

# Brugada Syndrome

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# Introduction

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- Cardiac sodium channelopathy i.e., faulty Sodium channels
- Potential for fatal arrhythmias like VF & sudden death
- Typically, in young males

# Pathology

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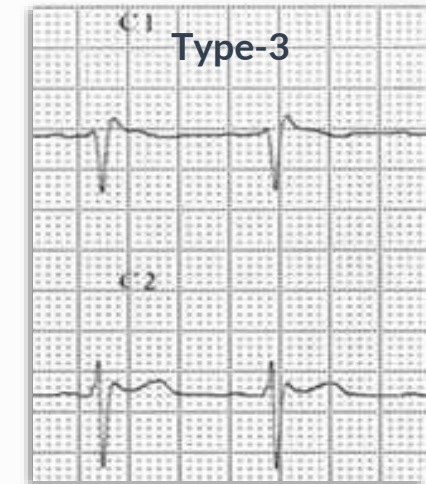
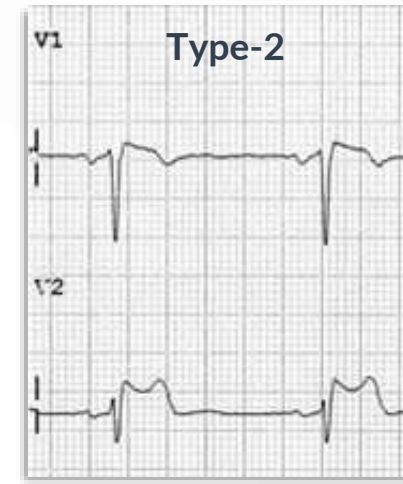
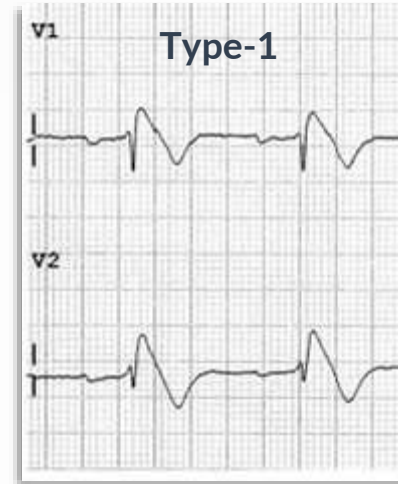
- Mutations in cardiac sodium channel gene
- >60 different mutations have been described so far
- ~50% - Spontaneous mutations
- Familial clustering & autosomal dominant inheritance have been demonstrated
- Mutations in the SCN5A gene encoding cardiac voltage-gated sodium channel found in 15-20% cases

# ECG Patterns

3 patterns

## Type-1

- *Coved* ST-segment elevation  $>2\text{mm}$  in  $>1$  of leads V1-V3, followed by a negative T wave
- Potentially diagnostic pattern & called as **Brugada sign**



## Type-2

- $>2\text{mm}$  of *saddleback* shaped ST-elevation

## Type-3

- ST morphology either type-1 or type-2, but ST-segment elevation  $<2\text{mm}$

ECG changes can be transient & can be unmasked or augmented by multiple factors

- Fever
- Ischemia
- Multiple Drugs -Sodium channel blockers (Flecainide & Propafenone), Calcium channel blockers, Alpha agonists, Beta-Blockers & Nitrates
- Cholinergic stimulation
- Cocaine
- Alcohol
- Hypokalemia
- Hypothermia
- Post DC cardioversion

# Diagnosis

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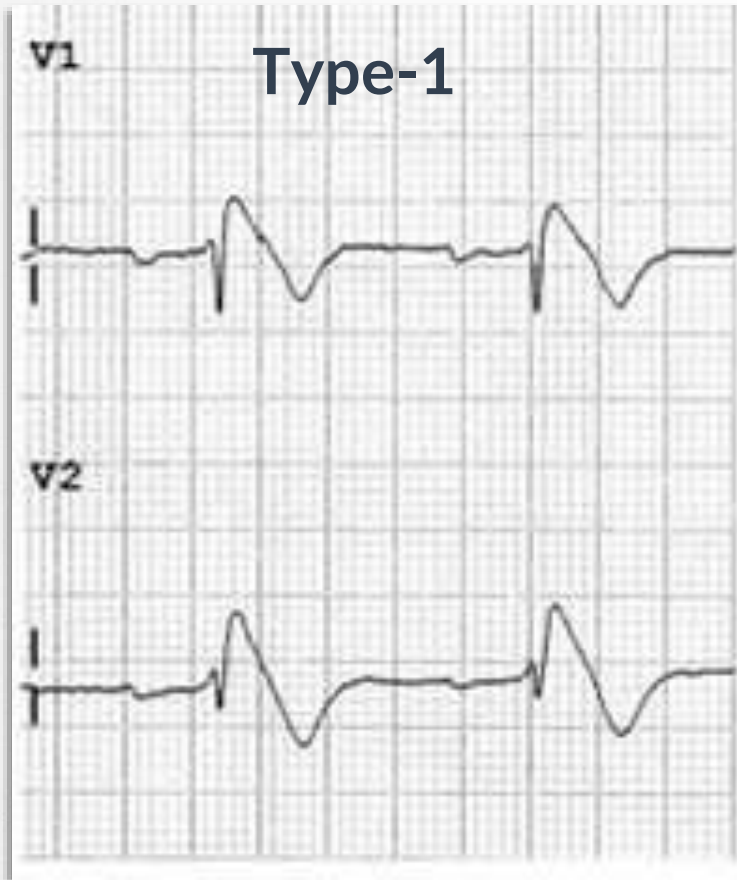
- Coved ST-elevation in V1-V3 + one of following clinical criteria
  - Documented VF or Polymorphic VT
  - Family history of sudden cardiac death at <45 years age
  - Coved-type ECGs in family members
  - Inducibility of VT with programmed electrical stimulation
  - Syncope
  - Nocturnal agonal respiration

# Treatment

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- Implantable cardioverter defibrillator (ICD) – Only proven therapy
- Quinidine - proposed as an alternative when ICDs unavailable or where they would be inappropriate e.g., in neonates
- Screen family members to pick up undiagnosed cases

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