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Multicenter Study of Ischemic Ventricular Tachycardia Ablation With Decrement Evoked Potential (DEEP) Mapping With Extra Stimulus

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ABSTRACT

OBJECTIVES The authors conducted a multicenter study of decrement-evoked potential (DEEP) – based functional ventricular tachycardia (VT) substrate modification to evaluate if such a mechanistic and physiological strategy is feasible and efficient in clinical practice and provides reduction in the VT burden.

BACKGROUND Only a fraction of the myocardium targeted in current VT substrate modification procedures is involved in the initiation and perpetuation of VT. The physiological basis of the DEEP strategy for identification of areas of initiation and maintenance of VT was recently established.

METHODS We included 20 consecutive patients with ischemic cardiomyopathy. During substrate mapping, fractionated and late potentials (LPs) were tagged, and an extra stimulus was performed to determine which LPs displayed decrement (DEEPs). All patients underwent DEEP-focused ablation: elimination of DEEP + further radiofrequency (RF) if VT was still inducible. Patients were followed during 6 months.

RESULTS Patients were predominantly male (95%), and their mean age ± SD was 64.6 ± 17.1 years. Mean ± SD left ventricular ejection fraction was 33.4 ± 11.4%. Mean ± SD ablation time was 30.6 ± 20.4 min. Specificity of DEEPs to detect the isthmus of VT was better than that of LPs (0.97 [95% confidence interval [CI]: 0.95 to 0.98] vs. 0.82 [95% CI: 0.73 to 0.89]), without significant differences in terms of sensitivity (0.61 [95% CI: 0.52 to 0.69] vs. 0.60 [95% CI: 0.44 to 0.74], respectively). Fifteen of 20 (75%) patients were free of any VT after DEEP-RF at 6 months of follow-up and there was a strong reduction in VT burden compared to 6 months pre-ablation.

CONCLUSIONS In a multicenter prospective study, DEEP substrate mapping identified the functional substrate critical to the VT circuit with high specificity. DEEP-guided VT ablation, by its physiological nature, may enable greater access to focused ablation therapy for patients requiring VT treatment. (J Am Coll Cardiol EP 2018;■:■–■)

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All authors attest they are in compliance with human studies committees and animal welfare regulations of the authors' institutions and Food and Drug Administration guidelines, including patient consent where appropriate. For more information, visit the *JACC: Clinical Electrophysiology* author instructions page.

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ABBREVIATIONS AND ACRONYMS

DEEP = decrement-evoked potential

EGMs = electrograms

ICD = implantable cardioverter defibrillator

ICM = ischemic cardiomyopathy

LAT = local activation time

LP = late potentials

NICM = nonischemic cardiomyopathy

RF = radiofrequency

S1 = stimulus 1

S2 = stimulus 2

VERP = ventricular effective refractory period

VT = ventricular tachycardia

Contemporary substrate-based approaches for ventricular tachycardia (VT) ablation targets abnormal potentials and late potentials, and can result in large areas of viable myocardium being ablated that may or may not be linked to the mechanisms of initiation and/or maintenance of VT (1,2). An alternative ablation strategy would be to identify which of those potentials actually participate in the initiation and maintenance of VT as targets for VT ablation. We have recently shown which of these many abnormal and late potentials actually initiate and/or maintain VT. In our previous study, regions that displayed decremental behavior evoked during right ventricular (RV) pacing with extra stimuli (decrement-evoked potential; DEEP), colocalized with the regions of the initiation and diastolic circuit of VT more accurately than those areas displaying nondecremental late potentials (LPs) (3).

This mechanistic physiological demonstration, where decrement precedes unidirectional block, allowed us to identify the region of diastolic path of VT by delivering extra stimulus and evoking the delay of the electrogram (EGM) so that the hidden mechanistic substrate could manifest itself. This obviates the need for VT induction for identifying the critical component of the circuit. This physiological substrate deduction comes from patients who underwent intraoperative global mapping of substrate and activation of VT including the moment of initiation of VT. That study provided the mechanistic basis of DEEP in substrate ablation of VT, allowing VT ablators to identify regions that are responsible for the mechanism of VT without inducing VT. However, that study did not test the practical use and feasibility in the catheterization laboratory. More importantly, it was not established whether DEEP-guided substrate ablation could be adapted to contemporary clinical practice and could be implemented by other operators and sequential mapping systems.

To address these issues, as a follow-up to our original mechanistic study, we designed a multicenter prospective observational study to: 1) establish if the methodology for DEEP mapping with extra stimulus to identify mechanistic substrate is implementable using contemporary 3-dimensional (3D) electroanatomic mapping systems in the catheterization laboratory; and 2) describe the initial results of a multicenter DEEP-guided ablation strategy for reducing VT burden using this focused ablation strategy in the ischemic substrate.

METHODS

PATIENTS. Consecutive patients with ischemic cardiomyopathy (ICM) and recurrent episodes of VT despite medical therapy listed for VT ablation at 4 different institutions (Toronto General Hospital, Ontario, Canada; John Hunter Hospital and Lake Macquarie Private Hospital, Newcastle, Australia; and Århus University Hospital, Skejby, Denmark) were evaluated for participation in the study. The protocol of the study was reviewed and approved by the research ethics boards at all institutions and complies with the Declaration of Helsinki; all patients provided informed consent. All patients underwent DEEP mapping (n = 20) and the ablation strategy focused on eliminating DEEP sites and perform further radiofrequency (RF) if VT was still inducible.

DEEP MAPPING. Substrate maps were created in sinus rhythm or during RV pacing. Access to the left ventricular endocardial surface was achieved with a retroaortic (n = 13) or transseptal approach (n = 7). In 16 patients, multielectrode catheters were used for substrate mapping (Decanav in 9 patients, Pentarray in 6 patients [Biosense Webster, Diamond Bar, California], and Orion in 1 patient [Boston Scientific, Marlborough, Massachusetts]). The rest of patients (n = 4) underwent mapping with a 3.5-mm irrigated tip ablation catheter (Thermocool SF catheter, Biosense Webster). LPs (potentials with complex high frequency or multicomponent after or at the QRS offset either present in sinus rhythm or seen during RV pacing) were identified and annotated either as location tags or as local activation time (LAT) maps by manually annotating onset of the delayed bipolar EGM.

For all the points showing LPs, a systematic assessment for local decrement was performed with a drive train (S1) from the RV at 600 ms with a single extra stimulus (S2, coupled at 20 ms above the ventricular effective refractory period [VERP] VERP). If the difference in the time interval measured from surface ventricular far field signal onset to the local LP bipolar EGM during the S1 drive compared to the same interval measured immediately after the S2 was >10 ms, the LP was defined as a DEEP (**Figure 1**). The same strategy was used for multicomponent EGMs from which DEEP were identified if their components split by >10 ms after S2. All DEEP and non-DEEP-LPs (hereafter referred as LPs) were given a different annotation marker in the substrate map. Care was taken to map the areas of interest with the same density of points to avoid over-representation of the DEEP or LP areas. Analysis of areas of DEEPs and LPs could also be performed by creating an LAT map