Abstract: P99

Rapid ventricular stimulation induces augmented conduction delay in Brugada syndrome patients

Authors:
N Yoon¹, HK Jeong¹, KH Lee¹, HW Park¹, JG Cho¹, Chonnam National University Hospital - Gwangju - Korea (Republic of),

Topic(s):
Brugada Syndrome

Citation:
Background: The exact mechanism for Brugada Syndrome (BrS) is still not clear. There are two main physiologic hypotheses that have been suggested: the repolarization and the depolarization disorder models. Right ventricular (RV) activation delay was verified by echocardiography, conduction time in an explanted heart or in computer simulation. Verification of prolonged longitudinal activation time in human RV of only 5 patients of type-1 BrS and 5 controls was reported in 2008.

Methods: Bidirectional longitudinal activation times were assessed between RV outflow tract (RVot) and RV-apex (RVA) by stimulating and mapping RV endocardium in BrS patients. Conduction velocity was calculated considering ventricle size and distance between catheters.

Results: The studies were performed in controls (n=18) and BrS patients (n=6). There was no statistical difference in RP interval and QRS duration (PR 146±21.7 vs 167±45.2 ms, p=0.325; QRS 102±28.2 vs 122±32.2 ms, p=0.163). There was no difference of longitudinal activation time on stimulation at 500 ms (RVA to RVot: 63±14.3 versus 80±34.2 ms, p=0.290; RVot to RVA: 50±12.2 versus 76±35.1 ms, p=0.122). The BrS patients had longer longitudinal activation time on stimulation at 400 ms (RVA to RVot: 61±15.2 versus 87±28.7 ms, p=0.009; RVot to RVA: 52±11.1 versus 76±35.3 ms, p=0.029). The difference was not significant when isoproterenol was infused.

Conclusions: BrS patients display bidirectional longitudinal conduction delay when rapid stimulation. These findings support that BrS might be partly attributable to depolarization abnormality.