

# Phase-2 re-entry

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Phase-2 re-entry is thought to underlie many causes of idiopathic ventricular arrhythmias as, for instance, those occurring in Brugada syndrome (BrS). J-point elevation, ST-elevation, and T-wave changes documented in the last sinus beat prior to ventricular extrasystoles are in agreement with phase 2 reentry, suggesting that this may be the responsible mechanism for ventricular extrasystoles and ventricular tachycardia/fibrillation. The reported pathogenesis of BrS is phase-2 reentry resulting from shortening of the epicardial AP duration at the right ventricular outflow tract (RVOT) (**Ohkubo 2010**). Studies in an isolated canine RVOT model of BrS demonstrated that reversal of the transmural gradient of repolarization caused the ECG characteristics and that major intraepicardial and transmural dispersion of APs initiated phase 2 reentry, PVCs, and tachyarrhythmias (**Morita 2009**). In BrS patients, ST-segment elevation is modulated by cardiac sodium ( $I(\text{Na})$ ), transient outward ( $I(\text{to})$ ) and L-type calcium currents ( $I(\text{CaL})$ ). ST-segment elevation in Brugada patients, is modulated by  $I(\text{to})$  and  $I(\text{CaL})$  (**Hoogendijk 2011**). PVC falling after the end of the T wave triggered VF at night and monomorphic ventricular tachycardia (MVT) during daytime. While  $I(\text{to})$  dispersion is necessary for phase-2 reentry, altered sodium inactivation kinetics influences the probability of reexcitation in a highly nonlinear fashion (**Cantalapiedra 2009**).

## References

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