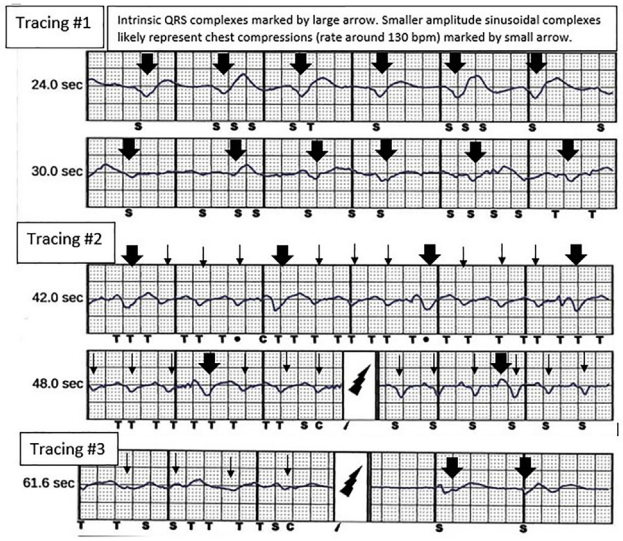
pacing wire was inserted. Ultimately the patient experienced pulseless electrical activity (PEA) arrest and CPR was initiated. SICD tracings show oversensing of widened QRS just prior to PEA. The clinical rhythm remained PEA throughout resuscitation but the SICD delivered multiple inappropriate shocks. Two shocks were felt by a nurse performing CPR who reported sensation of electric jolt. CPR resumed with a towel over the chest to protect rescuers. SICD interrogation revealed 15 tachy episodes, 11 of which were treated with SICD shock. Tracing #1: Pre arrest showing double counting of wide R waves. Tracing #2: Wide intrinsic rhythm with superimposed low amplitude sinusoidal activity at rate of ~130 bpm representing rapid performance of chest compressions. Oversensing is seen and inappropriate shock delivered. Tracing #3: Low amplitude waveform ceases when compressions are interrupted post shock.

Conclusion: CPR may result in inappropriate shocks from SICD due to oversensing artifact. Rescuers performing CPR should be educated in this regard. CPR can be performed from the right side so rescuer is not directly in the shock vector and may be exposed to less current if SICD fires.



B-PO02-012

USE OF STORED DEFIBRILLATOR ELECTROGRAMS AND EXTRA STIMULUS MAPPING FOR IDENTIFICATION OF VENTRICULAR ECTOPY TRIGGERING VENTRICULAR FIBRILLATION

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Background: Ablation using activation or pace mapping of PVCs from Purkinje tissue triggering VF; is associated with high success rates. However, sometimes PVCs are not observed during ablation.
Objective: NA
Methods: NA
Results: A 40 y/o female with a diagnosis of Idiopathic Ventricular Fibrillation, s/p ICD and bilateral sympathectomy presented with VF storm. Frequent short-coupled PVCs (LBB, left superior axis) localized to the RV papillary muscle (PM) as well as episodes of PVC induced VF (Fig 1A) were noted. The patient was stabilized with lidocaine and esmolol infusion and by pacing at a higher rate at 100 bpm. In the EP lab, despite administration of isoproterenol and calcium, clinical PVCs could not be induced. Pace mapping comparing the stored shock vector ICD EGM and surface ECG near the anterolateral and posteromedial PM of the RV and the moderator band was not good. However, extra stimulus pace mapping at an interval similar to the coupling interval of the PVC in these areas had a good match with the shock vector ICD EGM and the surface 12 lead ECG (Fig 1B) obtained during the procedure. Purkinje potentials in these sites were also targeted for radiofrequency ablation (Fig 1C). Ablation lesions were further consolidated. There was no recurrence of VF post ablation and during short-term follow up.
Conclusion: Successful ablation of short coupled PVC inducing VF can be obtained with extra stimulus pace mapping in areas known to have different exit sites due to preferential conduction like outflow tract, septum and PM. In the absence of clinical PVCs, careful analysis of stored ICD EGMs can be useful.

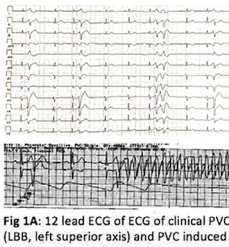
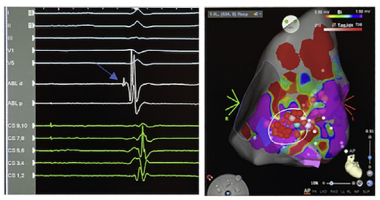


Fig 1A: 12 lead ECG of ECG of clinical PVC (LBB, left superior axis) and PVC induced VF.

Fig 1C. Presence of purkinje potential (arrow) at the site of ablation. Electro-anatomical map of the RV showing ablation lesions in between the medial and lateral pap muscles.



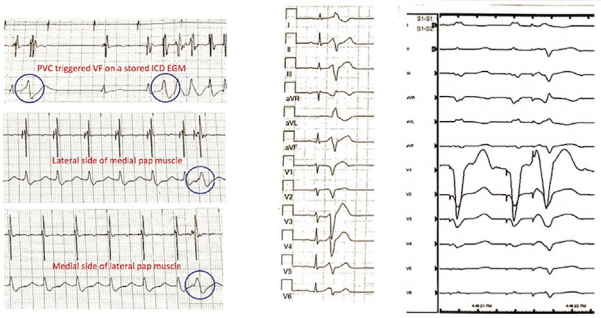


Fig 1B: Comparison of extra-stimulus pace mapping using stored EGM and 12 lead with the clinical PVC.

B-PO02-013

USE OF INTRACARDIAC ECHOCARDIOGRAPHY (ICE) AS AN ADJUNCT FOR SLOW PATHWAY ABLATION

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Background: Effective location of the slow pathway during ablation of atrioventricular nodal reentrant tachycardia (AVNRT) is crucial for ablation success.
Objective: We describe our experience with the use of intracardiac echocardiography (ICE), in conjunction with electroanatomic mapping (CARTO-Sound), to accurately demarcate critical structures in the apex of the triangle of Koch and to assist in identifying the location of the slow pathway in six patients with symptomatic AVNRT.
Methods: Six patients with symptomatic supraventricular tachycardia (SVT) underwent elective ablation for their arrhythmia. AVNRT was confirmed as the mechanism of SVT with a comprehensive electrophysiology study. ICE was used to