VVS caused by AV block due to the distinct GP innervation of the AV node. Patients with VVS due to a SN cardioinhibitory response should be considered for CNA of AVN-innervating GPs since vagal AVN response can be initially masked by SN arrest. Changes in AH interval response curves at different paced cycle lengths provide a clearer endpoint for AVN modification than comparing intervals during sinus rhythm before and after ablation.