surgical aortic valve replacement (SAVR); it also illustrates novel use of His bundle pacing in this setting.

**Methods:** N/A

**Results:** A 70-year-old man with dyspnea was found to have severe aortic insufficiency with prolapse of the right coronary cusp and no angiographic coronary artery disease (CAD). He underwent SAVR with a #27 Edwards Inspiris bovine valve. On post-op day one, he became hypotensive and was noted to be in sinus arrest with a junctional escape and inferior ST elevations requiring emergent transvenous pacing (Figure 1). He was subsequently taken for dual chamber PPM implantation, where fluoroscopy and a lack of atrial sensing or capture confirmed atrial standstill. A nonselective His bundle lead was implanted, with a capture threshold of 2.5V@1.0ms. The patient remained hypotensive despite adequate pacing. A transthoracic echocardiogram revealed a newly depressed ejection fraction of 40%, a mildly dilated and moderately hypokinetic RV, and otherwise normal valve function. CTA of the pulmonary arteries showed no evidence of pulmonary embolus. A Swan-Ganz catheter found a RAP of 18mmHg, a PCWP of 21mmHg, a CI of 1.78 L/min/m², an SVR of 1500 (dyn*s/cm⁵), and a mixed venous oxygen saturation of 40%. He was started on inotropes and vasopressors. While EKG showed a paced rhythm that did not meet sgarbossa criteria, a high sensitivity cTnI >50,000 ng/L and inferior ST elevations on initial ECG supported the decision to pursue left heart catheterization (LHC) despite recent catheterization having ruled out CAD. LHC revealed a severe, diffuse spasm of the entire right coronary system which improved with intra-coronary nitroglycerin. The cardiogenic shock subsequently resolved, and the patient was discharged home.

**Conclusion:** Atrial standstill is an unknown complication of coronary vasospasm. His bundle pacing was successful in this setting.

---

**40 YEAR OUTCOME OF THE CORRIDOR SURGERY FOR PAROXYSMAL ATRIAL FIBRILLATION**

Melanie Roberta Zammit Burg; Abdullah Al-Shaheen; Robert D. Anderson; Hanney Gonna; Praloy Chakraborty; Sameer Kushwaha; Eugene Downar and Vijay S. Chauhan

**Background:** The corridor procedure was a surgical therapy for paroxysmal atrial fibrillation (PAF) in the 1980s. It isolated the fibrillating atria from a narrow corridor of right atrial tissue connecting the sinus and atrioventricular nodes. This approach was eventually superseded by MAZE surgery and catheter ablation.

**Objective:** Long term outcomes of the now historical corridor are rarely described. We present a case of complex atrial arrhythmias 40 years post corridor.

**Methods:** N/A

**Results:** A 57 year old lady presented with atrial flutter and regular ventricular activation. She had undergone corridor surgery for atrial tachycardia 40 years ago. Electroanatomic mapping in the right (RA) and left atrium (LA) demonstrated 3 simultaneous arrhythmias: (1) a regular atrial flutter (CL 290ms) in the RA outside the corridor, (2) AF in the LA outside the corridor and along the septum within the corridor, and (3) sinus rhythm within the corridor which was dissociated from the AF/flutter and dissociated from ventricular activation. There was extensive low-voltage (<0.5mV) in the high corridor in the region of the sinus node. The atrial flutter demonstrated focal, earliest activation along the low posterolateral RA. Catheter ablation here only transiently disorganized it, suggesting passive activation. Pulmonary vein isolation (PVI) was also performed with the end point of entrance block. She was electrically cardioverted to a low atrial rhythm with 1:1 AV conduction. At 3 months followup, she has had no arrhythmia recurrence.

**Conclusion:** Our patient presented with complex atrial activation 40 years post corridor surgery. The dominant arrhythmia appeared to be AF in the LA and corridor itself. A more focal atrial flutter in the RA conducted regularly to the ventricles. This implied conduction across much of the corridor, but with sufficient block to organize the AF to flutter in the RA. There was also extensive structural remodeling within the corridor resulting in dissociated sinus activity during AF/flutter and a low atrial escape rhythm following cardioversion. As such, our PVI may not provide durable rhythm control and more extensive substrate ablation along the corridor and atria may be required. Understanding historical surgical treatments for AF is essential to interpret arrhythmias that may present decades later.
POSTER PO-643:
Posters: Heart Failure at Pod 15
Friday, April 29, 2022
3:00 PM - 5:00 PM

PO-643-01
ELECTRICAL DYSSYNCHRONY MAPPING IN CARDIAC RESYNCHRONIZATION THERAPY
Alan J. Bank MD; Christopher Brown; Kevin Burns PhD and Emanuel Espinoza

Background: There is no clinical method for measuring electrical dyssynchrony over a wide range of atrial-ventricular delays (AVD) and ventricular-ventricular delays (VVD) in cardiac resynchronization therapy (CRT) patients.

Objective: To describe a new methodology, based on wavefront fusion, for mapping electrical synchrony in CRT.

Methods: The area between combinations of 9 anterior/9 posterior electrograms (area under curve; AUC) was quantified and cardiac resynchronization index (CRI) was defined as % change in AUC compared to native AUC. CRI was measured in 20 ms steps over a wide range of atrial-RV pace (A-RVp) and atrial-LV pace (A-LVp) intervals in 90 patients 3.4±3.7 years post-CRT to generate electrical dyssynchrony maps (EDM). An optimal synchrony line (OSL) depicted the combination of AVD/VVD producing the highest CRIs.

Results: CRI at baseline programming was 54±39%. Patients with complete heart block (n=20) had an OSL parallel to the VV = 0 line with leftward shift showing LV preactivation (VVD) needed for optimal resynchronization. Patients with intact AV node conduction (n=64) had an OSL parallel to the VV = 0 line at short AVD (fusion of RVp and LVP wavefronts), upward curving at intermediate AVD (triple fusion), and vertical at long AVD (fusion of native and LVP wavefronts) except for the patients with poor LV lead position (n=6). On average, the LVP wavefront was 38±26 ms behind the RVp wavefront and 87±24 ms behind the native wavefront.

Conclusion: We describe a new methodology of EDM for quantifying and graphing electrical synchrony over a wide physiologic range of AVDs/VVDs. This methodology offers a noninvasive, practical, clinical approach for mapping electrical dysynchrony and optimizing CRT programming.

PO-643-02
CARDIAC RESYNCHRONIZATION THERAPY OPTIMIZATION IN NON-RESPONDERS USING ELECTRICAL DYSSYNCHRONY MAPPING
Alan J. Bank MD; Christopher Brown; Emanuel Espinoza and Matthew D. Olson MD

Background: Cardiac resynchronization therapy (CRT) non-response occurs in ~30% of patients. There are no well-accepted methods for optimizing CRT in non-responders.

Objective: To assess effects of CRT optimization using electrical dyssynchrony mapping (EDM) on left ventricular (LV) function in CRT non-responders.

Methods: We studied 33 patients with underlying LBBB/IVCD who had an EF <45%, 3.5+/−3.4 years after CRT. The area under the curves (AUC) between 9 anterior/9 posterior electrograms was measured at multiple combinations of atrial-ventricular and ventricular-ventricular delays (AVD, VVD). Electrical dyssynchrony was quantified by cardiac resynchronization index (CRI), calculated as % change in AUC compared to native. EDM depicted CRI over the wide range of settings tested. Patients were programmed to highest CRI setting with echos read blinded pre-optimization and ~6 months post-optimization.

Results: EDM (Figure) of nonresponder with LBBB, programmed simultaneous biventricular pacing at AVD 120 ms (CRI 47%) and optimized to LV-only pacing at AVD 110 ms (CRI 95%). EF improved from 21% to 28%. Table shows significant improvements in systolic function, LVESV, global and regional strain and interventricular mechanical dyssynchrony (AVO - PVO) in the study cohort.

Conclusion: CRT non-responders are 51% electrically resynchronized at baseline and improve to 90% with CRI-guided optimization. CRT optimization using a novel EDM technology results in significant improvements in LVEF, LVESV, LV global strain in multiple planes, anteroseptal/septal regional strain, and interventricular mechanical dyssynchrony. This methodology offers a non-invasive, practical clinical approach to treating CRT nonresponders.