included posterior wall isolation (93.2%), lateral mitral isthmus ablation (43.4%), and SVC isolation (9.7%). The PVI+ group had larger left atrial volumes and more likely to have a diagnosis of CHF, but similar characteristics otherwise (Table). There was no difference in primary outcome between PVI and PVI+ (86.9% vs 87.1%, respectively, p = 0.96).

**Conclusion:** In patients with PsAF that exhibit normal LA bipolar voltage on EAM, additional LA ablation beyond PVI did not improve freedom from atrial arrhythmias at 12 months. Operators were more likely to perform additional LA ablation in patients with CHF and larger LA volume. Larger, randomized studies are needed to investigate further.

<table>
<thead>
<tr>
<th>Table</th>
<th>PVI (n = 160)</th>
<th>PVI+ (n = 70)</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age</td>
<td>64.9 ± 9.2</td>
<td>64.5 ± 9.2</td>
<td>0.78</td>
</tr>
<tr>
<td>CHADS2-VAS score</td>
<td>2.29 ± 1.32</td>
<td>2.48 ± 1.27</td>
<td>0.32</td>
</tr>
<tr>
<td>LVEF</td>
<td>57.3 ± 14.9</td>
<td>49.6 ± 13.6</td>
<td>0.23</td>
</tr>
<tr>
<td>BMI</td>
<td>31.7 ± 7.1</td>
<td>32.5 ± 6.0</td>
<td>0.44</td>
</tr>
<tr>
<td>LA volume [cc]</td>
<td>99.4 ± 24.1</td>
<td>125.5 ± 33.8</td>
<td>0.0001</td>
</tr>
<tr>
<td>Female sex, n (%)</td>
<td>26 (16.3)</td>
<td>14 (20)</td>
<td>0.49</td>
</tr>
<tr>
<td>Hypertension, n (%)</td>
<td>127 (79.3)</td>
<td>60 (85.7)</td>
<td>0.26</td>
</tr>
<tr>
<td>Diabetes Mellitus, n (%)</td>
<td>42 (26.3)</td>
<td>15 (21.4)</td>
<td>0.44</td>
</tr>
<tr>
<td>CHF, n (%)</td>
<td>20 (12.5)</td>
<td>18 (25.7)</td>
<td>0.01</td>
</tr>
<tr>
<td>CVA, n (%)</td>
<td>10 (5.9)</td>
<td>3 (4.3)</td>
<td>0.55</td>
</tr>
</tbody>
</table>

**EPICARDIAL SUBSTRATE MAPPING FROM CORONARY VESSELS WITH OVER-THE-WIRE MICRO CATHETER FOR SCAR-RELATED VENTRICULAR TACHYCARDIA**

Takuro Nishimura MD, PhD; Masahiko Goya MD; Masateru Takigawa MD; Yuki Shimizu; Miki Amemiya; Takashi Ikemouchi MD; Tatsuaki Kamata; Tasuku Yamamoto; Susumu Tao MD, PhD; Shinsuke Miyazaki MD, PhD, FHRs and Tetsuo Sasano MD

**Background:** Epicardial access is often necessary to eliminate the reentrant circuit of ventricular tachycardia (VT). However, epicardial puncture accompanies the risk of complication. And in patients after cardiac surgery, adhesions make mapping challenging and require a surgical approach to epicardial mapping and ablation. In those cases, a noninvasive technique is desired to detect if epicardial substrate exists prior to the surgical approach.

**Objective:** To investigate the feasibility and safety of epicardial mapping from coronary vessels with 2.7 Fr over-the-wire multielectrode microcatheter.

**Methods:** Seven scar-related VT patients (all male, nonischemic: n = 5, prior cardiac surgery: n = 3) underwent endocardial high-density isochronal late activation mapping (ILAM), first. Then, the microcatheter was inserted into a branch of coronary vessels at the opposite of endocardial deceleration zone (DZ) or good pace map site.

**Results:** In all cases, the microcatheter was successfully inserted into the target branch of the vessel (coronary artery: n = 1, coronary venous system: n = 6). Epicardial abnormal potentials (late and/or fragmented potentials) were recorded in 3 patients, but one of them did not have a deceleration zone in the endocardium. During VT, the epicardial diastolic potential was recorded in 2 patients. One patient who had prior cardiac surgery underwent successful surgical epicardial ablation, and the other was bailed out by endocardial intensive ablation. There was no complication in all patients.

**Conclusion:** Epicardial mapping from a branch of coronary vessels is safe and has the potential to detect epicardial substrate noninvasively. This technique can be the first step to detect epicardial substrates, especially in patients after cardiac surgery.

**EFFECT OF BILATERAL CARDIAC SYMPATHETIC DENERVATION ON EXERCISE TOLERANCE AND CARDIAC FUNCTION**

Amber Tang; Aadhavi Sridharan MD, PhD; Julie M. Sorg MSN; Jane Yanagawa MD; Jason Bradfield MD, FHRS; Kalyanam Shivkumar MD, PhD, FHRS and Marmar Vaseghi MD, MS, PhD, FHRS

**Background:** Bilateral cardiac sympathetic denervation (BCSD) decreases sympathetic outflow to the heart and is used as a treatment strategy in patients with refractory ventricular arrhythmias. However, there has been concern regarding removal of bilateral sympathetic chains with regards to exercise endurance and cardiac inotropy, and hence, a left sided only procedure has been advocated.

**Objective:** To determine the impact of BCSD on measures of exercise capacity, left ventricular ejection fraction (LVEF), and right ventricular systolic pressure (RVSP).

**Methods:** Retrospective review of 101 patients who had undergone BCSD between 2010 and 2021 was performed. Patients who had undergone exercise testing, either with a treadmill test or cardiopulmonary exercise test, and an echocardiogram within one-year pre- and post-BCSD were included. Changes in heart rate from baseline, metabolic equivalents (METS), duration of exercise, LVEF and RVSP pre- and post-BCSD were analyzed.
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 non-invasive method of identification of scar. Late iodinated contrast enhancement and wall thinning with acquisition of sub-1mm slice thickness are required for identification of scar. Late iodinated contrast enhancement and wall thinning are used, which are surrogates for ablation targets. The relationship of deceleration zones observed on ILAM with substrate observed in 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 the superimposed substrate on imaging was evaluated.

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 ISOCHRONAL 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

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 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.

POSTER PO-638:

Posters: Clinical EP at Pod 10

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

Kristen Brown MD; Lindsey Safley RPh; Kara Stout DO, MPH; John W. Schleifer MD and Faris Khan MD, MS, FHRS

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, O) 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.