Conclusion: In an ovine model of chronic AV block, true dual-chamber (DDD) leadless pacing was achieved with ~99% rate of beat-by-beat AV synchrony.

CI-525-04

A NEW CRITERIA TO DETERMINE LEFT BUNDLE BRANCH CAPTURE BASED ON TEMPORARY HIS BUNDLE OR RIGHT VENTRICULAR SEPTAL PACING

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Background: How to differentiate left bundle branch pacing (LBBP) from left ventricular septal pacing (LVSP) still remains challenging, especially in patients who do not present with generally-accepted criteria.

Objective: We aimed to develop a new criteria to confirm left bundle branch (LBB) capture in patients with both normal cardiac function or heart failure (HF), using temporary His bundle pacing (HBP) or right ventricular septal pacing (RVSP) as references.

Methods: Patients were included if surface ECGs of HBP or RVSP were recorded during procedure. dSti-LVAT1 was defined as the difference of Sti-LVAT between HBP and LBBP/LVSP. dSti-LVAT2 was defined as the difference of Sti-LVAT between RVSP and LBBP/LVSP.

Results: A total of 80 patients with normal cardiac function (65 LBBP and 15 LVSP) and 23 patients with HF were enrolled. Among 23 patients with HF who were corrected by LBBP, surface ECGs of LVSP were also recorded in 11 patients during procedure. Patients with LBBP showed significantly shorter Sti-LVAT than those with LVSP. The cut-off value of 12.5 ms for dSti-LVAT1 showed 73.9% sensitivity and 93.3% specificity to confirm LBB capture in patients with normal cardiac function. dSti-LVAT2 > 31.0 ms was also useful to determine LBB capture with a specificity of 93.3%. For patients with HF, the cut-off value of 9.0 ms for dSti-LVAT1 exhibited the greatest accuracy to confirm LBB capture with a sensitivity of 91.3% and a specificity of 100%.

Conclusion: Utilizing temporary HBP and RVSP as references during procedure could help determine LBB capture in patients with normal cardiac function or HF with great sensitivity and specificity.

ABSTRACT HF-566:
The Crossroads of Inherited Cardiomyopathies and Arrhythmia Syndromes

Saturday, April 30, 2022
9:15 AM - 10:15 AM

HF-566-01

INTERACTION OF ARRHYTHMIA AND CARDIOMYOPATHY WITH GENETIC VARIANT GROUPS IN THE UK BIOBANK POPULATION

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Background: Dilated cardiomyopathy (DCM) is a common cause of heart failure and rhythm disorders. It is estimated that a large proportion of DCM cases are familial and more than 250 genes of various functional groups have been suggested to be involved in the disease. However, there is a paucity of robust evidence regarding the majority of these genes’ role in the pathogenicity of DCM. Recently, an international group has curated a list of 51 DCM genes grouped by their level of evidence, graded as definitive, strong, moderate, limited, disputed and unknown.

Objective: We evaluated the risk for DCM, arrhythmia and cardiovascular outcomes in individuals with rare variants in each of the curated gene groups using whole exome sequences (WES), cardiac MRI and clinical data from 200,630 UK Biobank participants.

Methods: We annotated WES rare variants (MAF, 4 x 10^-5) in four categories: nonsynonymous, nonsense, “ClinVar” pathogenic and likely pathogenic (P/LP), and SIFT predicted deleterious variants. We collected ICD codes for DCM, ventricular arrhythmias (VT, VF), supraventricular (SV)