Post-Infarct Ventricular Tachycardia Substrate: Characterisation and Ablation of Conduction Channels using Ripple Mapping

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## *“Ripple mapping substrate modification of post-infarct VT”*

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## Conflict of Interest Statement and Disclosures

Imperial College holds Intellectual Property relating to Ripple Mapping on behalf of PK and NL, who have also received royalties from Biosense-Webster. PK, NL, SJC and VL have received consulting fees with respect to Ripple Mapping from Biosense-Webster. The remaining authors have nothing to disclose.

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## Word Count

5000

# Abstract

## Background

Conduction channels have been demonstrated within the post-infarct scar and appear to be co-located with the isthmus of ventricular tachycardia (VT). Mapping the local scar potentials (SPs) that define the conduction channels is often hindered by large far-field electrograms generated by healthy myocardium.

## Objectives

We sought to map conduction channel using Ripple Mapping to categorise SPs temporally and anatomically. We tested the hypothesis that ablation of early SPs would eliminate the latest SPs without direct ablation.

## Methods

Ripple maps of post-infarct scar were collected using the Pentaray during normal rhythm. Maps were reviewed in reverse and clusters of SPs color-coded on the geometry, by timing, into early, intermediate, late and terminal. Ablation was delivered sequentially from clusters of early SPs, checking for loss of terminal SPs as the endpoint.

## Results

The protocol was performed in 11pts. Mean mapping time was 65±23min, to collect a mean 3050±1839 points. SP timing ranged from 98.1±60.5ms to 214.8±89.8ms post QRS-peak. Earliest SPs were present at the border, occupying 16.4% of scar, whilst latest SPs occupied 4.8%, at the opposing border or core. Analysis took 15±10 min to locate channels and identify ablation targets. It was possible to eliminate latest SPs in all patients without direct ablation (mean ablation time 16.3 ± 11.1min). No VT recurrence was recorded; mean follow up 10.1 +/- 7.4months.

## Conclusion

Conduction channels can be located using Ripple Mapping to analyse scar potentials. Ablation at channel entrances can eliminate the latest SPs and is associated with good medium-term results.

# Key Words

Ventricular Tachycardia, 3D Mapping, Ablation, Substrate Modification, Ripple Mapping

# Funding

This work was supported by a research IIS-516 conducted with financial support from the Investigator-Initiated Study Program of Biosense-Webster, and a Project Grant from St. Mary’s Coronary Flow Trust.

# Introduction

Patients with previous myocardial infarction are susceptible to re-entrant ventricular tachycardia (VT) maintained by a diastolic isthmus within scar. Catheter ablation of the isthmus is effective at preventing recurrences, but can be difficult when faced with hemodynamic instability, presence multiple VT morphologies or non-inducibility.

Substrate mapping has been proposed as an alternative method to identify and modify critical sites for re-entry, during normal rhythms.1, 2 3D navigation systems were initially used to create bipolar voltage maps, denoting areas of low amplitude signal as scar. Empiric ablation was delivered around scar borders, transecting scar.2 Ablations became increasingly extensive until the entire scar was ‘homogenised’.3, 4 Sophisticated approaches tried to identify channels of viable myocardium, that could also act as diastolic pathways.5, 6 Bipolar voltage mapping could differentiate channels with slightly higher voltage amplitude from the very low amplitude scar.6 However, differentiating the higher channel signal amplitude from far-field can be difficult and did not always correlate with the isthmus.5 Therefore, extensive ablation of all late potentials has been advocated as an alternative.3, 7

Recently, activation mapping has been used to locate the entrances to substrate channels, with attention to the timing of the electrogram signal rather than its amplitude. This was based on the concept that channels form an interconnected network and ablating their entrance should lead to loss of the signals within the scar core.9 This method of ablation, ‘dechannelling, is an effective method for substrate modification.8, 9 However, these techniques can be labour-intensive; there is a need to manually tag, allocate and interpret signals of interest as well as to specifically position catheters in a position that allows suitable signals to be mapped.

Ripple mapping (RM) was designed to overcome pitfalls of 3D mapping and shown to improve atrial tachycardia ablation by differentiating low-voltage critical isthmuses from non-functional scar.10, 11 It has been applied in ischemic VT to show co-location of conduction channels in scar with diastolic isthmuses; ablation of these channels can prevent VT recurrence.12, 13 We tested the feasibility of using RM to identify and ablate early scar potentials (SPs) in order to eliminate late SPs.

# Methods

## Population

We recruited patients with post-infarct VT undergoing catheter ablation. The study protocol was developed adhering to guidelines established by the Helsinki Declaration 2013 and approved by the London Queens Square Health Research Authority ethics committee (19/LO/0637). Patients were recruited at Imperial College Healthcare NHS Trust.

## Electroanatomic Mapping and Ablation Protocol

Patients underwent a mapping protocol designed to locate Ripple Mapping Conduction Channels (RMCC) as previously described13. The protocol for mapping is given in detail in the Supplemental Material and explained schematically in Figure 1. A dense bipolar voltage map of the LV was collected during SR with typical limits (0.50–1.50mV) to define scar.2 The RM window was set from the onset of the QRS to the end of the T wave. RMs were reviewed in reverse and clusters of Ripple bars (>3, to avoid misinterpretation of noise) activating simultaneously, in areas of scar, considered to represent SPs from local activation within RMCCs. Design lines were used to mark the geometry with a circle around each cluster. Lines were coloured according to the time quarter during which SPs within activated. Time quarters were determined by taking the time from a peak QRS deflection to the latest SP identified, dividing it into equal quarters. Ablation was delivered sequentially from clusters of early SPs, checking for loss of terminal SPs without direct ablation (full details in Supplemental Materials). The study acute endpoint was met if terminal RMCC SPs were eliminated without direct ablation. Conduction channels were analysed by area of clusters and conduction time (full details in Supplemental Materials)

## Follow-Up

Patients were followed for 12 months by ICD interrogation. A recurrence of VT was defined as a sustained episode of VT lasting >30s and documented either by the patient’s device, or, on a 12-lead body surface ECG and occurring after a 2-week blanking.

## Statistics

Categorical variables expressed as percentages and analysed using Fishers or Chi Squared Tests. Continuous variables as mean ± 1 SD, analysed using the T-test or a Mann Whitney U test. A 2-way ANOVA and multiple comparison models (Tukey’s) were used where appropriate. A two-sided p value was determined where applicable and p≤0.05 considered significant.

# Results

## Demographics

11 male patients were recruited: mean age 68.6±6.4 years and mean LVEF 34.1±7.8%. The indication for ablation was for recurrent ATP in 2 (18%), single ICD shock in 2 (18%), recurrent shocks in 4 (36%) and slow VT in 3 (27%). Full demographic details in the Supplemental Materials (Table I).

## Electroanatomic Mapping

Single transseptal access was acquired in all patients, double in 1 (9%) and retrograde in 7 (64%). The mapped rhythm was intrinsic in 8 (73%). The difference in mapped cycle length between intrinsic and paced rhythms was not significant (634.5vs600ms, p=0.32). The mean time required for mapping was 65.0±22.8mins for 3050±1839points per map. Details presented in Table 1. In 9, the presenting rhythm was normal, and a substrate map performed from the start. The remaining 2 presented for ablation in VT. Of these the arrhythmia was terminated by ablation in 1 and by pacing in the other, followed by substrate mapping. Therefore only 1 patient had any ablation prior to substrate mapping. PES was performed in 4 (36%) and induced stable monomorphic VT in 2 patients.

## Ripple Mapping Conduction Channels (RMCCs)

RMCCs could be identified in all 11patients. The mean time of analysis was 15.7 ± 10.1 minutes. Figure 2 is an example case following the process of identifying RMCCs and categorising the clusters of SPs by activation quarter. As the RM is reviewed in reverse-play, the time coding for the clusters of SPs is defined by the latest deflection (first electrogram component when viewed in reverse). Video 1, corresponding to Figure 2, is an example of a RM reviewed in reverse and identification of SP Ripple bar clusters in each timing quarter. Terminal RMCC activation appears in the basal scar core progressing septally, laterally and apically to reach early RMCC quarters. In Video 2, again corresponding to Figure 2, an apical RMCC is demonstrated, with entrances apically and laterally and activation progressing in one direction to the terminal RMCC in the scar core. Next, a basal RMCC with septal and lateral entrances is shown; activation has chevron appearance consistent with wavefront traversing scar in either direction and collision within the channel.

### Temporospatial Categorisation of RMCCs

The earliest quarter of SPs activated with a mean delay of 98.1±60.5ms from the peak QRS or pacing artefact. An activation gradient could be demonstrated by measuring delay to RMCC quarters (Figure I – Data Supplement). Intermediate quarter SP activation occurred with mean delay 138.9±64.9ms, whilst late SP activation with 192±86.9ms before terminal SP activation occurred with 214.8±89.8ms. Three patients mapped during ventricular pacing had longer delay to SPs in each RMCC quarter. The early RMCC quarter occurred with a delay of 66.0ms during intrinsic conduction mapping vs 167.3ms during ventricular pacing. This is probably due to activation of the conduction system and the distance from stimulus to scar during pacing.

In all patients, the clusters of early SPs were observed to occur at the scar border zone, whilst terminal RMCC activation occurred distally from these sites as shown in Figure 2 - latest activation occurs almost at the contralateral border of scar. Activation from one border to the other was observed in 5 (45.5%). In 6, the terminal patch of SPs was in the core of scar (54.5%). Figure 3 provides an example of the temporospatial organisation of clusters of SPs within scar. Arrows point to SPs in early, intermediate, late and terminal quarters. The pattern of activation is from border (septal and lateral) toward the scar core. The terminal patch is composed of SPs with double potentials suggesting either a line of block or collision within channels. Note that despite the most proximal anatomical segment of the RMCC being designated as intermediate on the lateral border, they are still considered activation entrance sites into scar channels – this particular SP patch lateness is most likely due to the arrival of the septal wavefront to the scar border prior to the lateral wavefront arriving.

Although general direction of activation within RMCCs can be appreciated by following the colours of activation quarters, as in Figure 2, the path of activation between of SPs is more complex. Because the entire scar activation is reviewed on the RM, activation during the same time quarter may appear simultaneously but at spatially distinct sites of scar. Activation appears “jump” between individual clusters of SPs within the same time quarter. These concepts are demonstrated in Figure 4, and Video 3, where activation is mapped backward from a terminal site toward earlier quarters. The stochastic pattern of channel activation is apparent – from borderzone toward the scar centre - as clusters of Ripple bars appears during the same activation quarter at spatially distinct sites within an RMCC.

RMCCs occupied 42.5% of scar area (Figure II – Data Supplement) with clusters of early quarter SPs covering 11.1±6.9cm2 (16.4%) of scar area. Clusters of intermediate and late quarter SPs formed 7.8±4.7cm2 (11.5%) and 7.2 ± 5.1cm2 (10.7%) respectively. Clusters of terminal quarter SPs were restricted to 3.2±2.5cm2 (4.8%) of the scar. Early quarters were larger in area than terminal quarters (p<0.0001).

An example sustained VT pre-ablation is shown in Figure 5, and Video 4, in a patient with previous antero-apical infarction. This was the only patient who had ablation during VT and therefore confirms that ablation of the mapped diastolic isthmus was associated with VT termination, as shown in Video 4.

## RMCC Ablation Endpoint

It was possible to abolish SPs in terminal quarter clusters without direct ablation in all 11 patients. Ablation early quarter SPs exclusively led to elimination of the terminal SPs in 1 (9%) patient. In 7 (63%) ablation of early and intermediate clusters only was required to achieve loss of terminal SPs. In the remaining 3 (28%), ablation of late quarter SPs was also required. Direct ablation of the terminal quarter SPs was not performed in any patient to achieve abolition. Figure 6 (and Figure III – Data Supplement) provides an example of terminal SP signal elimination by ablation of early and intermediate quarters. Video 5 shows the appearance of the RM for Figure 6 before, and then following re-mapping once the substrate modification endpoint was met. Figure 7 presents the ablation strategy for the case in Figure 3. Ablation lesions are delivered to early and intermediate clusters of SPs. During ablation of the RMCC septal entrance, delay between the double potential in terminal SPs increased compared to pre-ablation. Further ablation to apical and lateral entrances (early and intermediate quarter of SPs) leads to elimination of one component of the double potential, followed by significant delay of the second. Eventually, all terminal SP activity is eliminated without any direct radiofrequency applications. Sustained monomorphic VT was not inducible, with at least 3 extra-stimuli, in any patient after loss of terminal scar potentials. Further detail in the Supplementary Material (Figure IV).

## Follow-Up

The mean time of follow up in this selected cohort of patients was 10.1 ± 7.4 months. 1 patient received an appropriate ATP therapy during the blanking period and then had an inappropriate shock during follow up. Details in the Supplementary Material (Table II).

# Discussion

In this study we show that selective radiofrequency ablation of early scar potentials identified by Ripple Mapping eliminates the latest scar potentials at spatially remote sites, without direct catheter ablation. This endpoint was associated with good medium-term outcomes.

Following myocardial infarction, remodelling within ventricular scar results in slow conduction characterised by electrograms that are delayed with split, fractionated and isolated components reflecting abnormal local activation.14 We referred to these bipolar electrograms as scar potentials (SPs). At present the most validated endpoint for substrate modification is the elimination of all mapped SPs or complete scar homogenisation by voltage mapping.3, 7, 15

Functionally guided substrate modification approaches, that consider both activation and voltage patterns of SPs, have been described as alternatives to extensive ablation required to homogenise scar. Bipolar voltage mapping to identify discrete channels within scar was first performed by lowering the bipolar voltage threshold of maps, according to predefined limits. Early reports suggested these channels were associated with the isthmus mapped during VT, but later studies were not able to identify the same prevalence or predictive value.5, 16 One explanation for the discrepancy is related to dependence of bipolar electrogram amplitude on factors such as angle of electrode orientation to the wavefront, electrode size, spacing and degree of contact force.17 Furthermore, automatic annotation of bipolar voltage, and activation time, can be challenging owing to far-field deflections, particularly if there is fusion between far field and local signal.17 In practice, the issue of annotation is compounded by the need for high-density mapping with collection and analysis of thousands of points.

## We used RM to design a technique to handle the extensive data analysis required to locate conduction channels. The maps in this study had about 3000points; by using reverse-play with RM, it was possible to focus on the most important areas. Clusters of SPs categorised by activation time were able to guide selective catheter ablation to channel entrances. Given the interconnected nature of channels, we hypothesised that latest SP activity can be eliminated if all endocardial entrances are adequately mapped and ablated. In the present study it was possible to demonstrate elimination of the latest SPs without extensive scar ablation. This principle has been demonstrated using linear electrode catheters, strategically placed to record both early and late SPs on the same catheter.8 Selective channel entrance ablation has also been tested in a prospective clinical study that led to VT non-inducibility in 54.5% (78.2% after further ablation) and long-term freedom from VT in 80%.9 Furthermore, studies have also demonstrated that scar exit block (core isolation) is a predictor of success.18 Collectively these findings suggest that the substrate for scar-related VT is linked to interconnected activation propagating from the scar borderzone with increasing delay. Our approach is conceptually like previous reports, in that we aim to eliminate the path of activation within scar channels. However, we believe that by using RM the process can be more reproducible between operators. Showing the whole electrogram and avoiding annotation means there is less scope for variation, especially with fractionated electrograms. We demonstrate that it is possible to map the path of activation through RMCCs. However, it is difficult both to determine and prove the exact sequence of activation within RMCCs, as in Figure 4. As a result, in our previous study, we ablated the entire RMCC with good outcomes.13 However, for substrate modification it is not necessary to know the exact path of activation through the RMCC. If the terminal SPs are lost, by definition, the path has been rendered non-functional. Nonetheless, it is likely that some channels ablated are blind alleys that can’t support VT - further work is needed to distinguish these and reduce the ablation burden further.

## On this background, the purpose of the current study was to establish whether latest SP elimination could be achieved by ablating early and intermediate SPs only. We chose to ablate in this sequence as it proves that these were interconnected channels. However, early scar potentials are the most extensive and the most difficult to identify as the SPs are very close to the far-field signal. Therefore, there is an argument for ablating intermediate SPs only following the same endpoints.

## Limitations

Our main limitation is that patients were selected for being clinically stable with good quality maps and electrograms, so that the protocol could be tested without confounding. We do not know in what proportion of consecutive, unselected patients, this is feasible. In previous studies, PES is often performed routinely before mapping to inform the results of post-ablation induction, as well as guide ablation. We did not mandate this; acute non-inducibility should be interpreted with caution. We did not analyse the effect of altering the mapped wavefront direction. Such an approach could delineate functional and anatomical channel borders. More detailed pacing maneuvers may have been able to differentiate isthmuses from blind-alleys, whilst proving exit block with pacing from terminal cluster may potentially be more reliable than remapping. Finally, it was not always possible to ablate all clusters in a quarter due to catheter stability. In clusters where operators were not able to achieve stable contact of more than 2g, we did not mark these as ablated. It may have been possible to have loss of terminal potentials with just early clusters ablated.

## Conclusion

In this study we used RM to identify channels of activation within scar. Ablation at channel entrance sites was able to eliminate the latest scar potentials without direct ablation. These findings provide further evidence of interconnected channels within post-infarct scar as an effective target for substrate modification.

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# LEGENDS

## Figure 1: Concepts behind the approach to Ripple mapping the post-infarct VT substrate.

Panel A presents a theoretical model of scar activation during normal rhythm. Red areas denote dense, non-conducting, scar and purple areas are a network of channels of functional tissue within. White arrows show possible activation pathways within conduction channels.

Panel B shows a RM played in reverse from the end of the T-wave. The first panel shows Ripple bars seen within a blind alley in the scar with the corresponding terminal SPs shown against the QRS. The next panel shows progression of the Ripple bars to the channel entrance with the associated earlier SPs below. Clusters of >3 Ripple bars are marked with circles coloured according to the timescale given for each timing quarter (early, intermediate, late and terminal).

In Panel C radiofrequency (RF) ablation has been delivered to the channel entrances only. This blocks activation from progressing into channels, which are re-assessed with a recording catheter for confirmation of the endpoint.

## Figure 2: Constructing a Ripple Mapping Conduction Channel (RMCC).

This example corresponds to Video 1. The RM window (blue calliper) extends 180ms post QRS peak (reference – red calliper), based on the timing of latest SP. The window is divided into 45ms quarters, making up early, intermediate, late and terminal RMCC quarters for categorisation of the SPs. The bipolar voltage map is set to 0.50-1.50mV.

Panel A: The RM is stopped at 140ms post reference, a frame within the ‘terminal quarter’, demonstrated by an orange calliper. Clusters of >3 Ripple bars have been encircled in white following the colour code, based on their latest deflection.

Panel B: The RM is reviewed in reverse, frame-by-frame now stopped at 100ms reference. Activating SP clusters are within this ‘late RM quarter’ and encircled green.

Panel C: The RM has been stopped 60ms post reference, within the ‘intermediate quarter’. Clusters of SPs are encircled orange.

Panel D: The Ripple map has been stopped at 35ms post reference within the ‘early quarter’. Clusters of SPs are encircled red.

The Ripple bar clusters on the geometry represent activation within RMCCs. The color-coding gives an impression of direction, but the exact path is difficult to ascertain. The activation sequence also depends on the direction from which it enters scar. Whilst green circles represent late RMCC quarter SPs, these can be entrances if on the scar border contralateral to the direction of the approaching wavefront.

## Figures 3: The spatio-temporal relationship between RMCCs and scar potentials.

The bipolar voltage map of the LV in a modified AP with voltage settings 0.5-1.5mV. Color-coded clusters defining RMCCs are displayed within scar. Corresponding SPs are pictured adjacent. Earliest SPs are on the septal side where activation first enters scar. Activation proceeds from the septal border to the core where the white circles contain terminal SPs. The channel entrances on the lateral side fall within the intermediate quarter due to the time taken for LV activation to reach them. Activation in the terminal quarter, contains double potentials indicative of a local line of block within the RMCC.

## Figure 4: Determining the Activation Path within the RMCC.

The bipolar voltage map in this example is set to 0.5-1.5mV with an inferior scar. The left-hand RM has been stopped between the terminal and late quarters. As there are only few clusters adjacent, it is possible to propose a pathway of activation designated by black arrows. The right-hand RM has been stopped between the late and intermediate quarters. The progression of activation is not smooth; abrupt changes of direction manifesting as distant clusters of Ripple bars activating at a similar time with near Ripple bars. This type of pathway of activation is usually difficult to establish or prove.

## Figure 5: Spatial relationship between Ventricular Tachycardia Diastolic Isthmus and Conduction Channels.

12 lead ECG of incessant VT shown on panel A1. The RM of this VT is shown on a bipolar map, limits 0.5-1.5mV on the same panel, paused at a time of mid-diastolic activation, with signals indicated. White arrows demonstrate the circuit as viewed live on the RM (Video 4). Note the entire circuit is large and within ‘scar tissue’ (<1.5mV), consistent with 640ms cycle.

In Panel A2 the bipolar voltage map has been thresholded so that no diastolic Ripple bars appear in red areas. At this limit, 0.15-0.20mV, diastolic pathway boundaries appear to be anatomical scar medially, and functional line of block, between ‘islands’ of non-functional scar, on the other. Panel A3 shows the site of RF termination for VT near the exit of the isthmus, and all ablation lesions. Thresholding and Termination shown in Video 4.

Panel B1: Once the VT terminated a substrate map collected during RV pacing, 600ms. The RMCCs are mapped and shown on a bipolar voltage map, 0.5-1.5mV. The solid white line outlines the VT isthmus at threshold, 0.15-0.20mV. Electrograms collected during substrate mapping are shown in the adjacent panel, with the appearance of a chevron suggesting activation collision from two entrances.

Panel B2 shows the LV geometry with white circles around isthmus SPs, and red circles around substrate SPs. Though the isthmus co-locates with substrate SPs, there are several clusters that do not indicating complexity that needs further study.

## Figure 6: Loss of Terminal Scar Potentials Without Direct Catheter Ablation.

The completed RMCC clusters are shown on a grey geometry with scar border (1.5mV) traced round in white. The left-hand panel shows the Pentaray sitting over a terminal quarter patch and the electrograms from the catheter are shown adjacent. The terminal scar potentials are asterisked.

The right-hand geometry shows the red tags where RF has been delivered over red and orange clusters near the channel entrance. Note there are no RF lesions near the white patch where the Pentaray is positioned. The terminal scar potentials are no longer present on the adjacent electrograms and have been eliminated without direct ablation.

## Figure 7: Multiple Entrance Ablation in Unconnected Network of Channels.

This example shows the effect of ablation of RMCCs presented in Figure 3. Ablation lesions shown as red tags on the bipolar voltage map started at the septal early scar potentials (encircled in red). Following septal ablation, the double potential split prolongs from 70ms to 130ms. Ablation is continued at lateral RMCC entrances composed of early, intermediate and late quarter SPs. The ablation catheter is again returned to the terminal quarter and one component of the double potential is now lost. This indicates that the lost component was due to septal and lateral channel entrances. Ablation lesions were then delivered to early and intermediate quarters at the apex. Returning the ablation catheter to the terminal RMCC now demonstrates loss of all local SP activity, suggesting that the channel communicating with the apical entrance was not connected from septal to lateral entrances. The specific activation pathway is difficult to prove, but some fundamentals of the communication between channels can still be deduced.

# TABLE 1

|  |  |  |  |  |  |  |  |  |
| --- | --- | --- | --- | --- | --- | --- | --- | --- |
|  | **Points (n)** | **Rhythm** | **Rate (ms)** | **Scar** | **Scar**  **area (cm2)** | **Scar proportion (%)** | **Mapping**  **(mins)** | **Analysis**  **(mins)** |
| **1** | 2574 | Intrinsic | 600 | Inferior (base–apex) | 72.8 | 45.6 | 54.7 | 24.2 |
| **2** | 3511 | Intrinsic | 750 | Inferior (base) | 45.6 | 26.7 | 56.7 | 27.4 |
| **3** | 2087 | Intrinsic | 600 | Inferior (base) | 57.1 | 29.9 | 85.7 | 16.8 |
| **4** | 2234 | Intrinsic | 600 | Inferior (base–apex) | 107.5 | 38 | 48.9 | 20.9 |
| **5** | 2485 | Intrinsic | 600 | Inferior (base–apex) | 67.4 | 22.3 | 59.6 | 33.1 |
| **6** | 2790 | Intrinsic | 660 | Apical | 51 | 27.3 | 44.5 | 5.8 |
| **7** | 2450 | Paced  (RV) | 600 | Inferior (base) | 43.8 | 19.9 | 57.2 | 8.9 |
| **8** | 4419 | Paced (GCV) | 600 | Inferior (base–apex) | 130.7 | 48.7 | 87.2 | 9.5 |
| **9** | 7803 | Intrinsic | 600 | Anterior (septal) | 120.1 | 52 | 112.2 | 3.2 |
| **10** | 2685 | Intrinsic | 666 | Inferior (base–mid LV) | 55.9 | 29.5 | 74.9 | 3.9 |
| **11** | 512 | Paced  (RV) | 600 | Antero-apical | 52.3 | 28.8 | 33.4 | 19.6 |
| **Mean** | **3050 ± 1839** |  | **625.1 ± 48.5** |  | **73.1 ± 31.4** | **33.5 ± 10.9** | **65.0 ± 22.8** | **15.7 ± 10.1** |