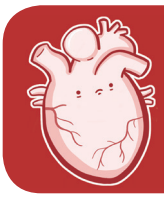

NOTES
VENTRICULAR TACHYCARDIA

GENERALLY, WHAT IS IT?

PATHOLOGY & CAUSES

- Depolarization wavefronts originate in ventricles → ventricles pump > 100 beats per minute ↓ stroke volume
- *Premature ventricular contractions (PVCs):* single instance of ventricle contracting prematurely
  - ≥ three PVCs consecutively defined as ventricular tachycardia (VT)

TYPES

Monomorphic VT

- Ventricular contractions have typical, uniform shape
- Typical for reentrant circuits
  - Depolarizations begin from same spot, for focal VT because one area of cells in ventricle is responsible
  - Often caused by reentry around scar in ventricular wall; e.g. from previous myocardial infarction (MI)

Polymorphic VT

- > One QRS complex morphology type
  - Includes Torsades de pointes
- Shape of contractions from each beat changes as signal begins in different areas of ventricle
- May occur when pacemaker cells stressed, increasing automaticity rates, including from severe hypoxia

RISK FACTORS

- Ventricular muscle ischemia, structural heart disease, coronary artery disease (CAD), electrolyte abnormalities

COMPLICATIONS

- Sustained VT may result in sudden cardiac death due to insufficient blood perfusion/ rapid ventricular fibrillation

SIGNS & SYMPTOMS

- Chest pain, syncope, dizziness, shortness of breath, palpitations

DIAGNOSIS

LAB RESULTS

- Serum electrolytes
- Toxicology studies (therapeutic/recreational drug use)
  - E.g. digoxin, tricyclic antidepressants, methamphetamine, cocaine

OTHER DIAGNOSTICS

ECG

- Determines cardiac rhythm

TREATMENT

MEDICATIONS

- Pharmacotherapy
  - Depending on cause

SURGERY

- Implanted devices

OTHER INTERVENTIONS

- Cardioversion, pacing
- Correct underlying cause

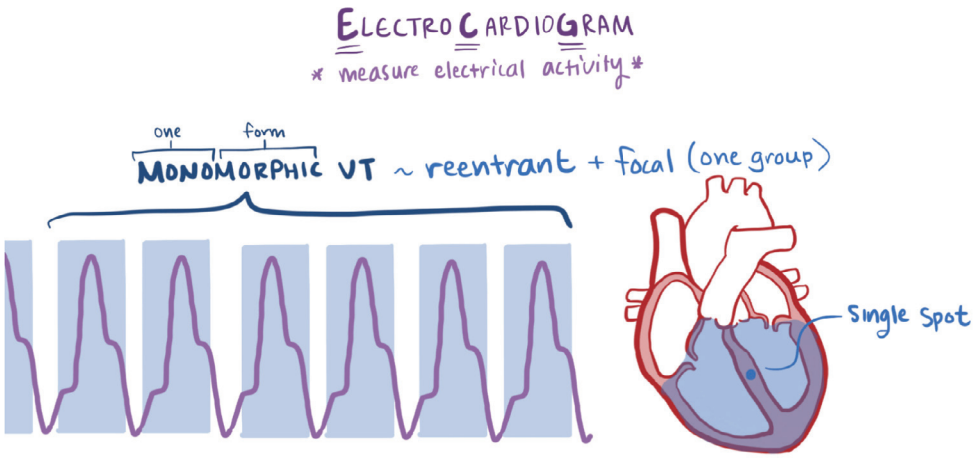

Figure 27.1 Illustration depicting ECG of monomorphic ventricular tachycardia.

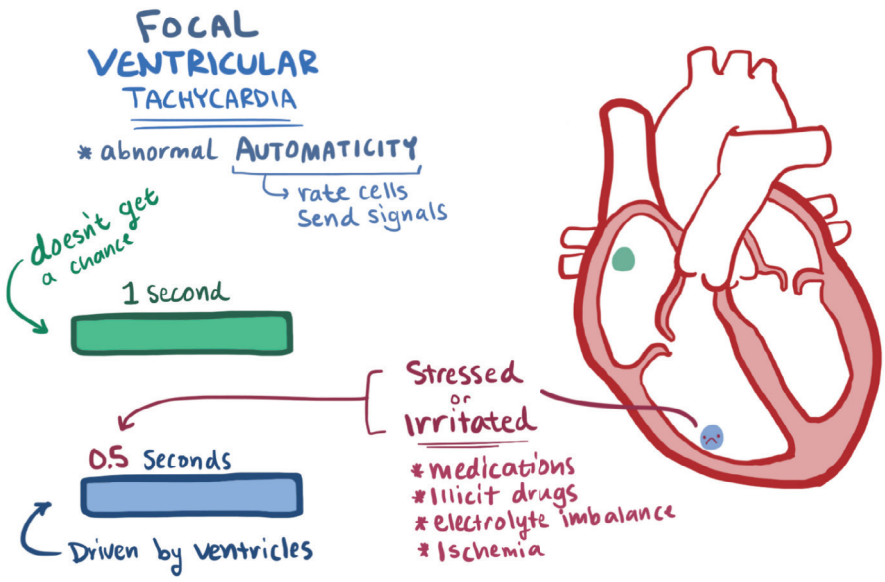

Figure 27.2 Illustration depicting focal ventrical tachycardia.

LONG QT SYNDROME (LQTS)

osms.it/long-qt-syndrome

PATHOLOGY & CAUSES

- Cardiac rhythm disorder characterized by prolonged ventricular repolarization
- Characterized by abnormally long QT interval
  - *QT interval:* total time from ventricular depolarization (QRS complex) to complete repolarization (T wave); measured from beginning of QRS to end of T wave
  - QTc (corrected) accounts for changes in heart rate: QTc = QT interval ÷ √RR interval (in sec); AKA Bazett formula
  - Adult normal = 420 ± 20 msec
- Results in ↑ risk of polymorphic ventricular arrhythmias (TdP), which can deteriorate into ventricular fibrillation

TYPES

Inherited

- Caused by mutations in genes associated with cardiac potassium, sodium channels
- Triggered by exertion, emotional events, stress, postpartum events, noise
- ≥ 13 types identified; associated with mutations in genes encoding myocyte ion channels
- *KCNQ1*, *KCNH2*, *KCNE1*, *KCNE2* affect potassium channels → ↓ outward potassium current
- *SCN5A* affects sodium channels → ↑ inward sodium current
  - Two distinct LQT phenotypes due to mutant alleles in same locus:
  - *Romano–Ward syndrome*: autosomal dominant; LQTS without hearing loss
  - *Jervell and Lange–Nielsen syndrome*: autosomal recessive; LQTS with congenital sensorineural hearing loss

Acquired

- Usually caused by certain drugs (e.g. anti-infectives, psychotropics, antiarrhythmics, antineoplastics, bronchodilators, gastric motility agents)
  - Common mechanism involves blockage of rapidly activating potassium channels (IKr) current in potassium channel encoded by *KCNH2* gene

RISK FACTORS

- Electrolyte imbalances (e.g. hypokalemia, hypomagnesemia, hypocalcemia)
- Underlying heart disease (e.g. HF, hypertrophic left ventricle, history of myocardial infarction)
- Bradyarrhythmias
- Biological females > biological males
- ↑ age
- Inherited genetic mutation
- Postpartum period
  - Related to physiologic stress, altered sleep patterns
- Anorexia nervosa

COMPLICATIONS

- Malignant arrhythmias (TdP, VF), syncope, seizures, sudden death

SIGNS & SYMPTOMS

- Palpitations, lightheadedness, hypotension

DIAGNOSIS

LAB RESULTS

- Serum electrolytes
  - Hypokalemia, hypomagnesemia, hypocalcemia may be present

OTHER DIAGNOSTICS

12-lead ECG

- Prolonged QTc (> 470msec in males, > 480msec in females); presence of tachyarrhythmias (TdP); altered T-wave morphology

Bicycle/treadmill stress test

- Presence of exercise-associated arrhythmias

Catecholamine drug testing

- Differentiates etiology
  - Provocative testing with catecholamine; e.g. epinephrine
  - Measure effect on QT interval

Clinical/family history, physical examination

- With compatible findings

Genetic testing

Schwartz score

- Diagnosis of congenital LQTS by scoring QTc, clinical factors, individual history
- *Scoring:* probability of congenital LQTS
  - ≤ *1:* low
  - *1.5–3:* intermediate
  - ≥ *3.5:* high

TREATMENT

MEDICATIONS

Congenital LQTS

- *Beta-blockers:* blunt adrenergic response
- *Mexiletine:* for sodium-channel mutations
- *Flecainide:* if *SCN5A* mutation

Acquired LQTS

- *Magnesium sulfate:* treatment, prevention of recurrence of long QT-related ventricular ectopic beats
- *Isoproterenol:* increase sinus rate, decrease QT interval
- *Lidocaine/phenytoin:* shorten duration of the action potential

SURGERY

Congenital LQTS

- Left cervicothoracic sympathectomy (LCTS), left cardiac sympathetic denervation; implantable cardioverter-defibrillator (ICD); pacemaker

Acquired LQTS

- Pacemaker → if bradycardia triggers arrhythmia

OTHER INTERVENTIONS

Acquired LQTS

- Address underlying cause; e.g. correct electrolyte abnormalities, discontinue offending drug; temporary transvenous overdrive pacing, electrical cardioversion/ defibrillation

Lifestyle modifications

- Avoidance of triggering drugs, avoidance adrenergic stimuli; e.g. strenuous exercise, emotional stress

DIAGNOSIS OF CONGENITAL LQTS

CRITERIA	POINTS
ECG FINDINGS	
QTc (ms)	
> 480	3
460-479	2
450-459 (males)	1
4-minute recovery from exercise stress test > 480	1
TdP	2
T wave alternans	1
Notched T wave (in 3 leads)	1
Low HR rate (age adjusted)	0.5

DIAGNOSIS OF CONGENITAL LQTS	
CLINICAL HISTORY	
Syncope	
With stress	2
Without stress	1
Congenital deafness	0.5
FAMILY HISTORY	
LQTS confirmed in family member	1
Unexplained sudden death in immediate family members (< 30 years of age)	0.5

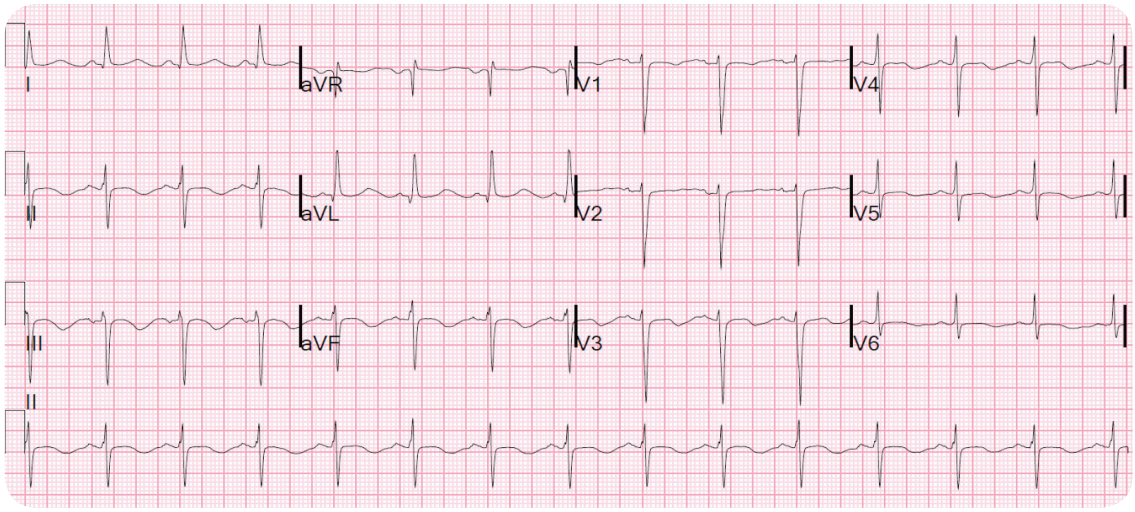

Figure 27.3 ECG trace demonstrating long-QT syndrome.

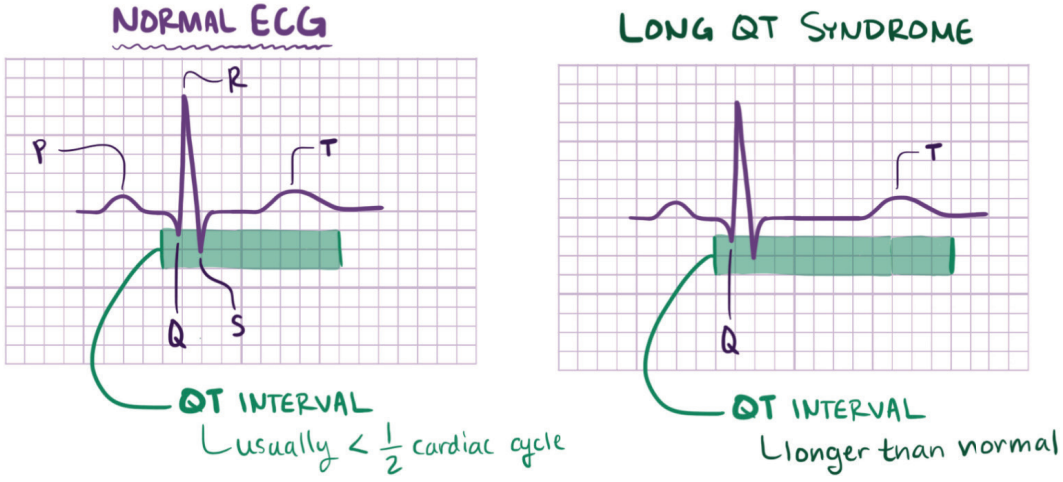

Figure 27.4 ECG trace demonstrating long-QT syndrome.

TORSADES DES POINTES (TdP)

osms.it/torsades-de-pointes

PATHOLOGY & CAUSES

- Literally means "twisting of the points"
- The peaks of QRS complex "twist" around isoelectric line on electrocardiogram
- Lengthening QT interval → early afterdepolarizations (EADs) → premature ventricular depolarizations → polymorphic VT (TdP)
  - May resolve spontaneously
  - Transmural reentry/abnormal automaticity may perpetuate TdP
  - May degenerate into ventricular fibrillation

RISK FACTORS

- LQTS, drugs associated with LQTS
- Bradycardia
- Electrolyte imbalance
- Biologically female
- Anorexia nervosa

COMPLICATIONS

- Ventricular fibrillation, seizures, sudden cardiac death

MNEMONIC: TO4SADE

Drugs that may induce QT prolongation

Thiazides

O4 - Oanzapine, Opioids, Quinidine, Quinolones

Sotalol/SSRIs

Antihistamines/antipsychotics

Anti**D**epressants like TCAs

Erythromycin (Macrolide antibiotics

SIGNS & SYMPTOMS

- Palpitations, lightheadedness, syncope

DIAGNOSIS

LAB RESULTS

- Serum electrolytes
  - Hypokalemia, hypomagnesemia, hypocalcemia may be present

OTHER DIAGNOSTICS

- 12-lead ECG
  - *Ventricular rate:* 150–300 beats per minute
  - *RR interval:* irregular
  - *P wave, PR interval:* absent
  - *QRS duration:* > 0.12 seconds; changes amplitude, shape, direction

TREATMENT

MEDICATIONS

- For acquired LQTS/other causes of TdP
  - Magnesium sulfate, isoproterenol, lidocaine, phenytoin
- For congenital LQTS
  - Beta-blockers, mexiletine

SURGERY

- Left cardio-thoracic sympathectomy

OTHER INTERVENTIONS

- Treat underlying cause; e.g. correct electrolyte abnormalities, discontinue offending drug
- Temporary pacing, permanent dual chamber pacemaker, implantable cardioverter-defibrillator (ICD)

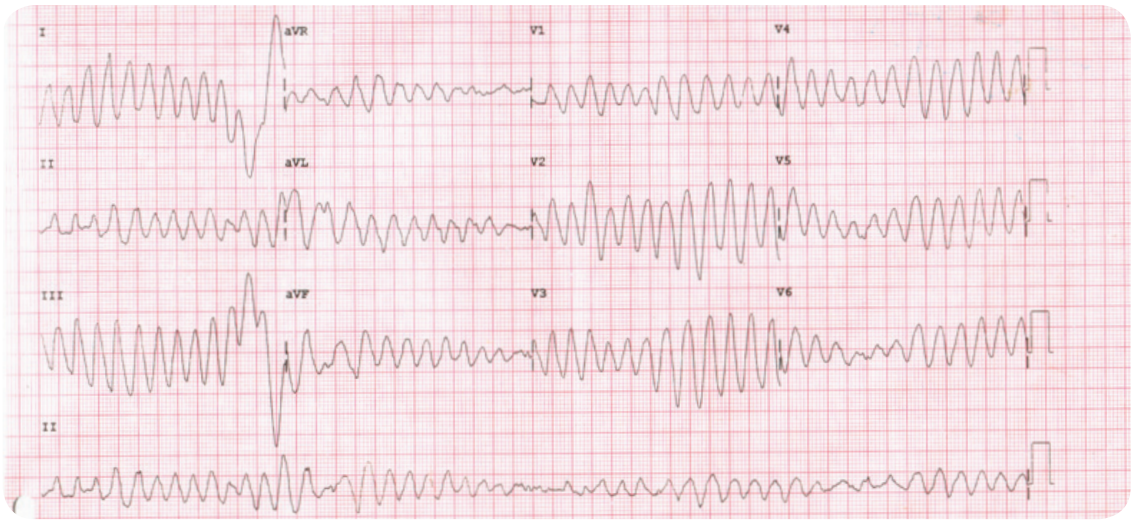

Figure 27.5 ECG demonstrating torsades de pointes.

VENTRICULAR TACHYCARDIA

osms.it/ventricular-tachycardia

PATHOLOGY & CAUSES

- Ventricular arrhythmia originating in ectopic ventricular pacemaker, resulting in ≥ three premature ventricular complexes (PVCs) occurring at ≥ 100 beats/min
- Dysrhythmia may be sustained (> 30 seconds)/nonsustained (< 30 seconds)/paroxysmal
- Abnormally fast ventricular contractions → ↓ ability for ventricles to fill → ↓ cardiac output → ↓ perfusion → impaired hemodynamics

TYPES

Non-reentrant/focal ventricular tachycardia

- Triggered by abnormal automaticity of specific area of ventricle
  - Ventricular pacemaker cells fire at high rate, preventing pacemaker cells in SA node from firing → heart rate driven by ventricular pacemakers
  - May be caused by certain medications, illicit drugs (e.g. methamphetamine, cocaine), electrolyte imbalances, myocardial ischemia in ventricles

Reentrant ventricular tachycardia

- More common than focal VT
- *Reentry:* perpetual electrical signal that occurs due to changes in refractory period length, rate of signal conduction
  - Cardiomyocytes can be altered when stressed/irritated by external stimuli; e.g. medications/illicit drugs: change conduction speed, refractory period
  - Dead cells in myocardial tissue create scar tissue → conduction signals go around scar → perpetual signal, AKA reentry

RISK FACTORS

- ↑ age
- Cardiac disease
  - Post-MI, cardiomyopathy, valve disease, HF
- Electrolyte imbalance
- Cardiac ion channelopathies resulting in long QT syndromes
- Infiltrative disease; e.g. amyloidosis
- Pericardial inflammation
- Blunt chest trauma
- Drugs; e.g. cocaine

COMPLICATIONS

- Cardiac ischemia, infarction
- May degenerate into ventricular fibrillation
- Sudden cardiac death

SIGNS & SYMPTOMS

- Chest pain
- Shortness of breath
- Dizziness
- Syncope
- Pallor
- Blood pressure: normal/↓

DIAGNOSIS

LAB RESULTS

- Serum electrolytes
  - Hypokalemia, hypomagnesemia, hypocalcemia may be present

OTHER DIAGNOSTICS

ECG

- *Rate:* >100 beats per minute, irregular
- *P waves:* may be absent
  - If present, atrioventricular dissociation common (hallmark of VT)

    - May be positive/upright or negative/inverted in Lead II
- *PR interval:* none
- *QRS complex:* wide (> 0.12 seconds); ≥ 3 consecutive; distorted shape: may be monomorphic/polymorphic
- *T-waves:* large; polarity may be opposite of major QRS deflection; may be difficult to distinguish

TREATMENT

MEDICATIONS

- Chronic prevention
    - Beta blockers, amiodarone, nondihydropyridine calcium channel blockers

OTHER INTERVENTIONS

- Acute treatment
    - Cardioversion
- Drug cardioversion
    - Procainamide, lidocaine, amiodarone frequently used
- Electrical cardioversion
    - Primary treatment for VT associated with hemodynamic instability/when drug cardioversion not immediately available
- Radiofrequency catheter ablation
- ICD