



This was. . .outflow tract ventricular arrhythmia non-contact balloon guided ablation

Ablação de arritmias ventriculares do trato de saída guiada por balão de não contacto: uma perspetiva histórica

João Primo

Centro Hospitalar Vila Nova de Gaia/Espinho, Vila Nova de Gaia, Portugal

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It has been a long time since the first report of an ablation of premature ventricular contractions (PVC) arising from the outflow tract, published in 1992 by Gursoy and Brugada.¹ There has not always been agreement on the use of ablation for premature ventricular contractions as it is considered a benign condition. After Zhu et al.² reported on the successful ablation of frequent, monomorphic PVC in 10 patients in 1995, Professor Hein Wellens expressed his concern in a famous editorial comment: “I am concerned that some of our colleagues will accept this article as an invitation to approach bothersome but otherwise innocent ventricular ectopic activity by catheter ablation. That situation may develop into a therapy in search of a disease rather than the reverse!”³

However, PVCs are not always only “bothersome”, as they can occasionally cause cardiomyopathy and heart failure (HF), leading to sudden death. Dukes et al. prospectively assessed the effect of PVC frequency on the risk of incident HF and mortality in individuals >65 years, with normal left ventricular ejection fraction and no history of congestive HF. The presence and frequency of PVCs was quantified using a 24 h Holter, and patients were fol-

lowed prospectively for incident HF and death. The authors found that a PVC burden of at least 0.7% had specificity in predicting 15-year incidence of HF of >90%.⁴ In a different study, Baman et al. identified a PVC burden of 10% as being the lowest burden which could result in reversible cardiomyopathy, while a burden >25% best separated the patient population with versus without impaired LV function.⁵ Nevertheless, most monomorphic PVCs are benign, especially in patients without obvious structural heart disease on cardiac magnetic resonance imaging. For these patients, treatment is usually unnecessary in the absence of debilitating symptoms. When PVCs are symptomatic, reassuring the patient of the benign nature of their condition can often be the approach.

When treatment is necessary, few antiarrhythmic drugs (AADs) are efficient in reducing PVCs and symptoms or the progression to tachycardiomyopathy. Class III AADs should be avoided in the long term due to potential side effects. The first choice after the initial use of beta blockers are Class I AADs such as propafenone and flecainide. However, Ling et al. showed that radiofrequency ablation (RFA) of right ventricular outflow tract tachycardia PVC is superior to AADs in the reduction of PVC burden and prevention of recurrence.⁶ In the same study, the authors reported that the efficacy of ablation in patients with QS morphology in lead I was higher than those with rsr'/rsR' and qR/R/Rs pat-

E-mail address: joaojoseprimo@gmail.com

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terns, which means that ablation success is not the same for all PVC locations.⁶ Some PVC locations such as the LV summit or the papillary muscles are associated with significantly worse success rates. The former location is not accessible from the endocardium, while the latter are mobile structures where catheter stability is an issue.

In the present manuscript, Almeida et al. present the experience of a single center in non-contact multielectrode balloon catheter guided ablation of ventricular arrhythmias originating from the outflow tract. To my knowledge, this is one of the largest studies on this technique.⁷ The Ensite Array NCM system (St. Jude Medical, St. Paul, Minnesota, United States) uses a multielectrode array catheter with a 7.5 ml balloon and 64 microelectrodes. It records electrograms from more than 3000 sites simultaneously from one single premature beat to create a three-dimensional map of electrical activation.⁸

There are two main challenges when we try to ablate PVCs: The ectopic focus location and the number of PVCs during the procedure. The Ensite Array may help overcome both challenges. It has the unique feature of globally mapping electrical activation in an entire chamber requiring only a single beat, which enables us to treat symptomatic patients who are experiencing some PVCs during the procedure itself. The array also helps to distinguish the earliest activation point (EA) from the break-out point (BO). This unique feature also provides additional insights into uncommon arrhythmic foci from the pulmonary artery (PA). The EA site for PVC arising from the PA is usually located at a mean of 10.8 mm above the pulmonary valve, while the BO point is typically located below the valve. Ablation is more successful when the first lesions are delivered at the EA point (88% success rate) rather than the BO (66% success rate).⁹

One of the limitations of the Ensite Array is that it marks the earliest activation point in the endocardium of the chamber where the array is located even when the true origin of the PVC is located in the epicardium or in another chamber. In the latter cases, the unipolar signal would show a rS deflection, suggesting that the true origin was at a close yet different location. Another important limitation of this system is the difficulty in creating an accurate map when the distance between the central axis of the balloon catheter and the ectopic focus is >4.0 cm.⁷ Also, the array can be difficult to handle and stabilize inside the heart chambers, particularly in left ventricle outflow tract. It can also cause mechanical PVC, which can be confused with clinical PVC, hindering free movement of the ablation catheter.^{7,10} Often, the array needs to be repositioned or reduced in volume. The authors mentioned these and other technical limitations that contributed to the discontinuation of this system.

However, the high rate of success reported in the present study would perhaps justify continued interest in non-contact mapping. In a case report published last year, Elbey et al.¹¹ used the AcQMap mapping system and AcQMap catheter (Acutus Medical, CA, USA). This mapping catheter combines 48 ultrasound transducers responsible for reconstructing the chamber anatomy and 48 engineered electrodes. This system enables charge-density-based activation patterns to be displayed along the endocardial surface of the heart. This technology seems promising. Nowadays, there are many tools available to help map and ablate PVCs,

including **i**) the possibility of anatomic segmentation of the target chamber, valves and coronary arteries with high quality computed tomography scan images with CartoSeg; **ii**) the use of tridimensional mapping with multipolar catheters such as the Pentarray or Decanav (Biosense), **iii**) pace mapping with PasoModule; **iv**) intracardiac echocardiography to allow real-time visualization of structures such as the papillary muscles or the aortic root, or even **v**) a contact balloon, Orion (UHDM), with ultra-high definition mapping using the Rhythmia system (Boston Scientific). However, it is difficult to prove that these more sophisticated methods are superior to the conventional ones, especially at high volume centers.¹² PVC ablation is sometimes very challenging due to the anatomic complexity of the outflow tracts and the difficulty in assessing certain locations (e.g., LV summit).¹³ Retrograde coronary venous ethanol ablation is safe and feasible as a bail-out approach to failed PVC RFA, particularly those originating from the LV summit.¹⁴

Finally, it is important to highlight that current guidelines favor ablation over antiarrhythmic drug therapy for PVCs in certain contexts, for example in symptomatic patients as first choice or after failed anti-arrhythmic drug therapy with beta-blockers, in patients with a decline in LV function due to a high PVC burden or poor biventricular pacing caused by PVCs in CRT patients. This will probably lead to a significant increase in the number of procedures in the future.¹⁵

Conflicts of interest

The author has no conflicts of interest to declare.

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