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Editorial

Substrate ablation for post-infarct and Brugada storm: Triggering the calm

Electrical storm (ES) represents the most pressing emergency situation confronted by electrophysiologists and one of the most difficult to manage. Although technically defined as a situation of recurrent ventricular arrhythmias requiring three or more appropriate electrical cardioversions in a given 24 h period, there is significant heterogeneity among different patients who fit this definition. Clearly a patient with slower post-myocardial infarct ventricular tachycardia (VT) who receives three appropriate shocks from an implantable cardioverter-defibrillator (ICD), each several hours apart, represents a completely different clinical problem to a patient with Brugada syndrome who sustains 35 ICD shocks for recurrent rapid pleomorphic VT and ventricular fibrillation (VF).

Regardless of this heterogeneity however, there are two themes underlying all ES: 1) a vulnerable myocardial and autonomic substrate, and 2) myocardial and autonomic triggers.[1–3] All management options for dealing with ES address one or both of these factors. After diagnosing and treating reversible factors such as electrolyte dyscrasias, therapy general commences with beta blockade followed by membrane-active antiarrhythmic drugs and then general anaesthesia with intubation. If ECG monitoring discloses a recurrent monomorphic triggering PVC initiating each salvo of VT/VF, then electrophysiologic mapping and catheter ablation of this triggering focus is usually considered early. If not, then neuraxial modulation with thoracic epidural anaesthesia, percutaneous stellate ganglion block, renal denervation and bilateral cardiac sympathetic denervation can all be potentially instituted. Mechanical circulatory support represents the last line option in refractory cases.

The majority of these therapeutic options are directed towards the triggers for ES, including when catheter ablation is directed at a triggering premature ventricular complex (PVC). However, catheter ablation can also be used to address the vulnerable myocardial substrate,[4] and this may be of particular importance when triggering PVCs are either inapparent or unmappable.

In this issue of the *Journal*, this concept is explored separately in what at first blush may appear to be two entirely different disease states, namely post-infarct cardiomyopathy and Brugada syndrome. On the one hand, ischemic cardiomyopathy is characterized by advanced structural changes, a subendocardial substrate location and a predilection for monomorphic VT. On the other, the substrate in Brugada syndrome is ultrastructural with preserved ventricular function, it is located on the epicardium of the right ventricular (RV) infundibulum and free wall,[5] and VT is usually polymorphic (although around 4% is monomorphic[6]). However, in the context of ES, significant similarities begin to emerge between them.

Firstly Rao et al[7] describe their experience with 12 consecutive patients with post-infarct ES. They treated genuinely unstable ES patients with a median of 9 ICD shocks or 23 external shocks delivered. Appropriate escalation of therapy occurred in this series with 8 patients undergoing catheter ablation and two additionally having stellate ganglionectomy. A comprehensive approach to electrogram mapping and substrate ablation in addition to the totality of care delivered resulted in an impressive 83% survival to hospital discharge. Two patients experienced isolated late VT recurrence but no patient developed recurrent storm.

Also in this issue, Shelke et al[8] present a series of 5 patients with Brugada syndrome and drug-refractory ES. Three of these patients appeared to have triggering PVCs on ICD electrograms but no ectopy was seen or induced in the EP lab and hence only substrate ablation was possible. This was performed predominantly on the epicardium and with the assistance of procainamide to unmask as much substrate as possible. Again, the majority of these patients did extremely well over medium term follow up, and only one required an appropriate shock for a VF episode at 2 years follow up.

No significant complications of catheter ablation occurred in either study. While extensive endocardial ablation in a post-infarct scar may carry some risk, the nature and magnitude of the risk is entirely different in Brugada syndrome. Percutaneous pericardial access for epicardial mapping and ablation has become routine in many centres but still carries a greater potential for misadventure than entirely endocardial ablation. A particular consideration in Brugada syndrome that has received scant attention in the literature is the relationship of the RV epicardial coronary arteries to the common substrate locations. In a recent published case, sizeable conus and RV marginal branches precluded safe elimination of the entirety of the substrate, although enough ablation had been performed to abolish the Brugada ECG pattern on ajmaline challenge.[9] Further systematic examination of this question is urgently warranted given the greater exposure of increasingly less symptomatic, non-ES patients to epicardial substrate ablation in Brugada syndrome.[10] This is particularly important in this context as the substrate in this disease is ultrastructural and electrical, and occurs in myocardium with preserved contractile function where coronary

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occlusion would be proportionately more detrimental than in cases of scar-related epicardial right ventricular VT ablation. There is also the theoretical proarrhythmic risk associated with damage to the conus artery.

Both the Rao et al[7] and Shelke et al[8] studies are commendable for the diligence of the investigators in characterizing, mapping and eliminating the vulnerable substrate underlying ES in their post-infarct and Brugada syndrome patients respectively. Despite the vast differences in that substrate between the two conditions, it is clear that it plays an enormous role in ES arrhythmogenesis, and targeting it is associated with elimination of recurrent ES in both disease states. As in prior experience with ES ablation, arrhythmic recurrences in these patients are generally with isolated episodes of VT rather than frank storm. While these results are interesting, it should be remembered that these and indeed all published studies regarding the ablative management of ES are observational. The largest of these is the multicentre collaboration published by Vergara et al[11] in which they describe the outcomes of 677 ES patients taken from a pool of 1940 VT ablation patients. The ES patients were older and had more advanced structural heart disease compared to the non-storm patients. Successful ablation was associated with a higher survival rate compared to those patients who were still inducible at the end of the procedure.

The results of catheter ablation presented in this issue of the *Journal* continue to solidify our mechanistic understanding of the importance of the underlying substrate in the genesis of ES and guide our present management strategies. However, demonstration of the efficacy of early substrate ablation in ES in different disease states will ultimately require prospective randomized trials.

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