Effect of activation wavefront on electrogram characteristics during ventricular tachycardia ablation

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Background
Catheter ablation of ventricular tachycardia (VT) in structural heart disease is challenging due to non-inducibility or haemodynamic compromise. Ablation often depends on elimination of local abnormal ventricular activities (LAVAs), but which may be hidden in farfield signal. We investigated whether altering activation wavefront affects activation timing and LAVA characterization and allows better understanding of isthmus anatomy.

Methods
Patients with ischaemic cardiomyopathy underwent mapping using the ultra-high density system. Maps were generated for all stable VTs, and with pacing from the atrium, RV apex and an LV branch of the coronary sinus.

Results
56 paced maps and 23 VT circuits were mapped in 22 patients. In 79\% of activation maps, there was =1 line of block in the paced conduction wavefront, with 93\% having fixed block and 32\% showing functional partial block.

Bipolar scar was larger with atrial than RV (\( ? 4.5 \pm 3.8 \text{ mm2}, p<0.01\)) or LV pacing (\( ? 5.15 \pm 5.4 \text{ mm2}, p=0.01\)); LAVA areas were smaller with atrial than RV (\( ? 6.3 \pm 1.9 \text{ mm2}, p<0.01\)) or LV pacing (\( ? 4.9 \pm 1.7 \text{ mm2}, p<0.01\)). LAVA areas were larger with wavefront propagation perpendicular vs parallel to the line of block along isthmus boundaries (\( 10.3 \pm 7.1 \text{ vs } 13.6 \pm 7.4 \text{ mm2}, p = 0.01\)).

All patients had successful VT isthmus ablation. In 11 ± 8 months follow-up, 2 patients had recurrence.

Conclusions
Wavefronts of conduction slowing/block may aid identification of critical isthmuses in unmappable VTs. Altering the activation wavefront leads to significant differences in conduction properties of myocardial tissue, along with scar and LAVA characterization. In patients where few LAVAs are identified during substrate mapping, using an alternate activation wavefront running perpendicular to the VT isthmus may increase sensitivity to detect arrhythmogenic substrate and critical sites for re-entry.

Figure

(A) VT activation map with a double loop re-entrant circuit and critical isthmus on the anterior wall. Black dots = lines of block. Bipolar EGMs shown from Orion catheter at points 1-4.
(B) Activation & (E) voltage map with atrial pacing. (F) Activation & (I) voltage map with RV apical pacing. White dots = lines of block. Black dots = isthmus projected from VT activation map. White stars = LAVAs. EGMs are shown from inside (C,G) & outside (D, H) the isthmus. The scar maps (E, I) are similar except a small area of more viable tissue at the thinnest point of the isthmus.

(B) demonstrates a region of late activation which co-locates with the VT isthmus. (F) is similar; however, with this direction of activation coming more perpendicularly to the isthmus of the VT circuit, a channel of earlier activating myocardium is visible running through the region of late activation, which co-locates with the isthmus. In (B, C) there is a low voltage signal but no LAVAs, whereas at the same point in (F), a fractionated signal with LAVAs is present (G).