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Please note: Drs. Guasch and Benito contributed equally to this work.

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The Challenge of Voltage Channels

We read with great interest the report by Mountantonakis et al. (1). In this study, the authors reported that ventricular tachycardia (VT) isthmus sites were contained within channels in only 37% of voltage channels, and raised concerns about the suitability of using such channels as a target for ablation of unmappable VT. The authors came to this conclusion after analyzing a subgroup of 24 patients from a group of 140 patients who underwent VT ablation. These findings differ from previous data in which the majority of channels were related with clinical or inducible VTs (2,3).

Could evidence derived from 17% of patients with monomorphic VT be applied to the great majority of VT patients? In our opinion, great care should be exercised in extrapolating these results to the full spectrum of VT because of these points:

1. Complete activation mapping during VT was not obtained, and the circuit exit sites were not identified. Therefore, a connection between the isthmus site and a close channel cannot be completely excluded.
2. VT isthmuses not related to conduction channels are shown in maps in which the lower voltage limit is set at 0.5 mV. Could it be that isthmus sites were in incomplete channels connected to the main channel, as it seems to occur in Figure 5A? In previous studies, VT-related channels were always located within dense scar defined by a voltage <0.5 mV (2).
3. Voltage channels were identified during sinus rhythm and VT isthmus sites during VT. How these sites were later incorporated into voltage maps is very relevant, taking into account that ventricular volume and spatial position of the left ventricle may change during VT.
4. The average cycle length of channel-related VT was significantly shorter than that of VT not related to channels (377 ± 67 ms vs. 440 ± 40 ms, p = 0.01), but similar to the VT cycle length (374 ± 59 ms) reported in the previous study in which the majority of VT isthmuses were in channels (2). Could it be that fast VT isthmuses are commonly located in voltage channels? Slow VT may have a more complex substrate in which differentiation of central isthmuses from the surrounding scar could be more challenging. If slow mappable VTs have a different substrate, studies based on entrainment mapping could introduce bias when studying the relationship of VT isthmuses and voltage channels.

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Reply

The Challenge of Voltage Channels

We would like to thank Drs. Ávila and Arenal for their comments and interest in our work (1). The demonstration of voltage "channels" during ablation of ventricular tachycardia (VT) was originally proposed in the seminal studies by Arenal et al. (2) and Hsia et al. (3); we do believe this finding continues to be of importance in the field of VT ablation. However, some have adopted these findings to the degree that "empiric" ablation of voltage channels is performed as part of a VT ablation procedure,

and we have noted several limitations to the technique. First, the presence of channels is highly dependent on the density and quality of points taken on the electroanatomic map. Even on Arenal's original publication (2), the density of points in Figures 3 and 5 is high in the region of the identified channel, but sparse in other areas of the left ventricle. It is possible that if a more uniform density of points was obtained, other channels may have been identified that were not related to the clinical or induced VTs. Dr. Arenal and colleagues were also careful to perform pacing in these channels and document a prolonged stimulus to QRS duration suggestive of slow conduction. We believe this finding heightens the significance of an anatomic channel, similar to the identification of late potentials within channels in our paper. We sought to obtain the true sensitivity and specificity of such identified channels in a population of patients with tolerated VT and detailed voltage maps. Although we found that the association of such channels with the clinical VT was much lower than those identified in Arenal et al.'s paper, we did find that channels associated with late potentials were specific (81%) for identification of the VT isthmus, similar to Arenal et al. (2) findings of 75%. The difference between the upper and lower voltage cutoff in the Arenal et al. (2) paper was quite small (0.01 mV)—in our experience, this may accentuate the identification of channels that are not clinically relevant when applied uniformly.

To specifically address the 4 points raised by Drs. Ávila and Arenal:

1. It is true that complete activation maps during VT were not obtained in our study (nor Arenal et al. [2]) as this can be quite time consuming and technically challenging during clinical VT ablation. The voltage maps we obtained were extremely detailed, and we chose only patients with tolerated VT so we could be sure to identify the true VT isthmus using entrainment criteria, which is the gold standard target for ablation. Therefore, we feel that channels remote from any identified VT isthmus could not represent a channel for the clinically relevant VT.
2. The observation that channels typically appeared at lower cutoffs <0.5mV in the Arenal et al. (2) study is interesting. After lowering the upper cutoff, we progressively lowered the lower cutoff down to 0.01 mV and still did not find any channels corresponding to the VT isthmus in the patient depicted in Figure 5 in our paper.
3. The criticism that the spatial location of a point on an electroanatomic map during VT may be different than the same point obtained during sinus rhythm is valid. However, as stated in the Methods section of our paper, "Once the critical isthmus was identified, radiofrequency ablation was performed at the isthmus site, and upon termination of the tachycardia, a new point was tagged in sinus rhythm." Therefore, all the isthmus points noted on the electroanatomic maps in our study were obtained during sinus rhythm *after* VT termination, and any change in left ventricular volume or catheter location during VT would not affect the relationship between the VT isthmus and the identified voltage map channels.
4. The authors note that in our study, VTs associated with channels were typically faster than those not associated with channels. The authors hypothesize that slower VTs may have a more complex substrate that makes identification of channels on an electroanatomic voltage map more difficult. Although it is certainly possible that slower VTs have a different mechanism and/or substrate than faster VTs, the literature on VT mechanism is based in large part on slow, tolerated VTs; there are no data we know of to suggest that slower VTs would have a different mechanism or substrate.

In summary, we find that our data provide strong evidence that the mere presence of a voltage channel demonstrated during electroanatomic mapping is not specific for a clinical VT isthmus. Additional electrophysiological surrogates such as late potentials, as described in our paper, or a long stimulus to QRS duration, as described by Dr. Arenal and colleagues, should be obtained before such channels are considered important for maintaining VT.

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