NOTES
SUDDEN CARDIAC DEATH

GENERALLY, WHAT IS IT?

PATHOLOGY & CAUSES

- Abrupt cessation of cardiac activity (cardiac arrest) in someone asymptomatic up until moment of arrest

SIGNS & SYMPTOMS

- Asymptomatic

DIAGNOSIS

- Made based on lack of pulse

TREATMENT

MEDICATIONS

- According to Advanced Cardiac Life Support protocols

OTHER INTERVENTIONS

Cardiopulmonary resuscitation (CPR)

- Maintains blood flow by mimicking pumping motion heart makes during a medical emergency

BRUGADA SYNDROME

osms.it/brugada-syndrome

PATHOLOGY & CAUSES

- Condition with characteristic abnormal electrocardiogram findings → increases risk of sudden cardiac death in healthy individuals
- Mixture of normal, abnormal sodium channels within adjacent myocardial tissue can set up heterogenous refractory periods necessary for development of reentrant rhythms → ventricular tachycardia/fibrillation

CAUSES

- Inherited
  - Autosomal dominant, variable expression
- 20% associated with *SCN5A* gene mutation which encodes for sodium ion channel in cell membranes of heart muscle cells

RISK FACTORS

- Biological males, more common in Asia than North America, Europe

COMPLICATIONS

- Ventricular fibrillation, high risk of sudden cardiac death

SIGNS & SYMPTOMS

- Brugada pattern
  - ECG findings, no symptoms
- Brugada syndrome
  - ECG findings, symptoms of sustained ventricular tachycardia (palpitations, syncope, dyspnea, lightheadedness)

DIAGNOSIS

LAB RESULTS

Genetic testing

- Confirms diagnosis

OTHER DIAGNOSTICS

ECG

- Type I/II Brugada electrocardiogram pattern
  - May present simultaneously, may be induced by certain drugs (e.g. calcium channel blockers), or may resurface due to unknown triggers
- Type I
  - Right bundle branch block pattern
  - Gradually descending ST elevations, at least 2mm (0.2mV) in leads V_1–V_3
  - Negative T- wave in leads V_1–V_3
- Type II
  - Class IV antiarrhythmic can convert to a Type I Brugada pattern—often needed for diagnosis
  - Saddle-back pattern with at least 2mm J point elevation, 1mm ST elevation (positive/biphasic T wave)

TREATMENT

- *Brugada pattern:* none

SURGERY

Implanted cardiac defibrillator (ICD)

- Brugada syndrome

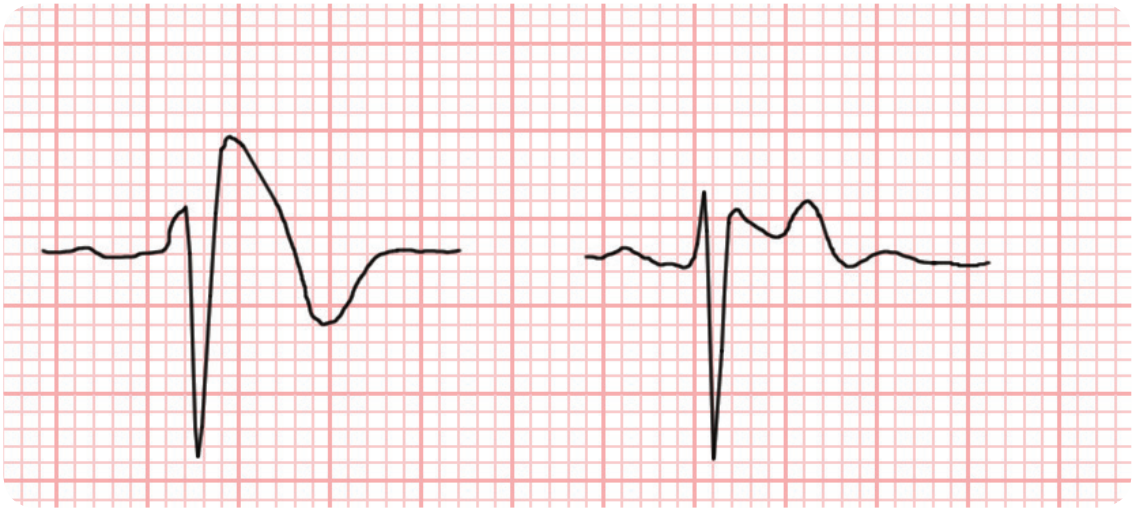

Figure 19.1 ECG (lead V_1) demonstrating Brugada waveforms type I (left) and type II (right).

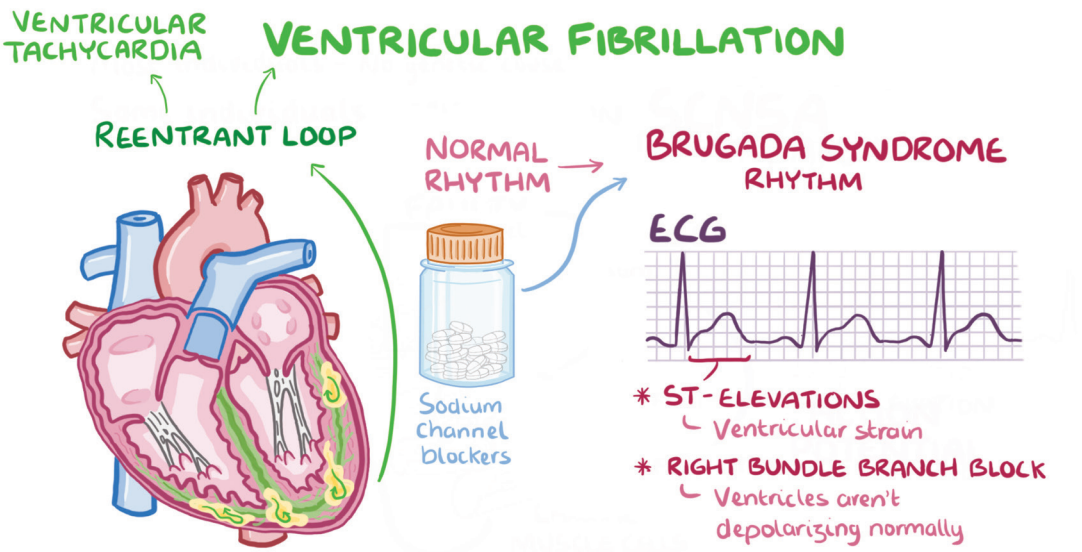

Figure 19.2 Calcium channel blockers can increase the chance of developing Brugada syndrome. The condition is typically associated with right bundle branch block, which makes the heart susceptible to developing a reentrant rhythm, which in turn causes ventricular tachycardia and sometimes ventricular fibrillation.

PULSELESS ELECTRICAL ACTIVITY

osms.it/pulseless-electrical-activity

PATHOLOGY & CAUSES

- Pulseless, despite electrical activity (evident on ECG) typically resulting in pulse
- Heart does not contract in spite of electrical activity/does not generate enough cardiac output to cause pulse
- Survival ~20%

CAUSES

- Abrupt drop in preload
- Abrupt pump failure

MNEMONIC: 6Ts & 6Hs

Obstruction to blood flow

Tablets/toxins (drug overdose)
Cardiac **T**amponade
Tension pneumothorax
Thrombosis (myocardial infarction)
Thrombosis (pulmonary embolism)
Trauma (hypovolemia - blood loss)
Hypovolemia
Hypoxia
Hydrogen ions (acidosis)
Hyperkalemia/hypokalemia
Hypoglycemia
Hypothermia

SIGNS & SYMPTOMS

- Loss of consciousness
- Breathing stops

DIAGNOSIS

OTHER DIAGNOSTICS

- Absence of pulse

ECG

- Organized/semi-organized electrical activity

TREATMENT

MEDICATIONS

- If cause unclear medicine used similar to asystole
  - Intravenous/intraosseous line, administer epinephrine 1mg/3–5 minutes

VENTRICULAR FIBRILLATION

osms.it/ventricular-fibrillation

PATHOLOGY & CAUSES

- Ventricular electrical activity disorganized to point that coordinated contraction is impossible
- Rapid, irregular electrical activity prevents ventricles from contracting in sync → cardiac output falls to zero
- *Often due to tissue heterogeneity:* heart cells stressed/damaged, tissues of different areas structurally, electrically different
- *Mechanism:* tissue heterogeneity in cardiac electrical system → asynchronous depolarization & contraction → inadequate blood pumped → oxygen deprivation → death
- *Functional reentry:* arrhythmia causes different areas of heart to depolarize & contract out of sync → heart non-functional

CAUSES

- Medications causing long QT syndrome
- Illicit drugs (e.g. methamphetamine, cocaine)
- Congenital arrhythmogenic syndromes (e.g. Brugada, hypertrophic cardiomyopathy, arrhythmogenic right ventricular dysplasia, Wolff-Parkinson-White syndrome, congenital long QT syndrome)
- Electrolyte imbalances: hypokalemia, hyperkalemia
- Ischemia to ventricular muscle
- Scar tissue from previous myocardial infarction
- Anatomical reentry
- Electrocution/external electrical stimulation, such as in unsynchronized cardioversion
- If heart tissue stimulated during T wave upslope (in an electrocardiogram), can induce fibrillation

SIGNS & SYMPTOMS

- Chest pains, dizziness, nausea, rapid pulse, dyspnea

DIAGNOSIS

OTHER DIAGNOSTICS

- Pulse check: no pulse

ECG

- Absence of PQRST waves; instead, fine, coarse fibrillatory waves
- Electrocardiogram appears chaotic
- Undulating baseline

TREATMENT

SURGERY

ICD

- Used when cause is unpreventable
- Surgically implanted
- Constantly monitors electrocardiogram
- ICD recognizes ventricular fibrillation, delivers responsive defibrillating shock
- Doesn't fix underlying condition; treats symptom, improves survival
- Primary prevention
  - Individuals with heart failure at risk of ventricular tachycardia/fibrillation
- Secondary prevention
  - Cardiac arrest survivors for whom triggers cannot be treated/prevented

OTHER INTERVENTIONS

Cardiopulmonary Resuscitation (CPR)

Defibrillation

- High energy shock depolarizes large enough portion of tissue (critical mass) that sinus node can take control

Electrophysiology study

- If individual has had previous MI/has survived cardiac arrest in whom signs are not apparent after routine, non-invasive testing
  - Evaluate for possible ventricular tachycardia ablation

Revascularization

- If ventricular fibrillation occurs in setting of myocardial infarction
  - Cardiac catheterization
  - CABG

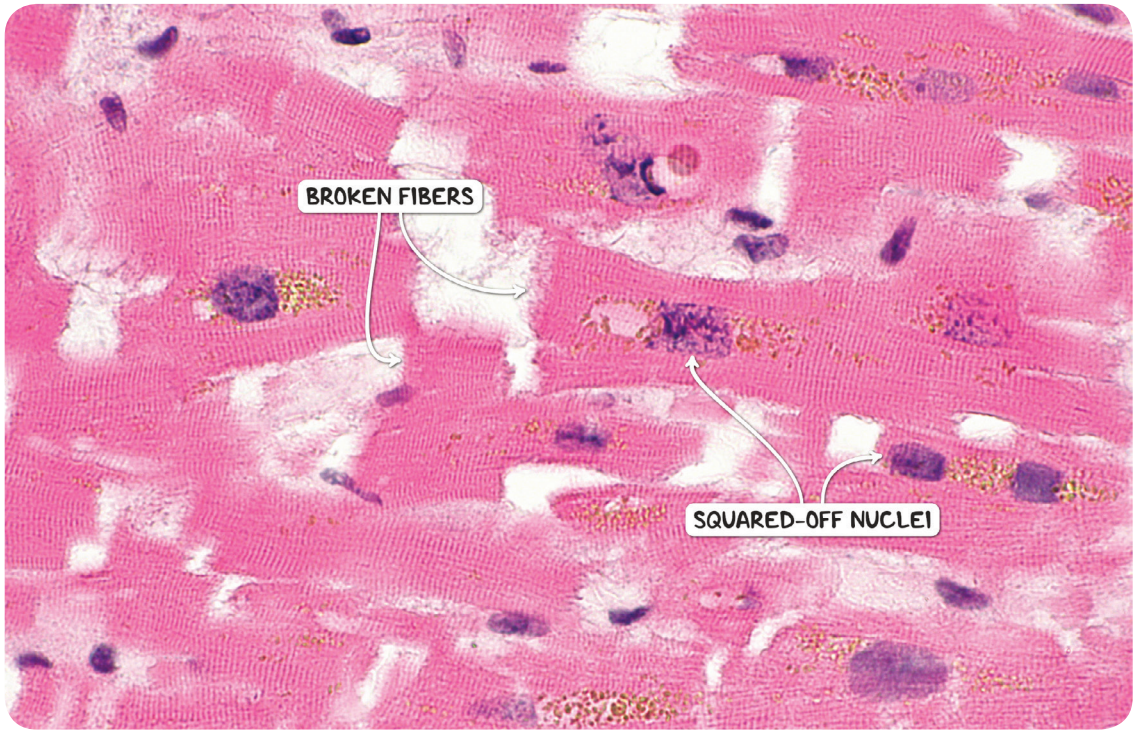

Figure 19.3 Histological appearance of fatal ventricular fibrillation demonstrating broken myocardial fibers and squared-off nuclei.

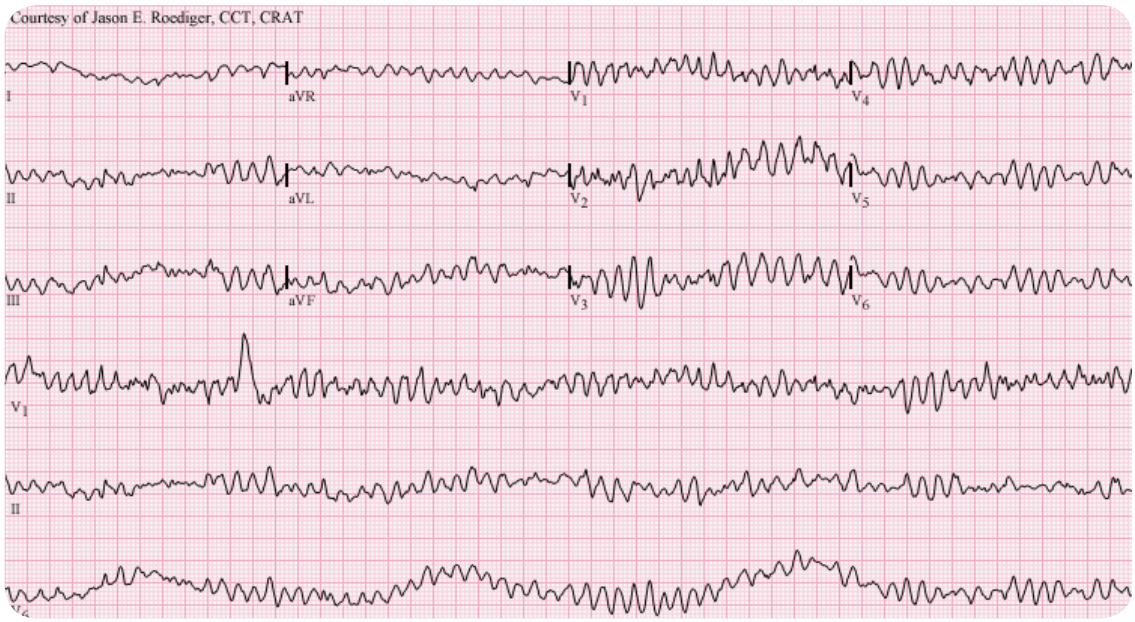

Figure 19.4 ECG demonstrating ventricular fibrillation.