INDUCTION OF BRUGADA SYNDROME IN THE CANINE RV WEDGE PREPARATION BY SIMULTANEOUSLY PERFUSION OF PINACIDIL, TERFENADINE AND PILSICAINIDE

Poster Contributions
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Background: The cellular basis for the Brugada syndrome (BS) is thought to involve an outward shift of net transmembrane current active at the end of phase 1 of the RV epicardial action potential (AP) where Ito is most prominent. In response to blockage of INa or ICa or augment of IK-ATP, IKr, IKs, or Ito, epicardium may first exhibit an accentuation of the spike-and-dome morphology of the AP, resulting in a delay in the development of the dome and accentuation of the notch, and leaving Ito unopposed during phase 1 of the action potential, leading to a predominance of outward repolarizing current at the end of phase 1. We simultaneously applied Pinacidil-a K+ channel opener, Terfenadine-a Na+ and Ca2+ channels blocker and Pilsicainide-a Na+ channel blocker to establish a model of BS in canine RV perfused wedge preparation.

Methods: In 20 arterially perfused canine RV wedge preparations, Pinacidil at 2ummol/L, Terfenadine at 5ummol/L and Pilsicainide at 5ummol/L were simultaneously applied. Transmembrane AP was recorded from epicardium and endocardium, as well as pseudo-ECG. Program stimulation was performed to induce arrhythmia.

Results: BS model was successfully established in 16 preparations presenting as significant J point elevation, increase of transmural dispersion of repolarization and high incidence of induced Tdp (Figure 1).

Conclusion: Simultaneously perfusion with Pinacidil, Terfenadine and Pilsicainide can produce an experimental model of BS in the canine RV wedge preparation.