Abstract 8 Figure 1 Stent expansion index (SEI) assessment process

independent predictors of MACE during follow-up. (Figure 1, Panel C).

Conclusions The present study identified $SEI \geq 0.85$, associated with lower incidence of MACE as optimal cut-off in daily practice. Along with SEI of <0.85 , coronary calcification was also found to be significantly associated with MACE during post-PCI follow-up.

9 TARGET VESSEL AND NON-TARGET VESSEL RELATED EVENTS AT 10 YEARS POST PERCUTANEOUS CORONARY INTERVENTION

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Background Patients who undergo percutaneous coronary intervention (PCI) are at risk of events related to the stented vessel (TVRE) and events related to progression of disease in non-stented vessels (NTVRE). Long-term follow up may be useful to define the relative contribution of TVRE and NTVRE to overall event rates post PCI and to guide patient care. AIM To define the incidence of TVRE and NTVRE through 10 year follow up in patients post PCI with newer generation DES.

Methods The current study is an analysis of patient level data from the ISAR-TEST 4 and ISAR-TEST 5 trials. The endpoints of interest were TVRE (a composite of first target vessel myocardial infarction or target vessel revascularization) and NTVRE (a composite of first non-target vessel MI or non-target vessel revascularization) through 10 years.

Results We included 4,953 patients in this analysis. Through 10 year follow up post PCI, TVRE occurred in 1,238 of 4,953 patients (cumulative incidence: 25.8%) and NTVRE occurred in 1,442 of 4,953 patients (cumulative incidence: 30.3%). The majority of events in both groups were

revascularization events. From 0-1 years, the cumulative incidence of TVRE was 15.9% and of NTVRE was 11.2%. From 1-10 years, the cumulative incidences of TVRE and NTVRE were 12.3% and 22.4% respectively.

Conclusions At 10-years post PCI with new generation drug eluting stents, events related to remote vessel disease progression account for a higher proportion of events than events related to the stented vessel.

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AUTOMATED HIGH DENSITY LEFT ATRIAL VOLTAGE MAPPING OF LOW VOLTAGE AREAS IN ATRIAL FIBRILLATION DIFFERS FROM SINUS RHYTHM

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Introduction Low voltage areas (LVAs) are used as surrogate markers for diseased left atrial (LA) tissue and scar distribution. LVAs can be overestimated when measured in atrial fibrillation (AF) due to factors such as variable contact between mapping catheter electrodes and the endocardium in the presence of multiple depolarisation wavefronts with typically decreased cycle lengths. Scar or substrate modification was previously recommended for those with persistent AF (PeAF). Correlation between voltages in sinus rhythm (SR) and AF have previously been measured in studies via point-by-point analysis. An automated voltage histogram analysis (VHA) software module (CARTO3, Biosense Webster) has recently been validated for rapid area assessment of LA voltage maps and allows for very high-density analysis. We sought to compare LA LVA burden in SR to AF using VHA.

Methods We included patients over age 18 with PeAF who underwent de novo pulmonary vein isolation (PVI) with no additional lines, maps with ≥ 1000 voltage points in each rhythm and a uniform procedure with initial mapping in AF then remapping in SR after PVI. We created 6 anatomical segments (AS) from each atrium for comparison in AF and SR

Abstract 10 Table 1 Correlation between LVAs identified in AF and SR

Table 1: Correlation values of measured LVAs in SR and AF via Spearman's correlation test of each anatomical segment. We have compared rhythms in either the Dense Scar (≤ 0.2 mV) category or Diseased LA Tissue (0.21-0.5mV) category. *Denotes significance. AS = Anatomical segment, AF=Atrial fibrillation; SR=Sinus rhythm.

AS	Correlation Coefficient, AF and SR ≤ 0.2 mV	P value	Correlation Coefficient, AF and SR 0.21-0.5 mV	P value
Roof	0.514	0.001*	0.152	0.247
Lateral	0.395	0.012*	0.165	0.209
Septal	0.425	0.006*	0.252	0.052
Floor	0.431	0.005*	0.091	0.49
Posterior	0.555	<0.001*	0.314	0.015*
Anterior	0.499	0.001*	0.235	0.071

including the Anterior, Posterior, Roof, Floor, Lateral and Septal AS (figure 1). We removed the mitral annulus, trans-septal puncture site and pulmonary veins for VHA analysis. The total area of each AS was categorised by voltage into 10 aliquots between 0-0.5mV, designating voltages ≤ 0.2 mV as 'Dense Scar' and voltages 0.21-0.5mV as 'Diseased Tissue'. Data attained from VHA outputs were analysed using SPSS v.26.

Results Using VHA we analysed 58,342 (n=10 patients) voltage points between all AS (N=120). While comparing corresponding AS in voltage ranges consistent with Dense Scar, ≤ 0.2 mV, we found comparable LVA burden between rhythms in each AS except for the posterior wall, where there was significantly greater burden in AF ($159.03 \text{ mm}^2 \pm 194.65$ Vs. $102.14 \text{ mm}^2 \pm 157.47$, $p = 0.02$). Utilising Spearman's correlation test (table 1) for voltages ≤ 0.2 mV we found significant correlation coefficients in the range of $R=0.395-0.555$ for each segment as displayed in figure 2. Assessing ranges consistent with Diseased Tissue, 0.21-0.5mV, we found greater LVA burden in all AS when measured in AF, and this reached

Abstract 10 Figure 1 Example of walls isolated from each LA

Figure 1: Demonstration of left atrial Anatomical Segments. A: Anterior. B: Roof. C: Floor. D: Posterior. E: Septal. F: Lateral.