Localization of Accessory Pathways With Echocardiography, a 40-Year-Old Story

Historically, motion-mode (M-mode) echocardiography was the first effective modality for the ultrasonic detection of early pre-ejection events, concomitant with the delta wave in WPW syndrome. Because of its high temporal resolution (1000–3000 Hz), M-mode echocardiography can display the reduced amplitude of left ventricle (LV) posterior systolic wall motion for AP, emerging from LV basal free wall. This modality can also display rapid pre-ejection posterior septal motion associated with a slow anterior motion of the septum for AP, emerging from the right ventricular (RV) free wall. Twenty years later, pulsed-wave tissue doppler imaging (TDI) and high frame-rate tissue velocity imaging enabled AP-accurate localization in 50% more patients, when compared with the M-mode method.2 High temporal resolution (at least 150 Hz) is a potential advantage of TDI; however, clinical use was limited by artifacts, caused by myocardial translational motion, nonoptimal Doppler alignment, and poor reproducibility.

Approximately 10 years ago, 2-dimensional speckle-tracking echocardiography gained popularity in quantification of amplitude and timing of myocardial deformation, with a lower influence of cardiac motion as compared with TDI.3 Optimal frame rate for speckle-tracking echocardiography is 50 to 90 Hz, a much lower rate than that of M-mode or TDI. Nevertheless, using customized software, De Boeck’s team demonstrated that speckle-tracking echocardiography mapping matched the electrophysiology localization (with a tolerance of 1 contiguous segment) of the AP in 28 patients (85%) with a WPW syndrome.4 Importantly, early deformation associated with the AP was subsequently followed by reduced local deformation during systole, especially in patients with AP involving the interventricular septum (IVS). In a cohort of 40 patients with WPW syndrome referred for radiofrequency ablation, longitudinal speckle-tracking echocardiography strain accurately identified early basal pre-ejection LV deformation in 38 patients (95%),5 whereas early abnormal basal motion was not observed in control patients. Moreover, contractile abnormalities were found to be at worst one adjacent segment different compared with the AP ablation site in all 38 patients. Right-sided AP was associated with IVS contractile abnormalities, as previously reported with M-mode. The decrease in IVS deformation in AP involving the interventricular septum was more obvious than the decrease in LV free wall deformation in AP involving the LV free wall.

Ishizu et al are to be praised for their careful description of the value of 3-dimensional ST strain echocardiography to noninvasively localize AP in WPW syndrome.6 The advantage of 3-dimensional ST strain is the ability to assess area strain, a combination of circumferential and longitudinal strain of the entire ventricle. From a technical point of view, full volume data sets of the LV and the RV are acquired from apical views with a wide angle, to cover both ventricles, with a volume rate of at least 20 Hz (31±3 Hz, range 22–40 Hz), which is significantly lower than with other techniques. The authors used noninvasive isochrone AI, a technique previously validated against electric activation mapping in 7 patients with cardiomyopathies, 5 of them receiving cardiac resynchronization therapy (CRT) and 2 with an left bundle branch block (LBBB) during catheter ablation therapy of ventricular tachycardia.6 This method identifies the timing of deformation on each LV or RV segment and applies a time-dependent color-coded mapping either on a plastic bag model or on a polar map of the LV or RV. Segment deformation is coded by means of a graded color scale increasing in time, and color is applied when segment
deformation is of at least 25% of maximal deformation. Hence, this method allows for visual identification of both early and late deformed segments. Using this approach, the sites for the first LV and RV endocardial activation (the breakthrough sites) were frequently identified at the LV and RV apical or mid-walls in normal subjects. In contrast, the breakthrough site was perianular and basal in WPW patients. In 2 patients who underwent CARTO® mapping, AI mapping completely matched the CARTO® mapping and the ablation site. Agreement between AI and AP localization was perfect in only 38% of patients, but was observed in 87% of patients when applying a 2 o’clock–range tolerance for AP localization. In addition, the AI method was more precise than the electrocardiogram to accurately detect AP localization. Method reproducibility was excellent. Unexpectedly, septal area strain was not different between controls and WPW patients with AP involving the IVS, but was significantly reduced in basal and midanterior LV walls in patients with an AP involving the LV free wall. It is noteworthy that image quality issue, similar to other ultrasonic techniques, is inherent to 3-dimensional ST strain. Despite these potential limitations, this study presents promising data regarding a new visual method to accurately diagnose short-lived early events and is thus a potential new tool for the assessment of myocardial dyssynchrony.

**WPW Syndrome, Septal Preexcitation, Dyssynchrony, and Cardiac Resynchronization Therapy**

Novel data released by Ishizu et al does not change the electrophysiology laboratory’s daily practice. Indeed, invasive electrophysiology is the gold standard to accurately identify and treat the AP site. Echocardiographic techniques identify...
the contractile abnormalities associated with AP, but not the exact site for ablation.

However, the results of the present study may be considered alongside recent reports demonstrating that early septal activation associated with septal AP may induce reduced IVS motion, as encountered in patients with native or RV pacing-induced LBBB.11,13 The development of LBBB-mediated LV dysfunction responsive to CRT has been well documented.7 Reduced IVS motion and LV dys synchrony associated with septal AP may result in LV dysfunction, which resolves after radiofrequency ablation, even in the absence of permanent tachycardia.8,9 In both septal AP (Figure A) and LBBB (Figure D), activation proceeds across the septum at the breakthrough point before reaching the LV endocardium. It then slowly propagates to the endocardium of the posterolateral wall, with a basal breakthrough site in the event of an AP and more often a mid to apical septal one in the event of an LBBB. This activation delay correlates with a specific LV contractile pattern characterized by an early rapid depo sition of the IVS at the breakthrough site,10 associated with early stretching followed by delayed peak systolic deformation of the posterolateral wall (Figure).11 Two-dimensional ST longitudinal strain identified a similar contractile pattern in the event of IVS AP (Figure B and C) and LBBB (Figure D and F). Interestingly, the abnormal septal motion associated with this contractile pattern is more frequently found in patients with a prolonged QRS duration in both WPW8,12 and LBBB patients.11 In patients with an LBBB and heart failure receiving CRT, this classical LBBB longitudinal strain pattern has been linked to the occurrence of LV reverse remodeling, improvement in LV function, and clinical outcome, even in patients with relatively narrow QRS (120–150 ms) in whom the benefit of CRT remains uncertain.11,13

Given the excellent accuracy of AI to detect and image the contractile consequences of this activation delay in the WPW syndrome, AI may be a suitable and clinically useful tool to identify the contractile substrate of CRT response and, therefore, patients who will derive benefit from CRT. However, this hypothesis deserves further specific research.

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References

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