Epicardial Wolff–Parkinson–White ablation

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A 45-year-old female patient without structural heart disease was referred for redo Wolff–Parkinson–White (WPW) ablation after an unsuccessful endocardial procedure. The refractory period of the accessory pathway had been measured at 230 ms and antiarrhythmic treatment with flecainide was unsuccessful.

1. The surface electrogram shows the largest negative $\delta$-wave in lead III.

2. During endocardial mapping of the ventricular insertion the earliest activation was found near the coronary sinus ostium (x); however, radiofrequency application was not successful. Note the morphology of the unipolar signal (Abl uni) with an initially positive small spike (red arrow) resulting from epi-endocardial activation followed by the broad negative ventricular activation signal.

3. Epicardial mapping via subxiphoidal access showed the earliest signal (XX) opposite to the endocardial site (X). The distance between the two locations was 1.5 cm. Note the morphology with an entirely negative small pathway spike (red arrow) followed by the broad negative ventricular activation signal. Irrigated radiofrequency ablation was successful after 25 s and increase of power from 20 to 30 W.

Epicardial access can be necessary, if the largest negative $\delta$-wave is in lead III on the surface ECG. The morphology of the unipolar signal indicates the true epicardial origin of the ventricular pathway insertion. Success can be observed even after delayed response to RF delivery.

Panel 1: ECG, electrocardiogram; LAO, left anterior oblique; Abl, ablation electrode; CS, coronary sinus; CSp, coronary sinus electrode proximal; Csd, coronary sinus electrode distal; RV, right ventricular electrode; LAT, local activation time measured relative to onset of QRS signals on surface ECG. The local activation time is measured conventionally (middle) and visualized in a coloured Carto-3D map (red earliest).

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