FACTORS IMPACTING FOLLOW-UP VENTRICULAR PACING PERCENTAGE (VP%) BURDEN AFTER TRANSCATHETER AORTIC VALVE IMPLANTATION (TAVI) FOR PATIENTS RECEIVING PROPHYLACTIC PERMANENT PACEMAKER (PPM) IMPLANT

Bradley M. Pitman; Natalie Montarello; Nicholas Montarello; Ross Roberts-Thomson; Glenn D. Young; Kurt C. Roberts-Thomson; Jerrett Lau; Joseph Montarello; Christopher X. Wong; Prashanthan Sanders and Dennis H. Lau

Background: Heart block is a known complication of transcatheter aortic valve implant (TAVI) due to implant proximity to conduction system (panel A), and high risk patients may receive prophylactic implant of a permanent pacemaker (PPM) prior to TAVI. The long term ventricular pacing percentage (VP%) burden for these prophylactic PPM patients is not well characterized.

Objective: To assess factors associated with VP% burden after TAVI for patients receiving prophylactic PPM.

Methods: We assessed our institution’s TAVI database to identify and obtain data for all TAVI patients who received prophylactic PPM implanted up to 100 days prior to TAVI. Additionally, we sought pacing follow up data for each patient through until Dec 31st 2021.

Results: 47 patients (37 male, 83±6 years at TAVI) were identified who had prophylactic PPM implanted 31 days (IQR 15 - 67) pre TAVI. We attained VP% data for 44 (94%) patients. Prior to TAVI, 55% of patients (n=24) did not use pacing (VP <1%) at baseline pre TAVI check. Pacing data was available both pre & post TAVI for 35 patients, with the first follow up check at 48 days (IQR 19 - 378) post TAVI, and for n=26 with further follow up checks the final follow up was 759 days (IQR 411 - 1038) post TAVI. As shown in figure; 13 patients remained VP <1% pre & post TAVI (nil-group), 10 had high VP% throughout (high-group), but the remaining 12 patients had a marked increase of VP% post TAVI (change-group). 83% (10/12) of change-group had right bundle branch block (RBBB) as PPM indication compared to only 31% (4/13, p = 0.03) of nil-group. Computer tomography assessed membranous septum (MS) length and fluoroscopic implant depth (ID) for 10 patients showed a positive delta MS-ID for the nil-group (2.1±3.3cm) compared to a negative delta MS-ID for the change-group (-3.1±4.0cm, p = 0.02).

Conclusion: RBBB and negative delta MS-ID are associated with greater VP% need after TAVI for patients receiving prophylactic PPM.

MICROTUBULE DETYROSINATION AND TRP1 DRIVE STRETCH-INDUCED ARRHYTHMIAS IN THE ISOLATED RABBIT HEART

Jessi Bak; Emma A. DeLong and T. Alexander Quinn PhD

Background: In hypertension, acute fluctuations in ventricular load can trigger arrhythmias due to myocardial stretch. In single rabbit ventricular myocytes, the incidence of stretch-induced arrhythmias (SIA) is increased by microtubule (MT) densification or detyrosination and transient receptor potential ankyrin 1 channel (TRPA1) activation, as occurs in hypertension.

Objective: To determine the dependence of SIA on MT densification or detyrosination and TRPA1 in the whole rabbit heart.

Methods: Isolated rabbit hearts were instrumented with surface electrodes to monitor electrical activity and an intraventricular balloon to alter left ventricular (LV) load. Transient changes in LV volume were applied (50-500 µL, 10 mL/s, 20 repetitions of each volume separated by 30 s) and the volume that resulted in a 50% SIA incidence (V50) was determined (Fig. 1A). Paclitaxel (5 µM) was applied alone to increase MT density and detyrosination, with parthenolide (10 µM) to prevent the increase in detyrosination, with colchicine (10 µM) to prevent the increase in density, or with HC-030031 (10 µM) to block TRPA1. Immunofluorescence was used to determine MT density (Fig. 2A) and the nature of SIA was assessed by voltage optical mapping.

Results: Paclitaxel reduced the threshold for SIA (decreased V50: 133±16 µL vs 173±11 µL in control; n=8, p=0.02 by paired t-test; Fig. 1A/B), which was associated with an increase in MT density (67±1% vs 56±2% in control; n=17, p=0.02 by one-way ANOVA Sidák post hoc test; Fig. 2A/B) and resulted in conversion of stretch-induced excitation to sustained activity. Parthenolide prevented the decrease in V50 (262±27 µL vs 256±26 µL; n=8, p=0.73; Fig. 1B) but not the increase in MT density (58±3%; n=17, p=0.12; Fig. 2B). In contrast, colchicine prevented the increase in MT density (49±3%; n=17, p<0.01; Fig. 2B) but not the decrease in V50 (190±36 µL vs 249±33 µL; n=8, p=0.04; Fig. 1B). The decrease in V50 with paclitaxel was also prevented by HC-030031 (249±26 µL vs 237±27 µL; n=8, p=0.60; Fig. 1B). Optical mapping showed stretch-induced focal excitation originating from the LV free wall, which after paclitaxel lead to re-entrant activity in some cases.

Conclusion: MT detyrosination, not densification, leads to a reduced threshold for TRPA1-dependent SIA in the rabbit isolated heart.