the VT noninducibility as a surrogate marker of success and makes an attempt to establish an alternative procedural endpoint (3). Thus, it provides the long awaited proof that the scar inhomogeneity should be the essential target for ablation.

However, we have the feeling that both endocardial and epicardial ablation of all abnormal signals could be more beneficial even though the studied cohort consisted of patients with ischemic heart disease. Unfortunately, epicardial ablation was performed only in a minority of the patients in the substrate-based ablation group. The possible benefit of combined endocardial and epicardial ablation of abnormal signals should be investigated in future trials. On the other side, even though the overall procedure duration between both groups was comparable, the mean radiofrequency time was expectedly longer in the substrate-ablation group, which raises concerns about myocardial stunning, fluid overload, and hemodynamic deterioration that can blunt the benefits of an achieved electrical stability.

Our biggest concern is the definition for "abnormal potentials" that gathers under the same roof local abnormal ventricular activities, double, split, late, and fragmented potentials, which certainly do not bear the same clinical relevance. We are worried that a nonselective ablation of all abnormal potentials such as the split but high-amplitude signals in the border zone may be associated with collateral damage due to expansion of necrosis to the adjacent viable myocardium. Therefore, we would like to appeal for a careful and contemplative implementation of this strategy in the clinical routine until more data about the safety of the scar homogenization appear.

*Julia Anna Lurz, MD
Arash Arya, MD
Gerhard Hindricks, MD
Borislav Dinov, MD

*Department of Electrophysiology
University of Leipzig - Heart Center
Struempelstrasse 39
04289 Leipzig
Germany
E-mail: julia.koch@me.com

http://dx.doi.org/10.1016/j.jacc.2016.03.606

Please note: Dr. Hindricks has received research grants from St. Jude Medical and Boston Scientific. The other authors have reported that they have no relationships relevant to the contents of this paper to disclose.

REFERENCES

1. Di Biase L, Burkhardt JD, Lakkireddy D, et al. Ablation of Stable VTs Versus Substrate Ablation in Ischemic Cardiomyopathy. J Am Coll Cardiol 2015;66:2872–82.

2. Di Biase L, Santangeli P, Burkhardt DJ, et al. Endo-epicardial homogenization of the scar versus limited substrate ablation for the treatment of electrical storms in patients with ischemic cardiomyopathy. J Am Coll Cardiol 2012;60:132–41.

3. Dinov B, Arya A, Schratter A, Schirripa V, et al. Catheter ablation of ventricular tachycardia and mortality in patients with nonischemic dilated cardiomyopathy: can noninducibility after ablation be a predictor for reduced mortality? Circ Arrhythm Electrophysiol 2015;8:598–605.

REPLY: A Prima Vista Ablation of Ventricular Tachycardia

Should We Abandon the Mapping of VT?

We read with interest the letter by Dr. Lurz and colleagues in regards to the VISTA (Ablation of Clinical Ventricular Tachycardia Versus Addition of Substrate Ablation on the Long Term Success Rate of VT Ablation) trial (1). Surviving myofibers within the scar tissue represent the most known arrhythmogenic substrate of post-infarction ventricular tachycardias (VT) (2). The identification of these myocytes and myofibers is achieved by three-dimensional voltage systems via a mapping catheter that identifies abnormal electrograms within the scar (3). Whether this can be better achieved in VT or in sinus rhythm has been one of the hypotheses generating the VISTA trial (1). Most VTs are not well tolerated by the patients requiring ablation in sinus rhythm. Lurz and colleagues are concerned about a possible damage to the heart performance with extensive scar "homogenization." However, as reported in the trial (1), we observed a trend toward improvement in left ventricular ejection fraction and New York Heart Association functional class at 1-year follow-up in the substrate-based group. Left ventricular ejection fraction improvement from baseline was higher in the substrate group (2.4 $\pm$ 6.6% vs. 2.1 $\pm$ 5.8%, respectively; $p = 0.13$) as well as the New York Heart Association functional class improvement (1). "Scar homogeneization" achieve better freedom from VT at follow-up. We did not observe myocardial stunning or fluid overload. Therefore, the authors' concerns about extensive ablation in the scar are without clinical evidence. We also disagree about their concern on the definition of "abnormal potentials." This is well characterized in the literature and there is nothing "magic" about it (3,4). In addition, high amplitude delayed and complex electrograms can be found in the border zone. In this respect, most of the present series on ischemic cardiomyopathy, including one from the Leipzig group (5), concentrate their lesions along the scar border.

In regards to the need of endo-epicardial homogeneization of the scar, we would like to clarify that

the 2012 paper excluded patients with coronary artery bypass graft (CABG) and showed that the need of epicardial ablation in patients without CABG appeared higher than previously reported. In the randomized trial, patients with CABG were included and the possibility of epicardial ablation was lower.

To conclude, although we consider the authors' appeal of interest, we would like to reinforce the concept that no data in regards to their concern are available and that the VISTA randomized trial confirms the validity of substrate-based ablation as a better way to perform ischemic VT ablation. We can certainly say that the present one is safe and efficacious and therefore valid not only "A PRIMA VISTA."

Luigi Di Biase, MD, PhD
J. David Burkhardt, MD
Dhanunjaya Lakkireddy, MD
Corrado Carbucicchio, MD
Sanghamitra Mohanty, MD
Prasant Mohanty, MBBS, MPH
Chintan Trivedi, MD, MPH
Pasquale Santangeli, MD
Rong Bai, MD
Giovanni Forleo, MD
Rodney Horton, MD
Shane Bailey, MD
Javier Sanchez, MD
Amin Al-Ahmad, MD
Patrick Hranitzky, MD
G. Joseph Gallinghouse, MD
Gemma Pelargonio, MD
Richard H. Hongo, MD
Salwa Beheiry, RN
Steven C. Hao, MD
Madhu Reddy, MD
Antonio Rossillo, MD
Sakis Themistoclakis, MD
Antonio Dello Russo, MD
Michela Casella, MD
Claudio Tondo, MD
***Andrea Natale, MD**

*Texas Cardiac Arrhythmia Institute
3000 North I-35
Suite 720
Austin, Texas 78705
E-mail: dr.natale@gmail.com

http://dx.doi.org/10.1016/j.jacc.2016.05.052

Please note: Dr. Di Biase is a consultant for Biosense Webster, Stereotaxis and St Jude Medical; he received speaker honoraria/travel costs from Medtronic, Boston Scientific, EPiEP Janssen, Pfizer, and Biotronik. Dr. Natale received speaker honorariums from Boston Scientific, Biosense Webster, St. Jude Medical, Biotronik, Medtronic, and Life Watch; she is a consultant for Biosense Webster, St. Jude Medical, and Janssen. Dr. Burkhardt is a consultant for Biosense Webster and Stereotaxis. All other authors have reported that they have no relationships relevant to the contents of this paper to disclose.

REFERENCES

1. Di Biase L, Burkhardt JD, Lakkireddy D, Carbucicchio C, Mohanty S, Mohanty P, et al. Ablation of stable VTs versus substrate ablation in ischemic cardiomyopathy: the VISTA randomized multicenter trial. J Am Coll Cardiol 2015;66:2872–82.

2. de Bakker JM, van Capelle FJ, Janse MJ, et al. Slow conduction in the infarcted human heart. 'Zigzag' course of activation. Circulation 1993;88:915–26.

3. Gardner PI, Ursell PC, Fenoglio JJ Jr., Wit AL. Electrophysiologic and anatomic basis for fractionated electrograms recorded from healed myocardial infarcts. Circulation 1985;72:596–611.

4. Fenoglio JJ Jr., Pham TD, Harken AH, Horowitz LN, Josephson ME, Wit AL. Recurrent sustained ventricular tachycardia: structure and ultrastructure of subendocardial regions in which tachycardia originates. Circulation 1983;68:518–33.

5. Arya A, Eitel C, Bollmann A, Wetzel U, Sommer P, Gaspar T, et al. Catheter ablation of scar-related ventricular tachycardia in patients with electrical storm using remote magnetic catheter navigation. Pacing Clin Electrophysiol 2010;33:1312–8.