Results: Ten patients (90% male, 47.6±21.4 years old, 70% non-ischemic cardiomyopathy) had exercise testing before and after BCSD (within 6±7 months pre-BCSD and 16±19 months post-BCSD). Baseline heart rate was 73±12 bpm before and 67±7 bpm after BCSD (p = 0.08). Change in heart rate from baseline during exercise was 58±26 bpm pre-BCSD, compared to 45±21 bpm post-BCSD (p = 0.1). Exercise duration was similar (9±3 min before and 8±3 min after BCSD, p = 0.2) and patients achieved an average of 8.8±3.2 METS pre- vs. 9.3±2.6 METS post-BCSD (p = 0.5). In 43 patients with echocardiographic data within one-year pre- and post-BCSD, there was no significant change in LVEF (36±13% and 35±14% respectively, p = 0.6) or RVSP (28±8 mmHg and 31±9 mmHg respectively, p = 0.12). The number of patients on guideline-directed medical therapy was similar before and after BCSD. Additionally, 83% of patients were on beta-blockers both before and after BCSD.

Conclusion: Despite decreased sympathetic input to the heart, cardiomyopathy patients after BCSD still have preserved exercise tolerance and cardiac function months after cardiac sympathetic denervation.

PO-637-08
A COMPARISON BETWEEN DECELERATION ZONES ON ISOCRONAL LATE ACTIVATION MAPPING AND SUBSTRATE FOR VENTRICULAR TACHYCARDIA USING CT LATE IODINATED CONTRAST ENHANCEMENT AND WALL THINNING

Daniel Levin; Aaron Matthews; Joshua Payne MD, MPH and Jeffrey R. Winterfield MD, FHRS

Background: Isochronal late activation mapping (ILAM) is an important tool for identification of targets for ablation without requiring induction of ventricular tachycardia. Computed tomography is also a promising a non-invasive method of identification of scar. Late iodinated contrast enhancement and wall thinning with acquisition of sub-1mm slice thickness are used, which are surrogates for ablation targets. The relationship of deceleration zones observed on ILAM with substrate observed on CT imaging is unknown.

Objective: To assess the correlation between deceleration zones seen on ILAM with CT substrate.

Methods: Consecutive patients who underwent VT ablation during a 2-year period using the NAVX (St. Jude Medical, Minneapolis, MN) electroanatomic mapping system and underwent CT imaging (InHeart, Bordeaux, France) were retrospectively reviewed. Patients with incomplete maps, low density maps, or incomplete imaging were excluded (n = 15). After CT image integration into the EA map, the presence of DZs was assessed and the presence of underlying substrate on imaging defined. All maps and images following integration were evaluated by 3 independent reviewers who were blinded to ablation lesion sets and clinical outcomes. The DZ was identified and demarcated and then the superimposed substrate on imaging was evaluated.

Results: There were 20 patients in the final analysis. The cohort was 80% male with mean age of 68 years with a left ventricular ejection fraction of 34%. Ischemic heart disease was present in 55% of subjects. In 17 of the 20 patients (85%) wall thinning of 1-3mm superimposed on the DZ sites was observed. Dense scar was present in only 7 patients (35%) at DZ sites.

Conclusion: DZs observed on ILAM area are highly associated with areas of wall thinning from 1-3 mm on CT. Dense scar was less frequently noted with DZs. These findings may serve as a basis for imaging guided VT ablation in future studies.

Figure 1: An isochronal late activation map of the left ventricle in a left lateral view. An image of the iodinated CT has been overlaid with purple and yellow areas representing wall thinning.

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Friday, April 29, 2022
3:00 PM - 5:00 PM

PO-638-01
THE FUTURE OF ACLS: IVABRADINE’S ROLE FOR THE TREATMENT OF REFRACTORY VENTRICULAR ARRHYTHMIAS

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Background: Ivabradine (IVA) is an attractive antiarrhythmic for the treatment of refractory ventricular arrhythmias (RVA) as it does not affect blood pressure or prolong the QT interval. IVA’s efficacy in ventricular tachycardia (VT)/ventricular fibrillation (VF) has been validated in multiple animal studies. We present a case series demonstrating complete resolution of RVA in humans with the addition of IVA.

Objective: Illustrate the outcomes of RVA with the treatment of IVA.

Methods: Retrospective case series analysis of 4 patients treated with IVA for RVA.

Results: Pt 1: 63M with ischemic cardiomyopathy was admitted for RVA despite amiodarone, quinidine, and a beta blocker. IVA 2.5mg BID was initiated with resolution of VF. Pt 2: 61F was admitted with cardiogenic shock secondary to giant cell myocarditis complicated with VT/VF. IVA 2.5mg BID was initiated for RVA despite amiodarone and quinidine. IVA was up titrated to 5mg BID with resolution of VT/VF. Pt 3: 58M without significant history was admitted with a VF arrest. The addition of IVA 2.5mg BID to amiodarone, quinidine, and procainamide resolved VF burden. Pt 4: 37M with cardiomyopathy with continued VT/VF despite amiodarone, and lidocaine, and Impella mechanical circulatory support. IVA 2.5mg BID was initiated, and lidocaine was weaned. These therapies stabilized his rhythm, allowing for successful left ventricular assist device implant. All four patients’ RVA (Images A, C, E, G) were converted to sinus rhythm (Images B, D, F, H) with the addition of IVA to standard antiarrhythmic drugs (Table 1/Figure 1).

Conclusion: IVA may be a beneficial additive treatment for RVA. However, randomized studies are required to determine the clinical success of RVA attenuation with IVA.
PO-638-02
INFLAMMATION BY FDG PET IS DISCORDANT WITH SITES OF FOCAL VENTRICULAR TACHYCARDIA IN PATIENTS WITH SUSPECTED CARDIAC SARCOIDOSIS
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Background: In pts with cardiac sarcoidosis (CS), late gadolinium enhancement by cardiac MRI predicts development of ventricular arrhythmias. Our previous analysis also suggested a relation between scar regions and focal ventricular arrhythmias (FVA) as determined by FDG PET CT. The role of active inflammation on FDG PET in focal ventricular arrhythmias (FVA) site of origin is unclear.

Objective: To assess relationship between FDG PET findings consistent with active inflammation and FVA in pts with suspected CS who underwent catheter ablation.

Methods: From 2010 to 2020, consecutive pts with suspected CS who had PET scans and VT catheter ablation were included. VT was classified as focal or reentrant based on procedural findings. Pts with indeterminate VT mechanism were excluded (n=4). FDG PET was independently analyzed (scan was within 17+/-25 days of ablation) and classified as to the location and presence of inflammation (e.g. focal FDG uptake). Diffuse and lateral wall FDG uptake were considered non-specific and excluded in describing the location of the FDG uptake.

Results: Twenty-seven (20%) of 135 pts (63±12.2 years, 19.4% female) were found to have FVA: aortic root in 8, aortomitral continuity in 6, papillary muscle in 3, crux in 3, RV lateral wall in 2, and LV anteroseptal wall in 1. Six (22.2%) of 27 pts had focal FDG uptake on the PET scan (OR 0.48 0.19-1.2, p=0.122, NS). One (3.7%) of the automatic VTs was qualitatively from a similar region to that of FDG uptake.

Conclusion: FVA in pts with suspected CS does not correlate well anatomically with specific areas of active inflammation on PET scan and occur in the absence of measurable FDG uptake.

PO-638-03
ISOLATED PREMATURE VENTRICULAR CONTRACTIONS ARE MORE DYSSYNCHRONOUS THAN PREMATURE VENTRICULAR CONTRACTIONS IN REPEATED PATTERNS
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Background: Premature ventricular contractions (PVCs) may lead to cardiomyopathy. Preclinical studies have demonstrated that more dyssynchronous PVCs lead to a greater degree of cardiomyopathy. However, dyssynchrony of isolated PVCs compared to repeated patterns of bigeminy and trigeminy have never been investigated.

Objective: To compare left ventricular (LV) myocardial function and LV dyssynchrony in patients with isolated PVCs compared to patients with PVCs in repeated patterns.

Methods: We prospectively included 85 consecutive patients referred for PVC ablation due to frequent PVCs. Isolated, bigeminal and trigeminal (PVC every third heart beat) PVCs were defined based on the dominant pattern. LV global longitudinal strain (GLS) and LV dyssynchrony (measured as SD of time to peak GLS) were quantified by 2D strain echocardiography.

Results: Of the 85 included patients (age 57±6 yrs, 46% female, LVEF 55% [45-60]), 58 had isolated PVCs, 13 had bigeminal PVCs and 14 had trigeminal PVCs, with no difference in PVC burden (20% [15-27] vs. 21% [15-25] vs. 22% [21-25]; p=0.7) or PVC QRS width (155 ms [146-168] vs. 161 ms [146-183] vs. 159 ms [136-174]; p=0.4) among the groups. PVC GLS was comparable (-8±6% vs. -7±5% vs. -9±3%; p=0.4, Figure 2A). However, LV dyssynchrony was significantly greater for isolated PVCs compared to bigeminal or trigeminal PVCs (113 ms [90-137] vs. 86 ms [69-99] vs. 58 ms [58-105]; p=0.001, Figure 2B).

Conclusion: In patients with frequent PVCs, isolated PVCs were more dyssynchronous than PVCs in bigeminal and trigeminal patterns, despite similar PVC QRS duration. Our findings indicate that patients with isolated PVCs may be at higher risk of developing cardiomyopathy than patients with PVCs in repeated patterns.