Ventricular Arrhythmias
Mechanisms, Features, and Management

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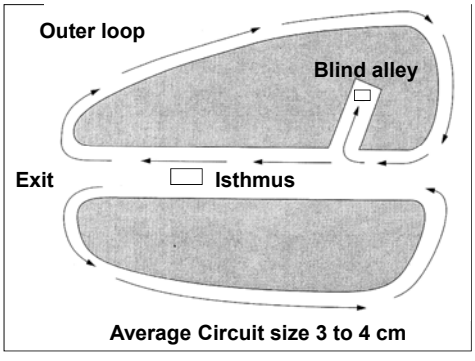
Scar Related VT's

- Healed MI
- Idiopathic dilated cardiomyopathy
- RV Dysplasia
- Hypertrophic cardiomyopathy
- Sarcoid
- Chagas disease
- Repaired Tetralogy of Fallot

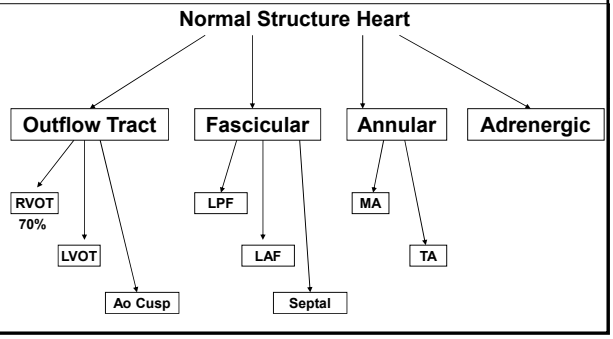
Ventricular Tacycardia (VT)
Classification by the heart structure

- Scar based VT
- Normal Heart Structure VT

VT Circuit – Ischemic VT



Idiopathic Ventricular Tachycardia Normal Structure Heart



Classification by VT Morphology

- **Monomorphic Vs. Polymorphic VT**
- **Torsades de pointes: polymorphic VT + long QT interval**
- **Bidirectional VT: (digitalis toxicity)**
- **Ventricular flutter is regular, rapid =300 bpm**
- **Ventricular fibrillation (VF): rapid, >300 bpm**

Mechanism of Idiopathic VT

- **Reentrant** → **Verapamil** sensitive
  Reproduced by program stimulation
  Can be **Cathecholamine-Sensitive**
- **Automaticity** → **Adrenergically** Mediated
  Usually Spontaneous
  Not induced by program stimulation
  Overdrive **Suppression**
- **Triggered Activity** → Mediated by stimulation of **c-AMP**
  Adenosine-Sensitive
  Delayed Afterdepolarizations (DADs)
  Catecholamine Sensitive
  Induced with Overdrive Pacing

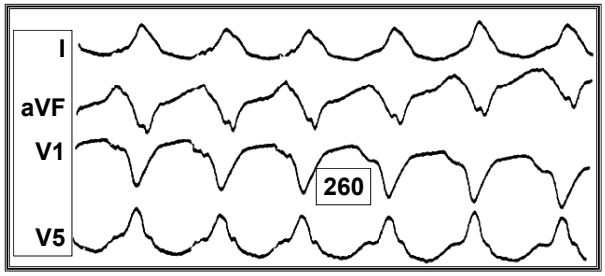
Classification by VT Duration

Sustained VT:
- **>30 seconds**
- **< 30 seconds need termination d/t hemodynamic instability**

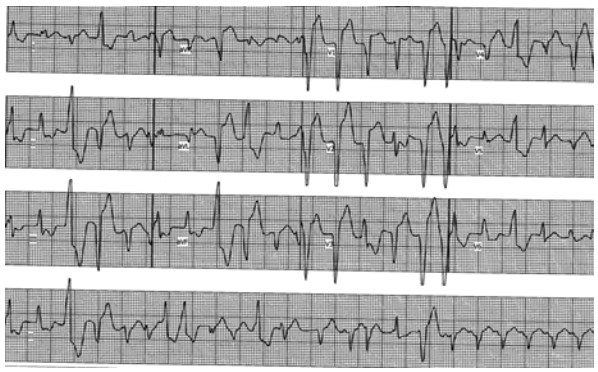
Nonsustained VT
- **> or = 3 beats VT (>100 beats/min)**
- **<30 seconds**

Incessant VT:
- **Sustained VT, recurrent post termination by cardioversion**
- **Repeated bursts runs of VT**

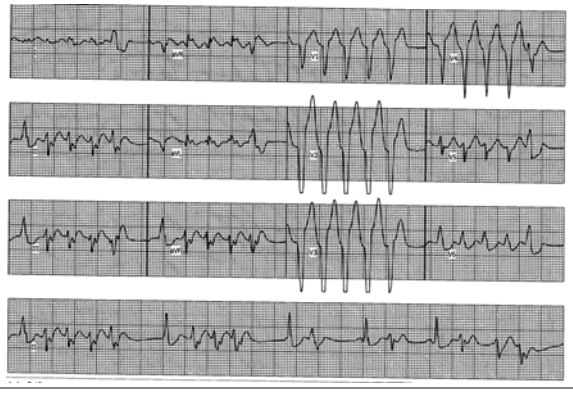
Sustained Monomorphic VT



Non-Sustained Polymorphic VT

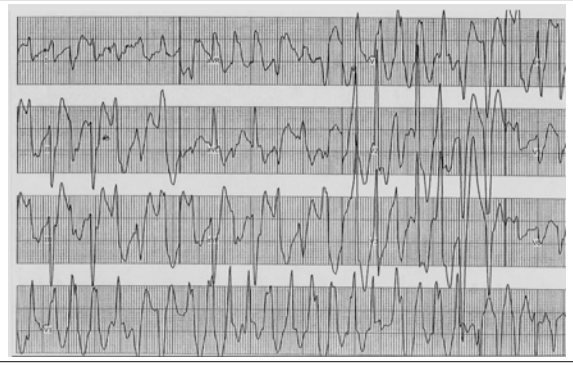


Non-Sustained Monomorphic VT



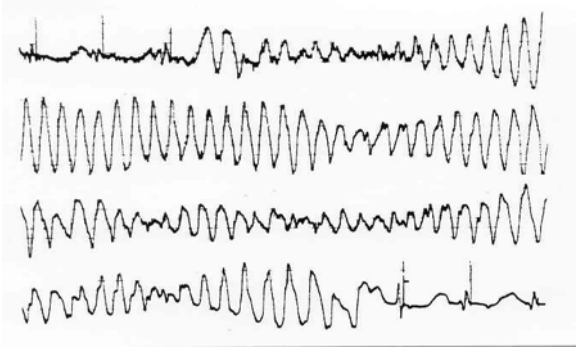
Sustained Polymorphic VT

Exercise induced in patient with no structural heart disease

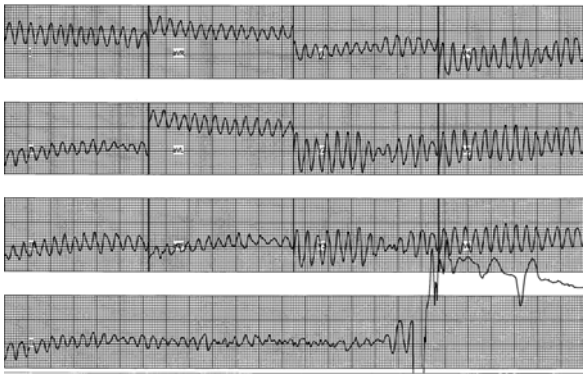


Torsades de Pointes

Spontaneous conversion to NSR (continuous lead II monitor strip)

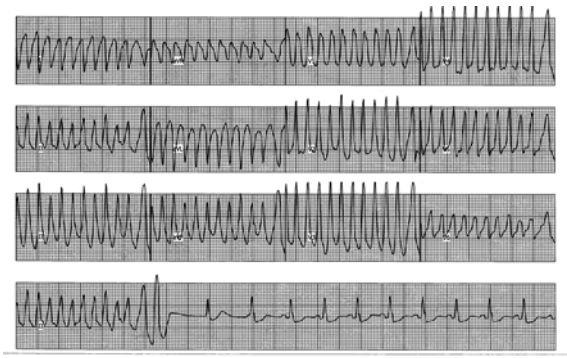


VF with Defibrillation (12-lead ECG)



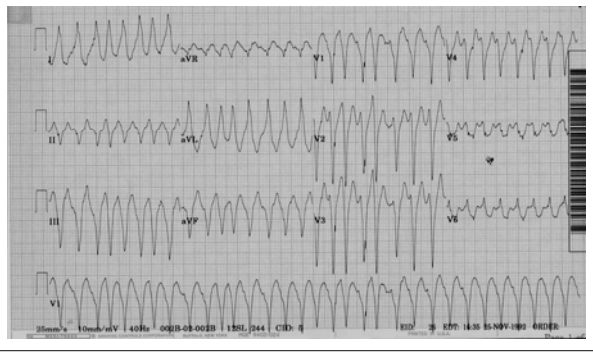
Ventricular Flutter

Spontaneous conversion of NSR (12-lead ECG)



Wide QRS Irregular Tachycardia:

Atrial Fibrillation with antidromic conduction in patient with accessory pathway – Not VT



Classification by Clinical Presentation

Hemodynamically stable

- ♥ **Asymptomatic**
- ♥ **Minimal symptoms, e.g., palpitations**

Hemodynamically unstable

- ♥ **Presyncope**
- ♥ **Syncope**
- ♥ **Sudden cardiac death**
- ♥ **Sudden cardiac arrest**

Mechanisms & Substrates of SCD

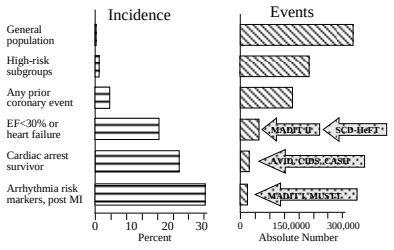
Mechanisms of Sudden Cardiac Death in 157 Ambulatory Patients

- Ventricular fibrillation - 62.4%
- Bradyarrhythmias (including advanced AV block and asystole) - 16.5%
- Torsades de pointes - 12.7%
- Primary VT - 8.3%

Bayes de Luna et al. Am Heart J 1989;117:151–9.

Epidemiology of VA & SCD

Incidence of Sudden Cardiac Death



Circulation 1992;85:12-10.

Clinical Presentations of VA & SCD

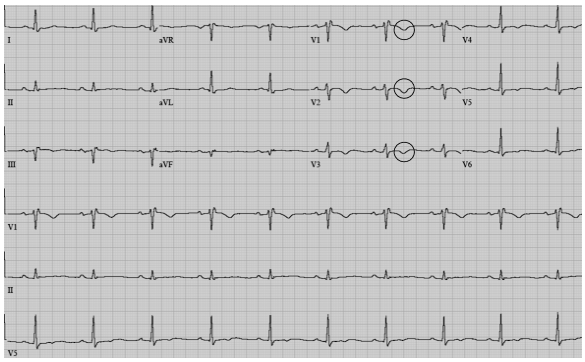
- **Asymptomatic** individuals +/- abnormal ECG
- Persons with symptoms potentially **attributable to VA**
  - ♥ Palpitations
  - ♥ Dyspnea
  - ♥ Chest pain
  - ♥ Syncope and presyncope
- VT that is hemodynamically **stable**
- VT that is **not hemodynamically** stable
- **Cardiac arrest**
  - ♥ Asystolic (sinus arrest, atrioventricular block)
  - ♥ VT
  - ♥ Ventricular fibrillation (VF)
  - ♥ Pulseless electrical activity

General Evaluation for Documented or Suspected VA

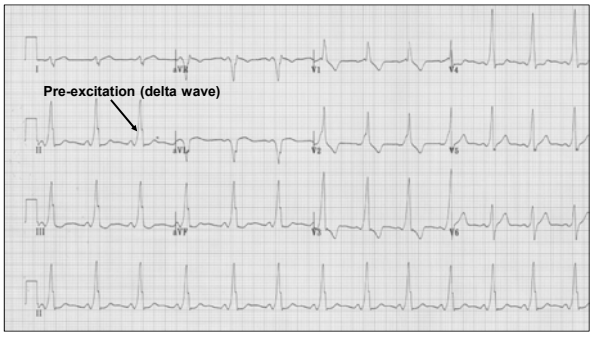
Resting Electrocardiogram

Resting 12-lead ECG is indicated in all patients who are evaluated for ventricular arrhythmias.

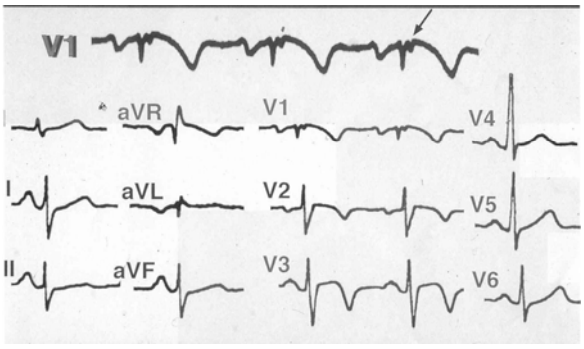
Arrhythmogenic RV Cardiomyopathy
(RV conduction delay, inverted T-waves V1-V5)



WPW ECG pattern
notice short PR interval and delta wave

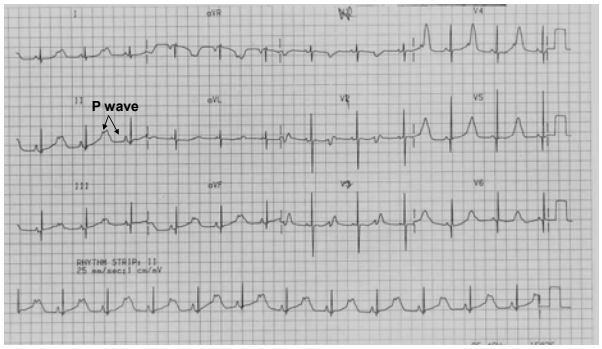


Arrhythmogenic RV Cardiomyopathy
12-lead ECG showing Epsilon wave



Long QT Syndrome in a 16-year-old girl

QT=520 ms; Atrial Tachycardia with 2:1 AV conduction



General Evaluation for Documented or Supected VA

Exercise Testing

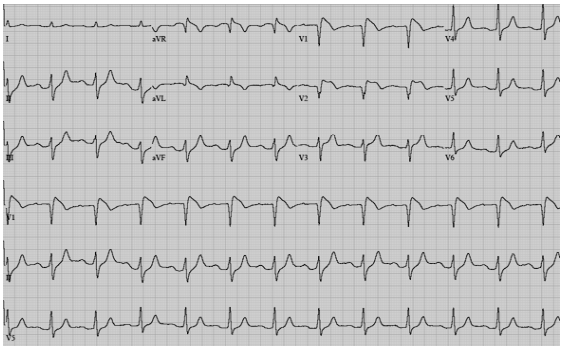
Intermediate or greater probability of having CAD by symptoms to provoke ischemic changes or VA

Known or Suspected exercise-induced VA, including catecholaminergic VT, to *provoke* the arrhythmia, achieve a *diagnosis*, and determine the *patient's response* to tachycardia.

Response to medical or ablation therapy in patients with known exercise-induced ventricular arrhythmias

Brugada Syndrome

(Typical ST-T abnormality V1-V2)



General Evaluation for Documented or Supected VA

Ambulatory Electrocardiography

QT- interval changes, T-wave alternans (TWA), or ST changes to evaluate risk, or to judge therapy.

Event monitors are indicated when symptoms sporadic

Implantable recorders are useful in patients sporadic syncope when a symptom-rhythm correlation cannot be established.

General Evaluation for Documented or Supected VA

Electrocardiographic Techniques

T-wave alternans: risk stratification for VA or who are at risk for developing life-threatening ventricular arrhythmias. (-ve predictive value)

Signal-averaged ECG (SAECG)
Heart rate variability (HRV)
Baroreceptor reflex sensitivity
Heart rate turbulence
} may be useful ??

General Evaluation for Documented or Supected VA

Stress testing and Imaging (nuclear or echo)

- Detect silent ischemia in patients with VA
- ECG assessment is less reliable because of :
  1- Digoxin use
  2- LVH
  3- > 1-mm ST-segment depression at rest
  4- Wolf-Parkinson-White (WPW) syndrome
  5- Left Bundle Branch Block (LBBB).
  6- Paced ventricular rhythm.

General Evaluation for Documented or Supected VA

Echocardiography

Suspected of having structural heart disease.

Subset of patients at high risk for VA or SCD:

- Dilated, hypertrophic, or RV cardiomyopathies,
- AMI survivors.
- Relatives of patients with inherited disorders associated with SCD (channelopathy)

General Evaluation for Documented or Supected VA

Left Ventricular Function and Imaging

MRI, cardiac computed tomography (CT), or radionuclide angiography (muga scan) can be useful VA when *echocardiography does not provide* accurate assessment of left ventricular (LV) & RV function

Coronary angiography can be useful to assess for any significant *obstructive CAD* in life-threatening VA or in survivors of SCD

General Evaluation for Documented or Supected VA

Conditions Associated With VA That Can Be Diagnosed With Echocardiography

Disease Entity	Diagnostic Accuracy
Dilated cardiomyopathy	High
Ischemic cardiomyopathy	High
Hypertension with moderate to severe LVH	High
Valvular heart disease	High
Arrhythmogenic right ventricular cardiomyopathy (ARVC)	Moderate
Brugada syndrome	Poor

General Evaluation for Documented or Supected VA

Electrophysiological Testing in Patients With Syncope

1- Impaired LV function or structural heart disease.

2- Bradyarrhythmias or tachyarrhythmias are suspected and in whom noninvasive diagnostic studies are not conclusive.

General Evaluation for Documented or Supected VA

Electrophysiological Testing in CAD

- **Remote MI with symptoms suggestive of ventricular tachyarrhythmias, including palpitations, presyncope, and syncope.**
- **Assess the efficacy of VT ablation.**
- **Diagnostic evaluation of wide-QRS-complex tachycardias.**
- **Risk stratification in remote MI, NSTV, @ LVEF ≤ 40%.**

Management of VA

Acute Management of Specific Arrhythmias

Sustained Monomorphic VT (SMVT)

- ***Wide-QRS tachycardia*** **should be** ***presumed to be VT*** **if the Dx is unclear.**
- ***Cardioversion*** **is recommended in suspected SMVT with** ***hemodynamic compromise***
- **Intravenous** ***procainamide*** **is reasonable for** ***stable*** **SMVT**

Acute Management of Specific Arrhythmias

Sustained Monomorphic VT (SMVT)

- ***Intravenous lidocaine*** **might be reasonable SMVT in acute myocardial ischemia or infarction.**
- ***Verapamil and diltiazem*** **should** ***not*** **be used in patients to terminate wide-QRS- complex tachycardia of unknown origin, especially history of myocardial dysfunction.**

Acute Management of Specific Arrhythmias

Sustained Monomorphic VT (SMVT)

- **Intravenous** ***amiodarone*** **is reasonable in SMVT:**
  1. **Hemodynamically unstable**
  2. **Refractory to cardioversion**
  3. **Recurrent despite antiarrhythmic medications**
- ***Transvenous catheter pace*** **termination can be useful in:**
  1. **Refractory to cardioversion.**
  2. **Recurrent despite antiarrhythmic medication.**

Acute Management of Specific Arrhythmias

Polymorphic VT (PMVT)

- ***Direct-current cardioversion*** **is recommended PMVT with hemodynamic compromise.**
- ***Intravenous beta blockers*** **are useful in recurrent PMVT, especially if ischemia is suspected.**
- **Intravenous amiodarone can be used in no congenital or acquired LQTS exist.**

Acute Management of Specific Arrhythmias

Torsades de Pointes (Tdp)

- ***Withdrawal*** **of any offending drugs**
- ***Correction*** **of electrolyte abnormalities**
- **Acute and long-term pacing**
  - **Heart block**
  - **Symptomatic bradycardia**

Therapies for VA

- **Beta Blockers: Effectively suppress PVC & arrhythmias; reduce incidence of SCD**
- **Amiodarone: No definite survival benefit; Has complex drug interactions and many adverse side effects (pulmonary, hepatic, thyroid, cutaneous)**
- **Sotalol: pro-arrhythmic > amiodarone, no survival benefit**
- **Antiarrhythmic drugs (except for BB) should not be used as *primary* therapy of VA and the prevention of SCD**

Acute Management of Specific Arrhythmias

Torsades de Pointes (Tdp)

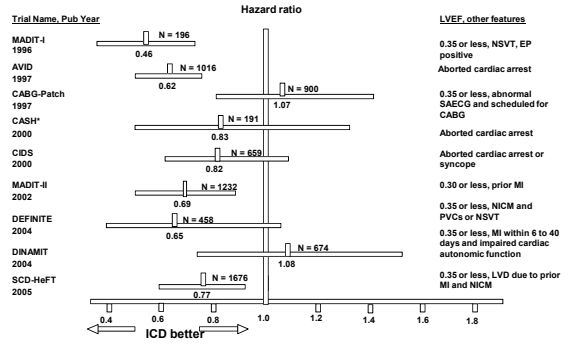
- ***Intravenous Magnesium sulfate*** **is effective in *LQTS* and few episodes of Tdp.**
- ***Acute and long-term pacing*** **is reasonable in *recurrent pause- dependent*** **Tdp.**
- ***Isoproterenol*** **is reasonable as temporary treatment in *recurrent pause- dependent* *do not*** **have congential LQTS.**

Therapies for VA

- **Electrolytes: esp in setting of *hypo*magnesemia and *hypo*kalemia**
- **ACE inhibitors, ARB and aldosterone blockers can improve the myocardial substrate through reverse remodeling**
- **Antithrombotic and antiplatelet agents: reducing coronary thrombosis**
- **Statins: have been shown to reduce life-threatening VA in high-risk patients with electrical instability**
- **n-3 Fatty acids: conflicting data exist for the prevention of SCD**

Therapies for VA

ICDs: Results from Primary and Secondary Prevention Trials



Therapies for VA

Primary Prevention of SCD

Nonischemic cardiomyopathy, LVEF ≤ 30%, NYHA class II, III

Nonischemic cardiomyopathy, LVEF 30-35%, NYHA class II, III

Therapies for VA

Primary Prevention of SCD

LV dysfunction due to **MI**, LVEF ≤ 30%, NYHA class I

LV dysfunction due to **MI**, LVEF ≤ 31-35%, NYHA class I

LV dysfunction due to **MI**, LVEF ≤ 30%, NYHA class II, III

LV dysfunction due to **MI**, LVEF 30-35%, NYHA class II, III

LV dysfunction due to **MI**, LVEF 30-40%, NSVT, positive EP study

Therapies for VA

Ablation

- **Low risk for SCD and have sustained predominantly monomorphic VT (drug *resistant*, drug *intolerant*, *do not wish* long-term drug therapy)**
- ***Multiple appropriate ICD shocks* due to VA.**

  (*not manageable* by reprogramming or drug *change*, and *do not wish* long-term drug therapy)
- **WPW syndrome resuscitated from *sudden cardiac arrest* due to AF and *rapid conduction* over the *accessory pathway causing VF*.**

Therapies for VA

Ablation

- Low risk for SCD & symptomatic non-sustained monomorphic VT (drug *resistant*, drug *intolerant*, *do not wish* long-term drug therapy)
- Low risk for SCD and frequent symptomatic monomorphic PVCs (drug *resistant*, drug *intolerant*, *do not wish* long-term drug therapy)
- Asymptomatic frequent PVCs may be *considered* to *avoid* or *treat* tachycardia-induced cardiomyopathy (TIC)
- *Bundle-branch reentrant VT*.

Risk Factors for Sudden Cardiac Death in Hypertrophic Cardiomyopathy

Major Risk Factors

- ✓*Cardiac arrest* (VF)
- ✓*Spontaneous* sustained VT
- ✓*Family history* of premature sudden death
- ✓*Un*explained syncope
- ✓LV thickness >/= 30 mm
- ✓*Abnormal* exercise BP
- ✓*Non*-sustained spontaneous VT

Maron BJ et al. J Am Coll Cardiol 2003;42:1687-713.

VA Associated With Cariomyopathies

Arrhythmogenic Right Ventricular Cardiomyopathy (ARVC)

- ICD implantation is recommended for the *prevention of SCD* in ARVC with *documented sustained VT or VF*
- ICD implantation can be effective for the prevention of SCD in ARVC
  - 1- *Extensive* disease
  - 2- *1 or more affected family* member with SCD
  - 3- Undiagnosed *syncope*

Arrhythmogenic Right Ventricular Cardiomyopathy (ARVC)

- **Amiodarone or sotalol *can be effective* in ARVC when ICD implantation is not feasible.**
- ***Ablation* can be useful as *adjunctive* therapy in ARVC with recurrent VT**

Long QT Syndrome

- ***Lifestyle modification* is recommended for LQTS patients**
- **Beta blockers are recommended in the presence of prolonged QT**
- **Implantation of an ICD + beta blockers is recommended for *LQTS* patients with previous *cardiac arrest***

Genetic Arrhythmia Syndromes

Brugada Syndrome

- ***An ICD* is indicated for *Brugada* syndrome patients with *previous cardiac arrest***
- ***An ICD* is reasonable for *Brugada* syndrome patients with *spontaneous* ST-segment elevation in V_1, V_2, or V_3 who have had *syncope***

Drug Interactions Causing Arrhythmias

Drug Interactions Causing Arrhythmias

Drug	Interacting Drug	Effect
Beta blockers propafenone	Quinidine (even ultra-low dose) Fluoxetine	Increased beta blockade Increased beta blockade
Flecainide	Some tricyclic antidepressants	Increased adverse effects
Dofetilide	✓ ***Verapamil (not Diltiazem)*** ✓ ***Cimetidine*** ✓ ***Trimethoprim*** ✓ Ketoconazole ✓ Megestrol	Increased plasma dofetilde concentration

Data from Roden DM. Proarrhythmia. In: Kass RS, ed. Handbook of Experimental Pharmacology: vol. 171. Boston: Springer Verlag, 2006:288–304.

Drug Interactions Causing Arrhythmias

Drug	Interacting Drug	Effect
Digoxin	Some antibiotics	Eliminating gut flora that metabolize digoxin
Digoxin	✓Amiodarone ✓Quinidine ✓Verapamil	Increased digoxin bioavailability, reduced biliary and renal excretion due to P-glycoprotein inhibition
	▪*Cyclosporine* ▪*Itraconazole* ▪*Erythromycin*	Digoxin toxicity
Quinidine	Ketoconazole	Increased drug levels
Cisapride	Itraconazole	
Terfenadine	Erythromycin*	
astemizole	Clarithromycin Some calcium channel blockers* Some HIV protese inhibitors (especially ritanovir)	

*These may also accumulate to toxic levels with co-administration of inhibitor drugs like ketoconazole. Data from Roden DM. Proarrhythmia. In: Kass RS, ed. Handbook of Experimental Pharmacology: vol. 171. Boston: Springer Verlag, 2006:288–304.

Drug Interactions Causing Arrhythmias

Drug	Interacting Drug	Effect
QT-prolonging antiarrhythmics	Diuretics	Increased T de P risk due to diuretic-induced hypokalemia
Beta blockers	Amiodarone, clonidine, digoxin, dilitiazem, verapamil	Bradycardia when used in combination
Digoxin	Amiodarone, beta blockers, clonidine, dilitiazem, verapamil	
Verapamil	Amiodarone, beta blockers, clonidine, digoxin, dilitiazem	
Diltiazem	Amiodarone, beta blockers, clonidine, digoxin, verapamil	
Sildenafil	Nitrates	Increased and persistent vasodilation; risk of myocardial ischemia
Clonidine	Amiodarone, beta blockers, digoxin, dilitiazem, verapamil	
Amiodarone	Beta blockers, clonidine, digoxin, dilitiazem, verapamil	

Examples of Drugs Causing Torsades de Pointes

Frequent (greater than 1%)*	Less Frequent
•Disopyramide	•*Amiodarone*
•Dofetilide	•Arsenic trioxide
•Ibutilide	•Bepridil
•Procainamide	•*Cisapride*
•Quinidine	•*Anti-infectives:* clarithromycin, erythromycin, halofantrine; pentamidine, sparfloxacin
•Sotalol	•*Antiemetics:* domperidone, droperidol
•Ajmaline	•*Antipsychotics:* chlorpromazine, haloperidol, mesoridazine, thioridazine, pimozide
	•Opioid dependence agents: methadone

* (e.g., hospitalization for monitoring recommended during drug initiation in some circumstances)

Roden DM. N Engl J Med 2004;350:1013-22.

Risk Factors for Drug-Induced Torsades de Pointes

- Female gender
- Hypokalemia
- Bradycardia
- Recent conversion from atrial fibrillation
- Congestive heart failure
- Digitalis therapy
- Severe hypomagnesemia
- Congenital long QT syndrome
- Baseline QT prolongation

Roden DM. N Engl J Med 2004;350:1013-22.