at the onset of the freeze. After thawing, a second freeze was administered. No further PJCs were noted at baseline or with isoproterenol infusion.

**Conclusion:** JET could originate from anywhere within the AV node or proximal His bundle. The application of cryoablation at a typical AV nodal slow pathway location with a preceding pre-potential and immediate obliteration of PJCs suggests that the origin in this case was from this region rather than a true His bundle extrasystole. Identification of pre-potentials to the junctional ectopy can guide safe ablation of this dysrhythmia.

**PC-578-03**

**RIGHT ATRIAL ATYPICAL FLUTTER WITH 2:1 INTRA-ATRIAL BLOCK MIMICKING LEFT ATRIAL FOCAL AND LOCALISED REENTRANT TACHYCARDIA**

David T. Boothe MD; Steven Song MD; Steven Leung MD, MHA; Andrew A. Zadeh MD; Ivan C. Ho MD, FHRS and Junaid A.B. Zaman MA, MD, PhD, CCDS

**Background:** Atrial-level blocks are known confounders for diagnosis of SVTs and commonly occur after previous linear ablation lesions.

**Objective:** 1. To demonstrate importance of recognition of atrial-level block in SVT diagnosis and treatment. 2. To review underlying assumptions in using entrainment to localize SVT origins. 3. To discuss the importance of thorough biatrial mapping in determination of unclear SVT mechanisms.

**Methods:** N/A

**Results:** A 74y M with a history of bioprosthetic MV replacement, atypical RA flutter ablation (2018), and permanent pacemaker (2020) for sick sinus syndrome presented with fatigue and 76% AF/AT burden. For index RA ablation, both an intercaval line using bridging scar near the atriotomy site anterior to the appendage and a cavotricuspid isthmus line were made. Tachycardia was induced via burst pacing. Initial entrainment from CS suggested a L-sided tachycardia. A Pentaray map of the tachycardia showed LA anteroseptal reentry with entrainment at the site giving a PPI-TCL, 30ms. PVI and anteroseptal line were performed which changed cycle length but not CS activation. LA re-mapping demonstrated focal breakout near RIPV; ablation lengthened cycle length to 290ms without rhythm cessation. After PVI confirmation, mapping of the RA showed 2:1 intra-atrial conduction in the lateral RA with 1:1 conduction in the septal RA (fig A). Mapping 1:1 areas of RA conduction showed a gap in previous IVC line with intact CTI block with the majority of the cycle length at the posteroseptal floor (fig B), immediately opposite the site of initial LA anteroseptal reentry (fig C). During ablation of IVC line gap, progressive lengthening of the TCL was noted followed by termination of rhythm without inducibility (fig D).

**Conclusion:** Response to entrainment assumes 1:1 conduction through the entire circuit, and contributed to the appearance of a left sided tachycardia upon initial CS entrainment. The finding of a LA RIPV tachycardia upon remapping the LA after PVI is consistent with a separate focal tachycardia but could represent epicardial connections from the CS to the posterior LA.

**PC-578-04**

**TRANSHEPATIC PERMANENT PACEMAKER LEAD PLACEMENT**

Kamal Preet Cheema; Dan Sorajja MD, FHRS and Sailendra Naidu

**Background:** Limited or compromised systemic venous access poses a significant challenge in patients who require a permanent cardiac pacemaker. Case reports of transhepatic access have been described in the congenital population.

**Objective:** We describe a case of symptomatic sinus node dysfunction in which the standard transvenous approach was not possible.

**Methods:** N/A

**Results:** A 44-year-old woman with fibrosing mediastinitis and occlusion of the superior vena cava developed symptomatic sinus node dysfunction and left bundle branch block. Given her anatomy the decision was made to proceed with surgical pacemaker implantation via a right thoracotomy approach, as she did not want leads placed via the femoral route. The right atrial (RA) and right ventricular leads were implanted into their respective chambers through purse-string sutures in the right atrium, with the device placed in right axilla. At the 6-week post-implant device interrogation, the RA lead impedance had increased from 693 ohms to 1045 ohms and the lead was detecting both atrial and ventricular signals. Chest x-ray confirmed RA lead dislodgement (Figure 1). She was brought to the EP lab for attempted RA lead revision, which ultimately was unsuccessful. The RA lead was then revised using a transhepatic approach. Percutaneous right hepatic vein access was performed by interventional radiology using ultrasound and fluoroscopic guidance. Through a 7-French sheath, the RA lead was positioned in the right atrial appendage, and slack was given to this lead to accommodate respiratory motion (Figure 2). The lead was secured at the access site with the suture sleeve, and...