PO-642-05
TENSION HEMOTHORAX DUE TO APICAL LEAD PERFORATION TWO YEARS AFTER IMPLANTATION
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Background: Late myocardial perforation secondary to a pacemaker lead is a rare, but potentially life-threatening complication.

Objective: Report a case of tension hemothorax secondary to lead perforation more than two years after implantation.

Methods: N/A

Results: A 76-year-old male with a prior surgical aortic valve replacement, mitral clip and a pacemaker, placed over two years ago, presented with acute onset left sided stabbing chest pain. Vitals were stable with mild volume overload on exam. The admission EKG showed atrial tachycardia with controlled ventricular response. The chest x-ray and echocardiogram were unrevealing. Two days later, he became hypoxemic. EKG showed evidence of ventricular under sensing and intermittent failure of ventricular capture. Interrogation of the device confirmed ventricular lead malfunction with failure to capture at high output. Repeat chest x-ray showed a new left pleural effusion and the chest CT showed an expanding hemothorax with lead perforation into the left pleural space. A few hours later, the patient became hemodynamic unstable secondary to tension hemothorax. In surgery, the right ventricular lead was found to penetrate through the septum and apex into the left hemithorax (Figure). Three liters of blood were evacuated from pleural space and the ventricular apex and septum were repaired. The existing leads were extracted with laser assistance and a new ventricular and atrial lead were placed. Postoperative device interrogation showed 100% ventricular pacing and normally functioning leads. There were no postoperative complications and the patient was discharged on fifth postoperative day.

Conclusion: We report a case of a very delayed ventricular perforation and lead migration post pacemaker implantation.

PO-642-06
A PULMONARY VEIN ISOLATION WITH WIDE AREA CIRCUMFERENTIAL ABLATION AND BILATERAL CARINAL LINE ABLATION VIA TRANSEPTAL APPROACH
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PO-642-07
ATRIAL STANDSTILL: A RARE COMPLICATION OF CORONARY VASOSPASM FOLLOWING SURGICAL AORTIC VALVE REPLACEMENT
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Background: Atrial standstill is a phenomenon characterized by the absence of both electrical and mechanical atrial activity, resulting in complete lack of excitability. Identified causes include drugs, electrolyte abnormalities, hypoxia, and myocardial infarction.

Objective: This case reveals atrial standstill as a newly described complication of coronary artery vasospasm after surgical aortic valve replacement.

Methods: N/A

Results: A 64-year-old woman with persistent AF for 8 years presented to our institution after unsuccessful rhythm control elsewhere with an inability to pass the catheter into the right atrium from the inferior vena cava. A CT scan confirmed congenital absence of the inferior vena cava with dilated azygous veins (Fig 1A). A transhepatic approach for AF ablation was offered. The hepatic vein was cannulated with contrast under ultrasound and fluoroscopic guidance, passing a wire to the superior vena cava. A significantly reshaped Baylis transseptal needle was used through a SL1 long sheath for transeptal puncture (Fig 1B) guided by intracardiac echocardiogram from internal jugular vein access (Fig 1C). The coronary sinus catheter was later placed through this access. The HD Grid catheter was used for 3D mapping and later exchanged for a 4 mm Tacticath bidirectional irrigated tip catheter, which was used for the WACA and carinal ablation. Entrance and exit block for each pulmonary vein was demonstrated. A 10-mm Amplatzer plug was placed within the transhepatic access site tract using ultrasound and fluoroscopic guidance (Fig 1D).

Conclusion: The hepatic veins are large enough to accommodate the sheaths and catheters meant for a femoral venous approach. In selected patients, the transhepatic approach can be considered for ablation of AF.
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.

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

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