# Ventricular Tachycardia Ablation in the Post Infarct Patient

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## Introduction

It is estimated that 70,000 of deaths in England and Wales are due to sudden cardiac death every year (1). Holter monitoring data suggests that approximately 85% of cases are due to some kind of ventricular arrhythmia. Whilst there is a large spectrum of aetiological causes of sudden cardiac death, coronary artery disease is most commonly identified in at least 80% of overall cases in the Western World (2).

Catheter ablation as a therapeutic option for ventricular arrhythmias has made significant strides over recent years with new evidence from prospective randomized studies on outcome in patients with ischaemic heart disease (3). Current international guidelines recommend catheter ablation of VT as an adjunct to anti-arrhythmic therapy in patients experiencing appropriate ICD shocks (4). Although there is no evidence that catheter ablation of VT reduces mortality, early referral for catheter ablation following ICD intervention has the potential to improve patient quality of life (5). In this BHRS editorial, we discuss how post infarct Ventricular Tachycardia can be targeted in the EP lab.

*The pathological basis for re-entry*

Following myocardial infarction, dead tissue is progressively replaced with scar tissue (6). Scar tissue is electrically inactive and forms a focus around which re-entrant circuits can develop. Critical to this are bundles of viable tissue that reside within the border zone between the scar and healthy myocardium. These bundles are known as conduction channels (7). Electrical conduction along these channels is slow as the coupling between myocytes is less efficient and occurs over a prolonged “zigzag” route from entry into the channel to exit (8). These “slow conduction channels” are protected by areas of dense non-conducting scar tissue that allow sufficient delay in activation, leading to recovery of the excitability of remaining ventricular tissue during tachycardia. During VT, activation exits this channel, usually at the scar border, and circulates by an outer loop back to the entrance to set up the next beat of VT. VT circuits often involve more than one channel, with inner and outer loops and bystander sites of activation (see figure 1). The prime target in VT ablation are “slow conduction channels” within scar.

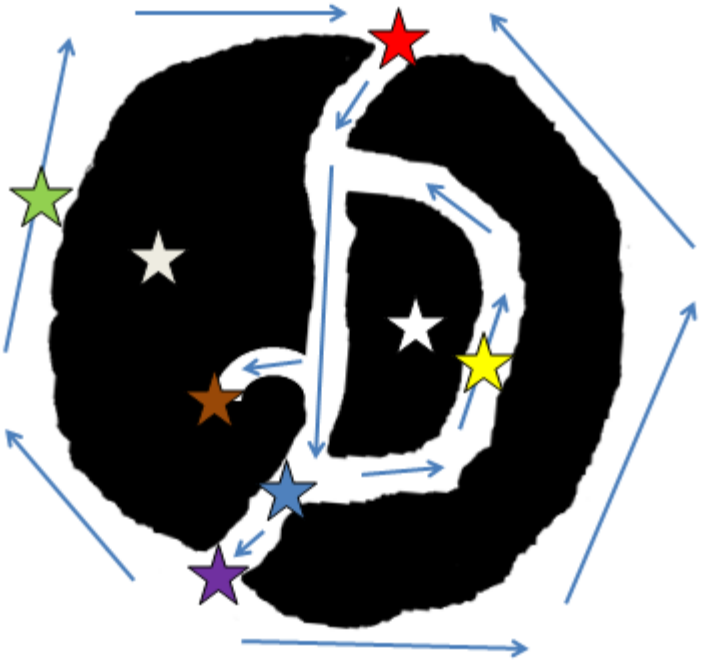


Figure 1: Illustration of a re-entrant circuit within scar. A channel of viable conductive tissue is seen bordered by 2 islands of scar (white stars) between the entrance (red star) and exit (purple star) at the scar border zones. The arrows show a typical “figure of 8” direction of activation through the channel, and around an inner loop (yellow star) and outer loop (green star). Some branches within a channel can also lead to a dead end – bystander site (brown star). A site shared by both loops within the isthmus is an ideal site for ablation (blue star).

## Locating "slow conduction channels” within scar

### The Surface Electrocardiogram

As the mass of tissue in the slow conducting channel is small, electrical activation within the channel during VT cannot be seen on a 12 lead ECG. Breakout of activation at the exit site depolarises the rest of the ventricular myocardium, giving rise to the surface QRS complex. Several algorithms exist to determine the exit site based on the ECG, an example of which is as follows (9).

**Right bundle branch block configuration indicates left Lateral exit**

**Left bundle branch block configuration indicates Septal or Right Ventricular exit**

**Superior axis – positive in AvR and AvL indicates Inferior wall exit**

**Inferior axis - positive in inferior leads indicates Anterior wall exit**

**Positive pre-cordial concordance suggests Basal exit.**

**Negative pre-cordial concordance suggests Apical exit.**

**Qs signal (negative vector) in any lead means the wavefront is moving away from that site - using the precordial leads: V2-V4: anterior wall, V3-V5: apex, V5-V6: lateral wall.**

**The wider and more slurred the QRS, the more likely the origin is epicardial.**

There are caveats: a single conduction channel may branch out to have multiple exit sites and multiple VT patterns. Furthermore, during VT areas of functional conduction block rather than anatomical block may give rise to VT morphologies not predictable by surface ECG alone.

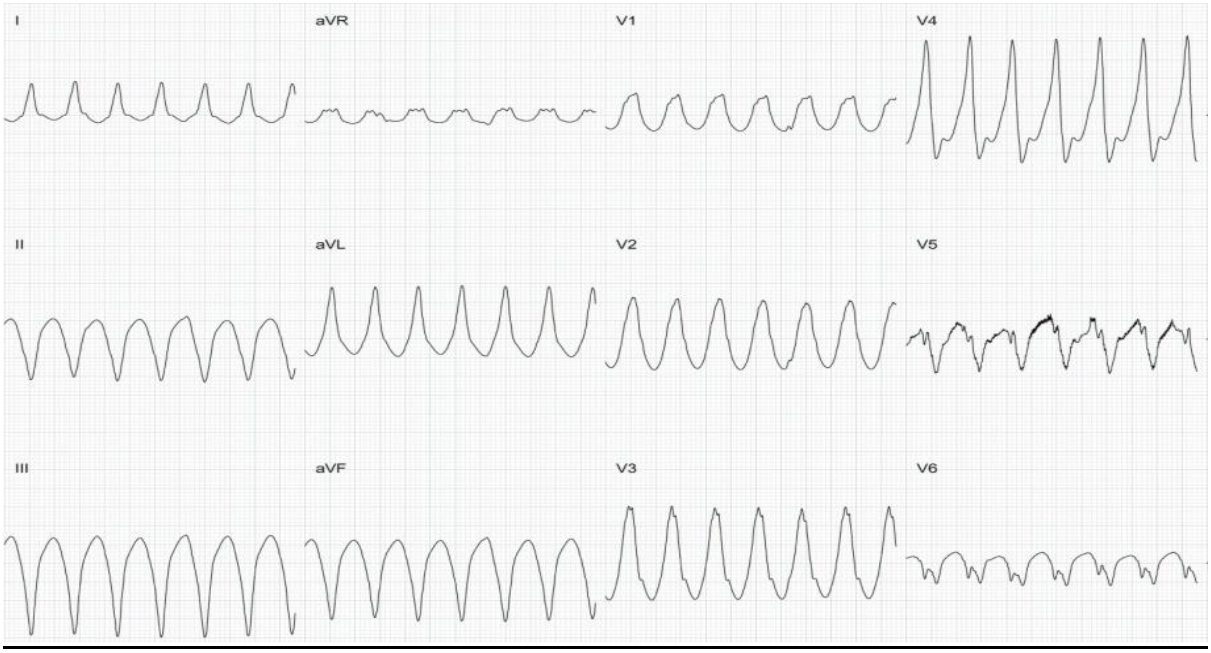



Figure 2: In this example, the VT has a right bundle branch block configuration, superior axis (negative in inferior leads, positive in AvL and AvR), Qs signal in V5 and V6. This is in keeping with an exit site in the left infero-lateral wall.

**During Electrophysiological Studies**

*Intra-cardiac Electrograms*

Bipolar signals localise intra-cardiac electrical activity between two electrodes at the distal end of the catheter. In coronary disease, 85% of VTs have their site of origin located in sites that contain “abnormal electrograms” seen either in sinus rhythm or during tachycardia (10).

a) Electrogram analysis during VT

If mapping is tolerated during VT, a roving mapping catheter can be moved within the ventricle to look for “diastolic potentials.” These are usually low amplitude signals within scar that occur prior to the VT QRS inscription (pre-QRS) and represents activation within the critical isthmus

before exiting the channel (11). Localisation of this signal is highly suggestive of proximity within the circuit.

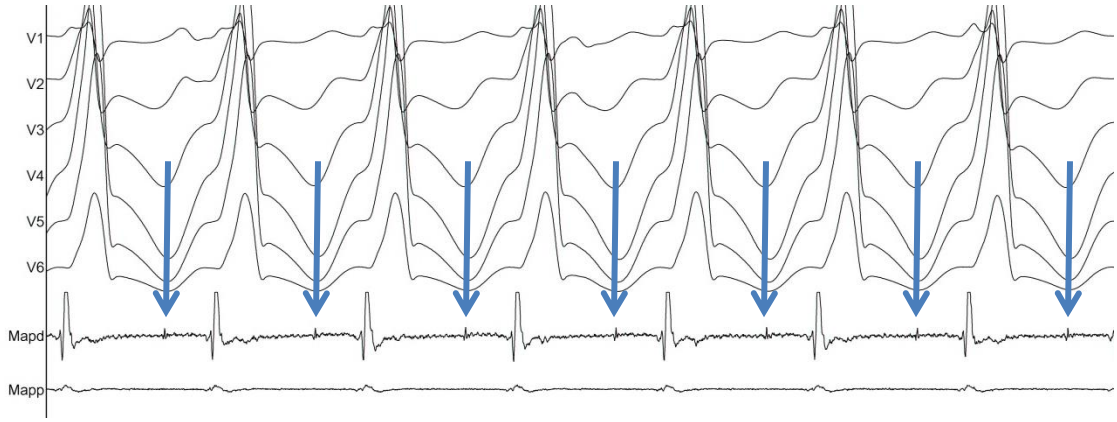



Figure 3: Intra-cardiac electrogram during VT – a clear “pre-systolic”/ “diastolic signal” can be seen in the distal bipolar mapping electrode before the inscription of the surface QRS. Ablation at this point terminated the tachycardia.

b) Electrogram analysis in sinus rhythm

During sinus rhythm, areas of slow conduction through the channel appear as long-duration, low amplitude, multi-component “fractionated” signals (12). These have been collectively described as local abnormal ventricular activities (LAVA) (13) - each peak of a fractionated electrogram may represent depolarization of a separate myocyte bundle separated by fibrosis from a neighbouring bundle (14).

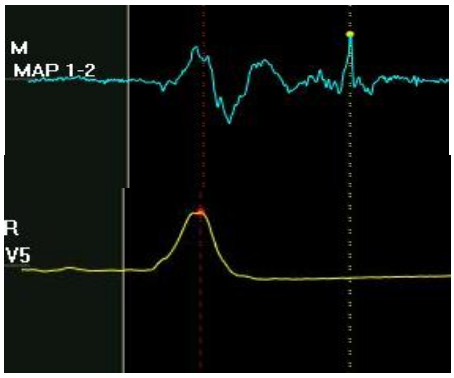



Figure 4: The distal electrode pair on the mapping catheter (MAP 1-2) has detected a long, low amplitude multicomponent signal that extends well beyond the body surface V5 electrogram signal. The first arrow reflects a far field V signal. The remaining is typical of fractionation.

A “late potential” reflects a type of LAVA where there is local depolarization of surviving fibre bundles that are well insulated by dense scar (10). As the wavefront travels slowly, local activation is recorded well after the higher-amplitude far-field electrogram and surface QRS complex (15).

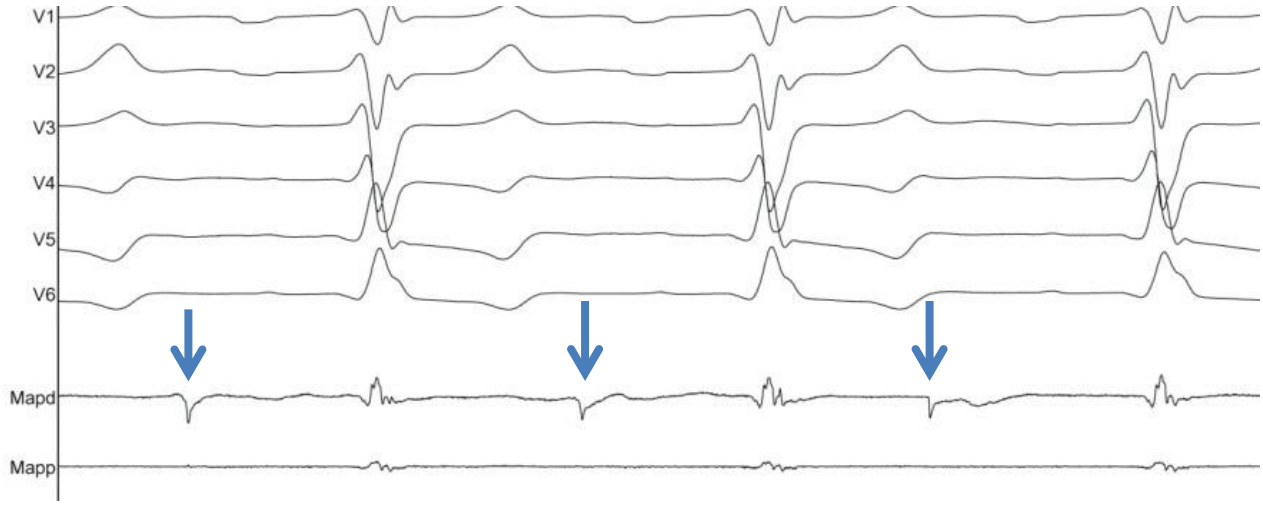



Figure 5: Intra-cardiac electrogram during sinus rhythm – a clear isolated "late potential" signal is seen well after the end of the surface QRS complex separated by a clear isoelectric portion.

*In the Electrophysiology lab:*

a) Activation mapping.

If VT is sustained and cardiovascularly tolerated, activation of the whole ventricle can be mapped in order to locate the critical isthmus of conduction. VT is induced using programmed electrical stimulation: A pacing catheter is placed at a site within either ventricle and usually a drive train of 8 stimulating beats followed by a number of extra-stimuli at progressively shorter coupling intervals are delivered in an attempt to enter the excitable phase of ventricular repolarisation and hence induce re-entry.

A roving or mapping catheter can then be moved to multiple ventricular locations, where sequential analysis of the underlying electrical signals in relation to the onset of the surface QRS is performed. Signals found by the catheter that precede the surface QRS onset (pre-systolic signals) are likely to be close to the channel as they precede the exit site. Signals found by the catheter during the inscription of the QRS are therefore likely to be outside the channel. Hence, an "activation map" is created by plotting the electrogram timing relative to the QRS onset, and the earliest signals usually represent the exit point and region of interest for ablation. 3D mapping systems represent activation as a colour coded map to help locate the exit site. Recent advances in multipolar mapping technology should allow collection of several activation points within a short period of time, speeding up the mapping process.

b) Entrainment mapping

Activation mapping often directs the operator to sites of interest that are early or pre-QRS during VT. Once in these areas, entrainment techniques can then be applied. Entrainment is continual resetting of a re-entrant circuit. During VT, entrainment can be performed by pacing the myocardium at a point of interest faster than the tachycardia (shorter cycle length) to capture the ventricle, entering the re-entry circuit and speeding it up (entraining). The response to entrainment can identify where in the circuit the pacing/mapping catheter is located. (16, 17).

*Concealed fusion:* When pacing from sites that are distant from the conduction channel, the paced wavefront and the VT wavefront will fuse, leading to a different QRS appearance to that of the clinical VT. When pacing from within the protected conduction channel, the paced wavefront will travel in the same direction as the tachycardia, using the same exit site, therefore the QRS morphology during entrainment should be identical to the VT QRS – this is known as "concealed fusion." (See figure 6)

*Stim-QRS:* The interval between the paced signal and surface QRS (stim-QRS) can be used to locate where in the isthmus the pacing site is – short intervals (<30% the tachycardia cycle length) suggest an exit point location, intermediate intervals (30-70% TCL) suggest locality within the channels itself, and >70% suggests localization outside the circuit.

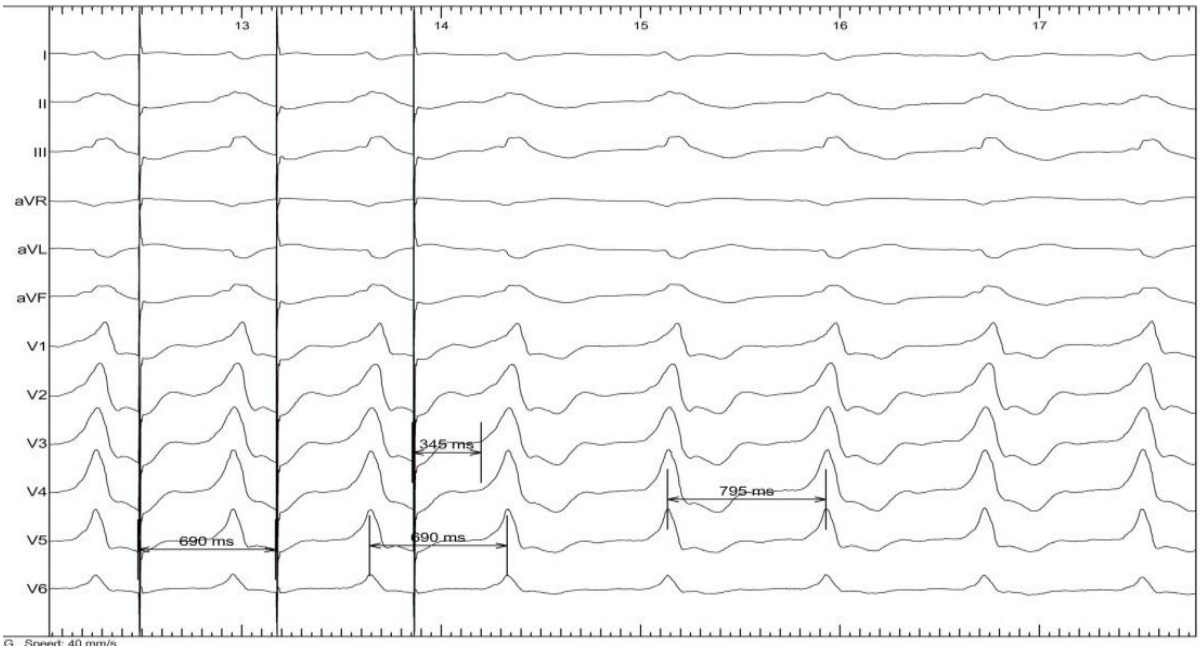
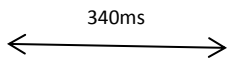



Figure 6. In this example, pacing at a cycle length (690ms) shorter than the tachycardia (795ms) has captured and entrained the VT. The paced QRS complexes match the tachycardia – there is concealed fusion. The Stim-QRS (348ms) is between 30-50% of VTCL. This is indicative of a mid-channel location.

*Post Pacing Interval*: The post-pacing interval (PPI) represents the time it takes for a paced wavefront to travel to the re-entry circuit, around the circuit, through the conduction channel, then back to the pacing site. Outside the circuit, the PPI will be longer than the time it takes for the tachycardia to travel around the circuit (i.e. one VT cycle length). Within the circuit, the PPI should be equal to the cycle length.



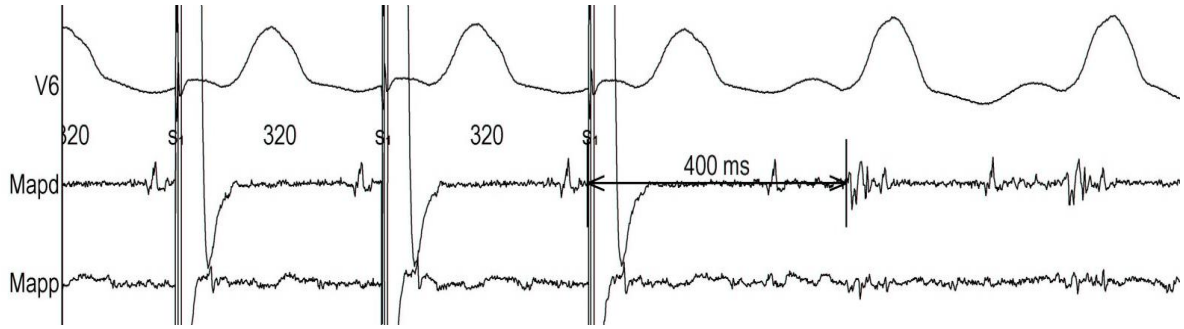



Figure 7: Measurement of the post pacing interval. PPI (400ms) is measured on the distal mapping electrode from the final stimulus artefact to the first deflection of the locally captured electrogram (note that the single deflection electrogram is a farfield signal and does not represent local activation).

*Stim-QRS & Electrogram – QRS*: Although the channel branches from the scar to exit at the border zone, some branches lead to a dead end – these blind alleys can lead to ineffective ablation. Bystander sites can be identified, by measuring the interval between the diastolic signal and the surface QRS (Electrogram-QRS) compared to the Stim-QRS. If the pacing interval is longer, this suggests the mapping catheter is located in a bystander region, taking longer to travel the extra distance out of this site. The stim-QRS delay should be equal to the electrogram-QRS at sites within the isthmus

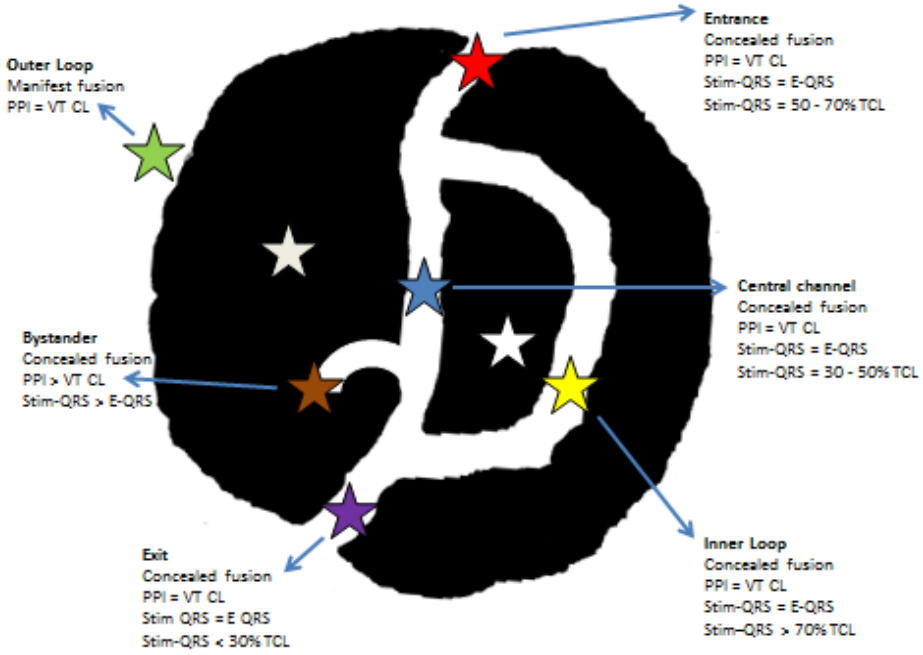



Figure 9: An illustration of a typical conduction channel, inner loop and bystander, as well as all the different entrainment strategies used to identify site location within the channel

Entrainment techniques can often be difficult, either failing to capture myocardium or risking termination or acceleration or change of the tachycardia which might result in cardiovascular collapse or deterioration to ventricular fibrillation, necessitating DC cardioversion.

Activation and entrainment mapping require a sustained cardiovascularly tolerated arrhythmia, which only accounts for ~15% of all VTs. For patients who present with intolerable, non-sustained or non-inducible VT (so-called “unmappable VT”) techniques exist for ablation without induction of VT as described below.

c) Pace-mapping:

A pace map is a comparison of the 12 lead ECG produced during ventricular endocardial stimulation, with the surface 12 lead ECG morphology of the targeted VT. Pace mapping is based on the principle that stimulation at the site of origin will mimic the electrocardiographic pattern of VT. A perfect pace map will replicate the VT QRS morphology in all 12 surface ECG leads (12/12 match) (see figure 7). As the majority of VTs exit at the scar border, pace-mapping in a systematic fashion at the border can be useful, particularly when also looking for areas of slow or abnormal conduction e.g. late potentials (18). 12/12 match with a short stimulus to QRS interval can identify exit sites. Typically the best sites are those with 12/12 match with long stim-QRS, denoting areas of slow conduction close to exit. Pacing however can be limited by noise when the stimulation output is too high – this is often needed to capture myocardial scar, leading to artefact. Furthermore, lines of functional block may occur during VT that do not exist during sinus rhythm which will affect the degree of matching between the clinical VT and paced QRS morphology (19).

d) Substrate mapping using 3D mapping systems

The development of 3 dimensional non fluoroscopic mapping systems has permitted the ability to log electroanatomical data of both the endocardial and epicardial layers of the heart. By logging voltage amplitudes, scar and scar border can be determined anatomically and targeted. One system (CARTO magnetic mapping system (Biosense, Inc., Diamond Bar, California)) uses electromagnetic fields emanating from coils beneath the patient that are measured from a location sensor embedded in the tip of a roving catheter. This allows a three-dimensional reconstruction of the chamber of interest. These can be colour-coded to display maps of myocardial voltage or electrical activation through the myocardium (20).

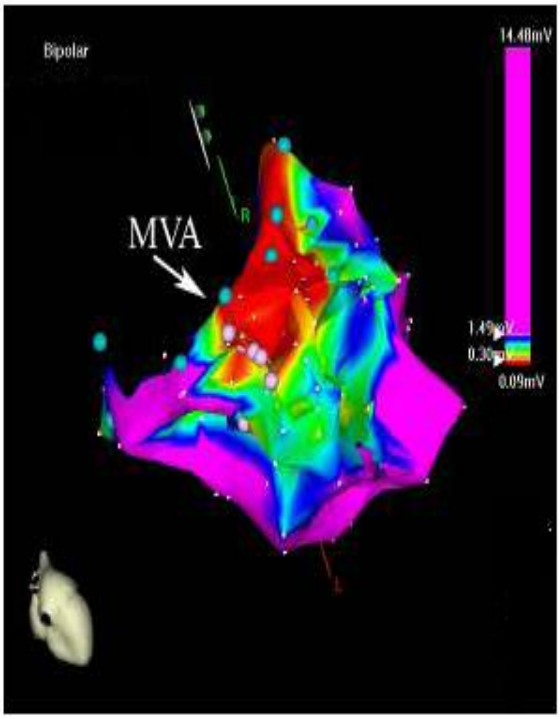



Figure 10: CARTO electro-anatomical voltage map of the endocardial LV surface during sinus rhythm. By convention, low amplitude electrogram signals <0.5 mV reflect “dense scar” – these are represented by one colour (red). Signals between 0.5mv and 1.5 mV indicate scar border zone – these are typically represented by a spectrum of colours that change with small increments (yellow to blue). Healthy tissue is defined as signals >1.5 mV – represented in pink.

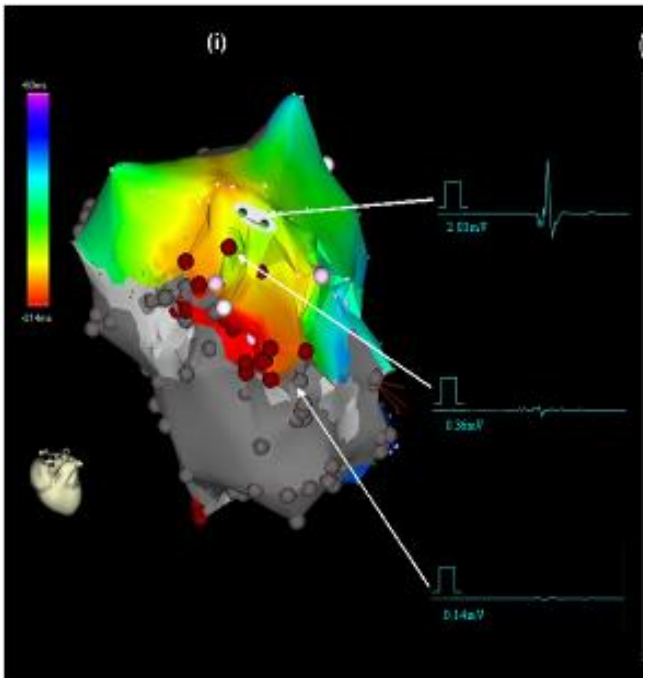


Figure 11: Activation map of the left ventricle in VT taken from a superior view. Colours are assigned against their activation time in relation to a reference electrode. Signals are taken from across scar border - from healthy tissue (top) to scar border (middle) to scar (bottom). In this example, red is the earliest signal, and travels down and away to orange, yellow and green as the activation become progressively later.

In theory, all channels lie within the scar and their entrance and exit sites reside in border zone tissue. Many different approaches to substrate guided ablation have been proposed. Multiple ablation lesions throughout the scar (scar homogenisation) theoretically eliminate all channels (21). Alternatively, all entrance and exit points could be targeted by ablating along the scar border zone (22). Both strategies risk damage to viable tissue, and creation of further scar and channels if the ablation lesions are incomplete. More targeted approaches have include ablating along lines where pace mapping approximated the QRS morphology of VT between scar border-zone and dense scar (23, 24), or targeting channels of relatively higher voltage within dense scar using a tiered decreasing-voltage scar definition (25); or targeting viable areas bordered by electrically unexcitable scar (26).

Abnormal electrogram signals have also been targeted via various approaches. Areas of late potentials within scar can be highlighted as a focussed late potential map and targeted (27). Alternatively all local abnormal ventricular activities (LAVAs) can be eliminated though this may be less specific for conducting channels within the scar (13). Whereas VT non inducibility with programmed ventricular stimulation is the accepted endpoint for most procedures, both these techniques present a new endpoint of complete signal elimination within the scar. A more focussed approach includes targeting the electrogram with the shortest delay between the far-field ventricular component and the delayed local fractionated component suggestive of an entrance into a channel (28).

Finally, sites where multiple QRS morphologies are seen with pacing within the scar can be a surrogate for channels with multiple exit sites. Ablation at these sites has been performed when pacing has induced VT (29). Table 1 summaries some of the published substrate ablation approaches that have employed for scar related VT substrate ablation.

| ***Target*** | ***Ablation strategy*** | ***Endpoint*** |
|---|---|---|
| Complete Scar | Ablation homogenisation of all scar seen on 3D mapping system | Non-inducibility with VT stim |
| Scar border-zone | Circumferential scar isolation | Scar isolation |
| Scar border-zone areas with excellent pace-maps | Ablation lines from borderzone to dense scar | Non-inducibility with VT stim |
| Tiered voltage defined | Lines | Non-inducibility with VT stim |

| | | |
|---|---|---|
| conducting channels | | Elimination of voltage channel seen on 3D mapping system |
| Viable tissue between electrically unexcitable scar | Lines | Non-inducibility with VT stim |
| LAVA | Point by point ablation | Elimination of LAVA<br><br>Non-inducibility with VT stim |
| Late potential | Islands of late potentials | Late potential abolition<br><br>Non-inducibility with VT stim |
| Scar de-channeling | Electrograms with the shortest delay between the far-field ventricular component and the earliest delayed fractionated local component (in sinus rhythm) | Non-inducibility with VT stim<br><br>Channel elimination |
| Critical central isthmus location | Sites where multiple QRS morphologies are seen with pacing within the scar. Associated pacing induced VT | Non-inducibility with VT stim |

Table 1 Proposed strategies for substrate-guided VT ablation

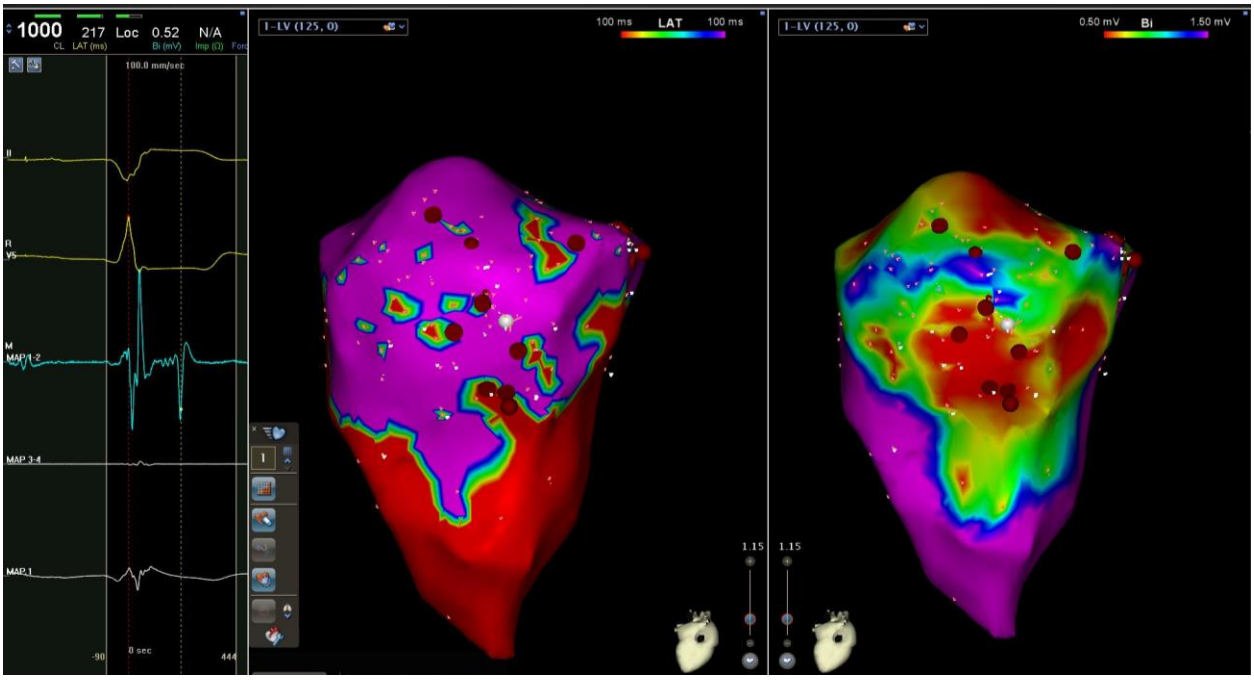


Figure 12: CARTO3 electro-anatomic map during an ischaemic VT ablation. Electrogram signals have been collected within the LV during sinus rhythm. The image on the right shows bipolar voltage map set at standard scar setting (0.5-1.5mV). Scar core is seen in red and border-zone in yellow/blue/green. The image on the left shows how a manual annotation of the latest and clearest signal on the mapping catheter is recorded for each electrogram signal collected. The image in the middle is a local activation time map where all latest signals recorded >100ms after the surface reference are represented in purple, forming a late potential map. The electrogram signal of the white tag seen in both maps demonstrates a clear late potential signal. Multiple late potential signals where seen in this area which also co-localised to the junction with scar/borderzone tissue. Ablation tags (red) are seen in the map targeting late potentials in this area during the case. As the case progressed with multiple further ablation lesions (not shown) targeting late potential signals, VT became non-inducible with programmed stimulation.

The ideal strategy to ablate scar-related post-infarct VT may well turn out to be a multipronged approach incorporating all of the above strategies. The optimal order of these steps and the precise endpoints for the procedure remain to be determined.

Another 3D mapping approach includes the non-contact mapping system (ESI 3000/St. Jude) that uses a multi-electrode (64-pole) array placed in the ventricle that can collect electrical data simultaneously within a single beat of tachycardia rhythm. When activation is plotted on a virtual 3D LV geometry, it has been used to locate earliest diastolic activity (27). In sinus rhythm the location of the slowest conduction in the region of latest endocardial activation correlates with clinical VT maps in locating a channel, and thus can be used to predict channel location in the absence of VT (28).

**Current Problems with VT ablation:**

Scar related VT ablation is technically challenging, not without risk and is not 100% effective. Ablation in VT is too infrequent as the majority of clinical VTs are poorly tolerated. The biggest prospective study to date assessing the effectiveness of post infarct VT ablation is the multi-centre “Thermocool Ventricular Tachycardia Ablation Trial (3).” This enrolled 231 patients with scar related VT (median of 11 episodes in preceding 6 months). During the 6-month follow-up period, 49% of patients were free of VT at 6 months. The frequency of VT was reduced by >75% in 67% of patients. Procedure mortality was 3% - in 6 out these 7 cases, this was related to peri-procedural uncontrollable VT and progressive haemodynamic decompensation as opposed to the procedure itself.

ICD shocks are painful and psychologically distressing such that the mortality benefit is offset by the significant morbidity caused by the device (32). In addition, it does not offer complete protection from death from arrhythmia (33). SMASH VT examined whether prophylactic substrate ablation in patients with secondary prevention ICD’s (off anti-arrhythmic agents) reduced the incidence of future ICD therapies (34). In this randomized trial, 128 patients were followed up for just under 2 years. Peri-procedural mortality was 0%. Over a 2 year follow up

interval, VT ablation resulted in a 65% reduction in the risk of future ICD therapy (73% reduction for ICD shock therapy) compared to conventional therapy.

Who is an ideal candidate for a VT ablation? What is the best mapping and ablation strategy? What percentage success and complication rates should be explained to the patients? Clearly, in patients with structural heart disease the optimal approach to ablation is still developing, and there remain many challenges and unanswered questions (35).

## Conclusion

VT ablation in the context of ischaemic heart disease and post-infarction is a complex arrhythmic problem. Whilst the mechanism for any single VT appears to be relatively well understood, the substrate is complex and the propensity for recurrence is high. It remains a useful adjunct to ICD therapy but requires better tools to identify potential isthmuses and robust elimination of these areas to prevent recurrence.

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