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LETTERS TO THE EDITOR

Noninvasive Voltage and Activation Mapping of ARVD/C Using ECG Imaging

Demonstration of electrical abnormalities may improve the sensitivity and specificity of diagnosis of arrhythmogenic right ventricular dysplasia/cardiomyopathy (ARVD/C) (1), but current invasive techniques are usually restricted to the right ventricle (RV), miss epicardial disease (critical to this pathology [2]), and are risky. We report first results with noninvasive imaging (ECVUE; CardioInsight Technologies, Inc., Cleveland, Ohio) using body surface potentials for reconstruction of epicardial electrograms (3). A 55-year-old woman with ARVD/C and ventricular arrhythmias was studied. Echocardiography showed RV dilation but a normal left ventricle (LV). Findings on electrocardiography (ECG) and computed tomography are shown in Figure 1A.

Voltage mapping identified a large subtricuspid abnormality (Fig. 1B) and a previously unknown small inferior LV lesion (position 6). During activation mapping in normal sinus rhythm (Fig. 1C), the earliest RV activation occurred at the lateral base 18 ms after onset of QRS. Thereafter, RV depolarization completed within 37 ms (total RV activation 55 ms from onset of QRS). The premature ventricular complexes initiating ventricular tachycardia originated from the anterior RV wall (Fig. 1D). On electrophysiological study, CARTO (Biosense Webster, Inc., Diamond Bar, California) mapping confirmed these findings. After ablation of premature ventricular complexes, the patient remained arrhythmia-free.

These findings illustrate several important points. Electrical abnormalities underlie the predilection for sudden death in ARVD/C, and modified task force criteria elevate their importance for diagnosis (4). However, the RV is for the most part electrically silent during conventional surface ECG, generating weak forces mostly concealed by the effects of LV depolarization (5). Early disease may be completely masked. Here, ECG criteria were not all positive because the interval from the nadir of the S-wave up to the isoelectric line in V_1 to V_3 (proposed criterion of "total activation duration") was <55 ms (Fig. 1A), although RV disease was marked. In contrast, enhanced electrocardiography (ECVUE) revealed unambiguous abnormalities (i.e., low-voltage lesions) with a characteristic sharp transition to normal myocardium both for advanced RV disease and the smaller LV lesion (1). Prolongation of RV depolarization time has been proposed as a diagnostic parameter and may be measured during activation mapping (Fig. 1C). Visualization of fractionated electrograms, which form channels for re-entrant circuits, and of ventricular tachycardia origin may guide ablation (2). The detection of a small zone of inferoseptal LV infiltration with typical electrogram characteristics (Fig. 1B, position 6), which had evaded detection previously, suggests utility for screening for early disease, for which conventional diagnostic criteria lack sensitivity.

In summary, electrocardiographic imaging has significant advantages for the diagnosis and management of ARVD/C. It

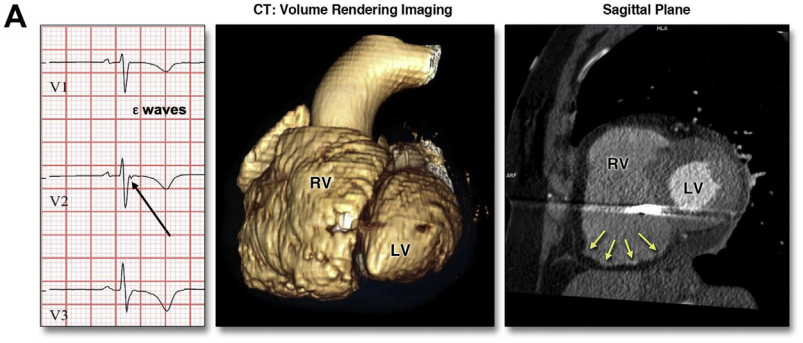

Figure 1. Imaging Sequences: ECG, CT, and Electrocardiographic Imaging

(A, left panel) A precordial T-wave inversion, epsilon waves **(arrow)**, and QRS duration of 98 ms are shown. The interval between the nadir of the S-wave up to an isoelectric line was 44 ms and to the end of all depolarization deflections was 50 ms (total activation duration). **(A, middle panel)** Gross right ventricular enlargement compared with a volumetrically normal LV is shown. **(A, right panel)** Right ventricular dilation with diffuse myocardial thinning is shown **(arrows)**, giving a "parched" appearance with epicardial fat apparently extending to the endocardial surface. The LV is normal in this view. **(B to D)** Biventricular maps with electrocardiographic (ECG) imaging are shown (ECVUE, 3 projections). **(B)** Sinus rhythm voltage map. A large subtricuspid scar **(red)** is shown, with a narrow border zone with rapid spread of intermediate colors indicating steep voltage gradients. Electrograms **(inset)** are normal in positions 1/2 and show reduced amplitude in 3/4 (border zone) but are grossly diminished/fractionated in the scar center (5). The LV is normal except for a small inferoseptal area with abnormal electrograms but with a sharp transition to normal tissue (6). **(C)** Sinus rhythm activation map. The earliest ventricular activation **(red)** in the lateral RV free wall was 18 ms after onset of QRS onset, with slow conduction within the scar (compressed midrange colors) and then confluent RV activation **(green)**. The duration of RV activation (earliest to latest free wall depolarization) was 37 ms. **(D)** Premature ventricular complex activation map. The earliest activation **(red)** from the anterior scar margin (QS electrograms characteristics of epicardial origin) is shown, followed by centrifugal RV activation. CT = computed tomography; LV = left ventricle; MV = mitral valve; NSR = normal sinus rhythm; PVC = premature ventricular contraction; RV = right ventricle; RVOT = right ventricular outflow tract; TV = tricuspid valve.

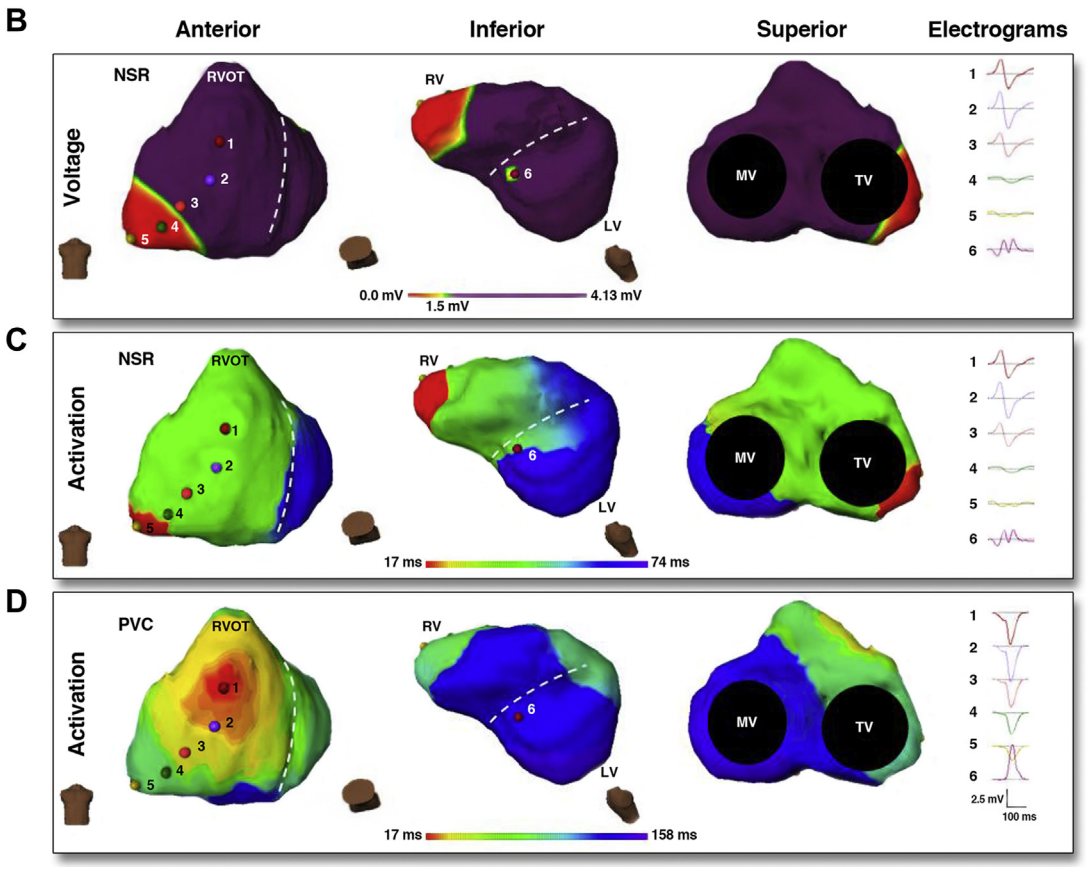

Figure 1. Continued

gathers data noninvasively and rapidly in a single heartbeat, reconstructs local electrograms, permits voltage and activation mapping of both ventricles simultaneously, and, by virtue of its ability for epicardial imaging, may detect nascent disease.

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Impact of Regurgitant Orifice Height for Mechanism of Aortic Regurgitation

A classification of aortic regurgitation (AR) by transesophageal echocardiography (TEE) has been considered a critical pre-operative assessment, particularly for valve repair operations (1). This study aimed to evaluate the mechanism of isolated AR by quantitative analysis of aortic valve apparatus (AVAp) by 3-dimensional (3D) TEE using novel software and to identify a discriminatory index for AR mechanisms.