## Mechanical dispersion identifies patients with extensive electroanatomic abnormalities in Brugada syndrome

M. Ciabatti, M. Nesti, M. Reccia, E. Saletti, P. Notarstefano, M. Pieroni, L. Bolognese

San Donato Hospital of Arezzo, Arezzo, Italy Funding Acknowledgement: Type of funding sources: None.

**Background:** Brugada syndrome (BrS) was initially described as a pure electrical disorder caused by ion channel abnormalities in the absence of structural heart disease. However, imaging, autopsy and endomyocardial biopsy studies have increasingly demonstrated in patients with BrS the presence of myocardial structural alterations of the right ventricle (RV), particularly in the outflow tract. Indeed, electroanatomic mapping studies identified electroanatomic abnormalities of the RV outflow tract in both unipolar and bipolar maps with a significant correlation between the extension of low-voltage areas and the inducibility of arrhythmias at electrophysiological study or the incidence of malignant arrhythmias during the follow up. New echocardiographic parameters have been proposed to identify subtle myocardial alterations associated with arrhythmic events. Mechanical dispersion (MD) of the left ventricle (LV) has been identified as a prognostic marker in the arrhythmic risk stratification in various cardiac diseases including some cardiomyopathies.

**Purpose:** In this study we evaluated MD and global longitudinal strain (GLS) of RV and LV in patients with BrS to identify echocardiographic correlates of the abnormalities detected by electroanatomic mapping.

**Methods:** We performed 2D-Echocardiography with speckle tracking analysis of RV and LV in patients with BrS previously submitted to RV electroanatomic mapping. All studies were performed by investigators blind to clinical features and electrophysiological findings. Echocardiographic data were compared with electroanatomic mapping and electrophysiological study findings and with clinical data.

**Results:** We enrolled 18 patients (52±11 years, male 44%). Patients with a LV MD value  $\geq$ 40 ms showed a pathological unipolar area with voltage <5.5 mV significantly more extended than patients with a LV MD value <40 ms (28.49±21.06 vs 10.47±8.22; p=0.03). Patients with LV MD  $\geq$ 40 ms also showed a trend to greater extension of the unipolar area with voltage <4 mV (13.94±13.11 vs 4.94±3.12; p=0.07), a greater extension of higher inducibility at programmed ventricular stimulation (70% vs 37.5%, p=0.34). No correlation was observed between RV MD or GLS values and the extent of the low-voltage areas or with the presence of genetic mutations associated with BrS.

**Conclusions:** In patients with BrS a LV MD  $\geq$ 40 ms is associated with a greater extension of low-voltage areas at unipolar mapping. Echocardiographic evaluation with MD analysis may represent a valuable noninvasive tool to identify electroanatomic alterations prompting further invasive studies including electronatomic mapping and electrophysiological study. Prospective studies on larger series may further clarify the potential role of MD and electroanatomic mapping in the prognostic stratification of patients with BrS.

