ABSTRACT PE-568:
Pediatric Electrophysiology
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PE-568-01
A CLINICAL ALGORITHM TO DETERMINE RISK OF RECURRENCE IN INFANTS WITH SUPRAVENTRICULAR TACHYCARDIA: A COHORT STUDY OF 460 INFANTS
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Background: Data regarding risk factors for supraventricular tachycardia (SVT) recurrence in infants has varied in prior studies.

Objective: To evaluate a large cohort of infants diagnosed with SVT and create a clinically useful risk algorithm for physicians to identify recurrence risk at the time of initial diagnosis.

Methods: A retrospective single-center study (1984-2020) with prospective phone follow-up of infants with structurally normal hearts diagnosed with re-entrant SVT at ≤1 year of age. Infants with atrial flutter or permanent junctional reciprocating tachycardia were excluded. The primary outcome was SVT recurrence after initial hospital discharge. Risk factors were evaluated using time-dependent survival analysis. Classification and regression tree (CART) analysis was then performed to determine a risk algorithm.

Results: Among 460 infants (62% male), 33% had SVT recurrence. Median cardiac follow-up was 5.2y (IQR 1.8-11.2). On multivariable analysis, factors associated with recurrence included: fetal or postnatal diagnosis >60 days (HR 1.90, 95% CI 1.26-2.86 and 1.73, 95% CI 1.07-2.77, respectively), Wolff-Parkinson-Syndrome (WPW) at diagnosis (HR 2.46, 95% CI 1.26-2.86 and 1.73, 95% CI 1.07-2.77, respectively), and need for multiple antiarrhythmic or second-line therapy defined as flecainide, sotalol, or amiodarone (HR 1.91, 95% CI 1.13-3.21). Risk further doubled if multi-antiarrhythmics or second-line agents were at highest risk. All other infants are at moderate risk for recurrence.

Conclusion: One-third of infants with SVT have a risk of recurrence. Recurrence risk can be stratified into low, moderate, and high-risk categories based on factors present at initial diagnosis and hospital discharge. Infants with no risk factors are at low risk. Infants with WPW or non-WPW with fetal or postnatal diagnosis >60 days and discharged on multi-antiarrhythmics or second-line agents are at highest risk. All other infants are at moderate risk for recurrence.

PE-568-02
IDENTIFICATION OF NOVEL THERAPEUTIC TARGETS FOR PACING INDUCED CARDIOMYOPATHY: A PILOT STUDY IN CHILDREN WITH CONGENITAL COMPLETE ATRIOVENTRICULAR BLOCK
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Background: Chronic ventricular pacing in children can lead to pacing-induced cardiomyopathy (PICM). It is difficult to predict who will develop PICM, and CRT may have limited effectiveness when used too late. Micro RNAs (miRs), small non-coding RNAs which regulate gene expression, are emerging as tools to evaluate the mechanisms of heart failure and may aid in early diagnosis of PICM.

Objective: To identify a circulating miR signature of adverse myocardial remodeling in paced patients preceding PICM to benefit follow disease progression.

Methods: Clinical data and blood were collected from children with normal cardiac structure and function with RV pacing for >5 yrs for congenital complete AV block (N=9) and non-paced controls (N=13). miR microarrays were performed and differentially expressed miRs (FC>2, corrected p<0.05) and target pathways were identified using GeneSpring and mirPATH.

Results: RV paced patients and controls were age matched (15.7±2.4 vs 15.0±2.0 yrs, p=NS). Average pacing duration was 12.31±3.2 yrs, ventricular pacing occurred ≥95% of the time. No difference in EF was seen between groups (62±0.06% vs 65±0.03%, p=NS). 468 miRs were differently regulated between the groups. 296 miRs were upregulated, predicting downregulation of FoxO: gluconeogenesis and glycogenolysis; adherens junctions (miR-548); and protein turnover and degradation (miR-205). 192 miRs were downregulated, predicting upregulation of fatty acid metabolism (miR-195, 15), neurotransmitter and estrogen signaling (miR-128, 148). miRs regulating proliferative TGF-B and pro-apoptotic Hippo pathways, were both up and downregulated.

Conclusion: We identified a unique, noninvasive miR signature in chronically paced children implicating heightened fatty acid metabolism to support increased energy demands of the paced heart, and protective estrogen signaling to promote nitric oxide metabolism and ion homeostasis. Profibrotic signaling was upregulated while cell-cell interaction was impaired. This miR pattern suggests early evidence of adverse myocardial remodeling despite normal EF on echo. Changes in circulating miRs may reflect an early signal of heart failure prior to the onset of ventricular dysfunction thus paving the way for the development of biomarkers predicting PICM.

PE-568-03
ASSOCIATION OF CENTER ABLATION VOLUME AND PROCEDURAL OUTCOMES IN CHILDREN: AN ANALYSIS OF THE IMPACT REGISTRY
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