Sudden Cardiac Death: Who Is At Risk and What Can We Do?

Silence cell phones please!

My own near syncopal episodes!!!
Definition

• Unexpected death caused by cardiac causes that occurs within 1 hour of symptom onset

• Describes sudden collapse, loss of consciousness and loss of effective circulation that preceded biologic death

Incidence of Sudden Cardiac Death: Lowest to Highest

• General population
• High risk sub groups
• Any prior coronary event
• EF < 30% or HF
• Cardiac arrest survivors
• Arrhythmia risk markers; post MI
Cause of Sudden Death in Sample of Ambulatory Patients

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


Best survival with VT / V Fib Arrest

Ventricular Arrhythmias
Mechanisms of VT

- Disorder of impulse initiation
  - Abnormal automaticity
    - Acute MI / Ischemia
    - Electrolyte abnormalities
    - Acid / base imbalance
    - Increased sympathetic tone
  - Triggered mechanism: disturbance in recovery or repolarization (less common)
    - Early after-depolarization
      - Long QT
    - Delayed after-depolarization
      - Digitalis toxicity

- Disorder of impulse conduction
  - Reentrant Circuit (most common)
    - Reentrant Circuit within Myocardium
    - Bundle Branch Reentrant
Polymorphic VT with normal QT:

- Seen frequently in ischemic conditions
- Compared to monomorphic VT associated with scar

Ventricular Flutter
Spontaneous conversion to NSR (12-lead ECG)
 VF with Defibrillation (12-lead ECG)

Associated Pathophysiology
2 classifications

Structural
- CAD
- Myopathy
- Valvular heart disease

Non-structural
Approximately 5% of SCD patients have no structural heart disease

Structural Heart Disease – CAD

- Coronary artery disease (CAD) present in 80% of SCD population
  - 50% of those have acute changes in coronary status (plaque rupture / thrombus)

- Acute Coronary Syndrome
  - Vulnerable plaque
  - Highest risk of mortality within first 24 to 48 hours
    - V fib arrest
Tachyarrhythmias and CAD

- Highest risk of SCD – severe LV dysfunction
- Suppression of asymptomatic NSVT
  - No evidence of decreased mortality
- Sustained VT
  - Treatment guided by frequency and symptoms
- V-fib arrest not within 24 to 48 hours of event
  - Risk of recurrent arrest
- Electrophysiology study
  - Done after ischemic evaluation
  - Major goal to identify presence of ventricular tachyarrhythmias
  - ICD indication
    - CAD and syncope and inducible monomorphic VT (LVEF not a factor)

Ventricular Arrhythmia and Sudden Death related to Prior MI (Class I)

- Aggressive attempts to treat HF and ischemia
- Revascularization
- If revascularization cannot be done – ICD as primary therapy
- ICD in LV dysfunction from prior MI who present in unstable sustained VT
- ICD for primary prevention
  - 40 days post MI
  - LVEF ≤ 30-40%
Structural Heart Disease – Cardiomyopathy

• Cardiomyopathy
  – EF < 30% most powerful predictor of SCD
  – Left ventricular hypertrophy
    • Increases ventricular ectopy

Patients with Non Ischemic Dilated Cardiomyopathy

• Increased mortality with syncope
  – Tilt table testing not as useful
  – EP study to diagnose Bundle Branch Reentrant Tachycardia
• Self terminating episodes of VT most common cause
• No evidence for empirical treatment with antiarrhythmic therapy
• ICD reasonable in patients with syncope
• ICD for primary prevention of SCD
  • LVEF < 30% to 35%
  • NYHA Class II or III
Bundle Branch Reentrant Ventricular Tachycardia

A

B
Arrhythmogenic Cardiomyopathy

- Inherited muscle disorder
- Often referred to as arrhythmogenic right-ventricular dysplasia (ARVD)
- Manifest as an arrhythmia, heart failure, or sudden death
- Genetic characteristics include autosomal dominance inheritance (most common)
- Most frequently affects the right ventricle
- More often than thought also affects left ventricle
- More often males than females

Arrhythmogenic Cardiomyopathy Pathophysiology

Cardiomyocyte replaced with fibro fatty tissue
Initially patchy infiltration
Arrhythmogenic Cardiomyopathy Pathophysiology

- Progressive loss of muscle leads to thinning of the ventricular wall, dilation and pump dysfunction
- Thinnest portions of the right ventricle affected first
  - Triangle of dysplasia: Inflow, outflow, apical regions of RV

Arrhythmogenic Cardiomyopathy Disease Progression

- Four Phases
  - Early / Concealed phase
    - Subtle structural changes
    - Often asymptomatic
  - Overt Phase
    - Noticeable structural and functional changes
    - Palpitations, pre-syncope, syncope, arrhythmias
  - Impaired contractility and right-sided failure
    - Right ventricular dilation
    - Decreased contractility
    - Signs of right sided heart failure
  - Bi-ventricular failure
    - Disease spreads to left ventricle
    - Signs of biventricular failure
Arrhythmogenic Cardiomyopathy

Presentation

- Palpitations
- Presyncope
- Syncope
- Often episode of sudden cardiac death is first presentation
- Signs of heart failure are late sign

Diagnosis

- ECG
  - T Wave inversion in leads V1-V6
  - Epsilon wave
  - VT with LBBB pattern
  - Conduction delays through right bundle
- Endomyocardial biopsy
- Echocardiogram
  - RV enlargement and dysfunction
- MRI / CT
  - Detect fatty infiltrate

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Arrhythmogenic Cardiomyopathy Treatment

- No cure
- Goal: Manage arrhythmias
- Antiarrhythmics
- Implantable Cardiovertor Defibrillator
- Radiofrequency catheter ablation if unsuccessful in treating VT with antiarrhythmics
- Refrain from competitive / intense sports
- Screening of family members
  - 1\textsuperscript{st} and 2\textsuperscript{nd} degree relatives
Hypertrophic Cardiomyopathy

• 1 of every 500 (Maron et al, 2003)
• Primary genetic cardiomyopathy
• Effects men and women equally
• Hypertrophy of myocardial muscle mass
• Associated with decreased ventricular filling and decreased cardiac output
• Most common cause of sudden death in young adults
• Cause unknown
  – 50% transmitted genetically
Hypertrophic Cardiomyopathy

- Disarray of cardiac myofibrils with hypertrophy of myocytes
- Cells take on a variety of shapes
- Myocardial scarring and fibrosis occurs

![Normal Muscle Structure vs. Myocardial Disarray](image)

Hypertrophic Cardiomyopathy

- Usually only effects LV
- Changes may be symmetrical
- Asymmetrical septal hypertrophy is more common

![Heart with Hypertrophic Cardiomyopathy](image)
Hypertrophic Cardiomyopathy

- May involve entire septum or only a portion of septum

Hypertrophic Cardiomyopathy OBSTRUCTIVE

- 30-50% of HCM patients have obstruction
- Obstruction of outflow tract
- Septal wall enlarges into ventricular cavity
- Anterior leaflet of mitral valve drawn towards the septum during ejection
- Early closure of aortic valve, decreased ejection time, decreased cardiac output
Hypertrophic Cardiomyopathy

OBSTRUCTIVE

HCM During Relaxation

HCM During Active Contraction

SAM

Hypertrophic Cardiomyopathy Presentation

- Many asymptomatic for years
- Incidence of sudden death often first presentation
- Identified during screening of relative of patient with HCM
- Symptoms related to severity of diastolic dysfunction or mitral regurgitation
- Dyspnea / activity intolerance
- Palpitations
- Chest pain
- Syncope / Presyncope
- Sudden Death
Hypertrophic Cardiomyopathy 
Treatment

- Goals
  - Relief of symptoms
  - Preventing complications
  - Preventing or reducing risk of sudden death
  - No evidence to support treatment of non-symptomatic patients

- Beta blockers
  - 1st choice (with or without HOCM)
  - Symptomatic benefit / improved exercise tolerance
  - Changes in HR, contractility, and conduction

- Calcium Channel Blockers
  - If BB not effective
  - Use with caution due to vasodilatation

Hypertrophic Cardiomyopathy 
Treatment

- Antiarrhythmic Therapy
  - AF
    - Most common arrhythmia
  - Disopyramide
    - Negative inotrope and Class I antiarrhythmic
    - Decreases SAM
    - Decreases MR
    - Use with BB to assist in HR control
  - Amiodarone
    - Obstructive or non-obstructive OK
    - Ventricular or atrial arrhythmias
    - Anticoagulation

- Other Medications
  - Diuretics
    - With caution
  - ACE Inhibitors and NTG
    - Avoided in HOCM
  - Caution with all vasodilators
  - Positive Inotropes
    - Strictly avoid any medication that increases contractility in OBSTRUCTIVE disease
Hypertrophic Cardiomyopathy
Treatment - Surgical

- Ventricular Septal Myectomy
  - Marked outflow obstruction
  - On maximum medical therapy
  - NYHA Class III or IV
  - MV Replacement or repair at same time (increases operative mortality)
  - Improvement noted immediately and last 20-30 years
  - Survival Rates 80% at 10 years
  - May need pacer due to development of LBBB.

Hypertrophic Cardiomyopathy
Treatment - Surgical

- Percutaneous Alcohol Septal Ablation
  - Symptomatic with full therapy
  - NYHA Class III or IV
  - Not appropriate if MVR needed
  - Cath Lab Procedure
  - Catheter in septal perforator
  - Ethyl alcohol injected
  - Myocardial infarction occurs
  - Enlarged septum eventually shrinks
  - Better for patients > 55
  - Often need pacemaker
Hypertrophic Cardiomyopathy
Sudden Cardiac Death

- Risk for Sudden Death
  - One or more 1st degree relative with an episode of SCD
  - More common in patients under 40
  - Normally occurs during strenuous activity
  - Left ventricular wall thickness greater than 30-35 mm
  - Prolonged or repetitive non-sustained ventricular tachycardia on Holter monitor
  - Hypotensive BP response to exercise
  - Syncope or near syncope

Hypertrophic Cardiomyopathy
Activity

- No intense competitive sports
- Most SCD occurs with football or basketball
- No “burst” exertion
- No restrictions in patients with family history but no evidence of disease themselves
- SCD may occur at rest
Structural Heart Disease – Valvular and Congenital Heart Disease

• Valvular heart disease
  – Aortic valve replacement
    • Risk peaks at 3 weeks and plateaus at 8 months

• Congenital disease associated with SCD
  • Tetralogy of Fallot
    – VSD, Aorta lies over VSD, Pulmonic valve stenosis
      (other obstructions)
  • Transposition of great arteries
  • Aortic stenosis
    – Risk increases with exertion
  • Functional single ventricle

Myocarditis / Endocarditis

• TTVP with symptomatic bradycardia or HB during acute phase of myocarditis
• Acute aortic regurgitation associated with VT should be surgically treated
• Acute endocarditis complicated by aortic or annular abscess and AV block should be surgically treated

Renal / Endocrine Disease

• Treat potassium, magnesium, and calcium
• VT and SCD can be induced by excess or insufficient hormone activity on myocardial receptors
  • Hypothyroidism
  • Pheochromocytoma
Other Causes of SCD

- Cerebral or subarachnoid hemorrhage
- Choking
- Pulmonary HTN
- Pulmonary embolus
- SIDS
- Obesity / dieting / anorexia

- Dissection of aortic aneurysm
  - Clinical Pearl:
    - Diastolic murmur
    - Blood pressure / pulse assessment

- Digitalis Toxicity
  - Clinical Pearl
    - If in doubt hold

Close Up Look At Arrhythmogenic Causes
Torsades De Pointes

- Recognition of this life-threatening arrhythmia is important because it is not treated like other VTs
- Two groups: Acquired and congenital
- Acquired
  - Drugs prolonging repolarization (class 1a, 1c, and sotolol)
  - Electrolyte abnormalities
  - Severe bradycardias
- Congenital

Acquired Torsades De Pointes

- Warning Signs:
  - QT prolongation
    - Usually greater than 0.5 sec
    - Rate adjustment QTc
  - T Wave aberration
  - Prominent U waves
  - Pause dependent couplets
- Short bursts: QRS peaks first appear to be up and then to be down (Can degenerate into V fib)
Emergency Treatment for Acquired Torsades de Pointes

- Immediate discontinuations of all agents that may be potentially responsible
- IV potassium is given to correct electrolyte abnormalities
- IV magnesium is regarded as the treatment of choice for TdP and is suggested even in normomagnesemia
- If IV magnesium is unsuccessful, overdrive ventricular pacing or medication to increase HR may be necessary
- DC cardioversion is usually transiently effective in terminating TdP.

  - Note: Class IA drug induced TdP usually appears soon after the initial administration of the drug

Case Example
Cardiac Ion Channel Abnormalities

• Long QT Syndrome (LQTS)
• Brugada disease

  – Diagnosed by family history and ECG

Congenital LQTS

• QTc > 450 ms
• Genetic defect in either potassium (LQT1 or LQT2) or sodium (LQT3) channels
  – Delayed repolarization (1 and 2)
  – LQT1 and LQT2 = 95%
    • Beta blockers
  – LQT3 = 5%
    • Beta blockers may be harmful
• Autosomal dominant trait
• QT prolongation important risk factor for SCD
  • QTc < 440 ms / < 5%
  • QTc 460 to 500 ms / 20%
  • QTc > 500 ms / 50%
**LQTS**

- **Syncope**
  - Ominous
  - Secondary to torsades de pointes
    - Occurs with increase in sympathetic tone
    - Usually terminates spontaneously (can degenerate into VT)

- **Treatment**
  - Family screening
  - Beta-blockers / pacemakers
  - ICDs
  - Avoid strenuous exercise
  - Avoid medications that prolong QT
    - www.QTdrugs.org
    - www.torsades.org

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**Brugada Syndrome**

- Disorder of cardiac sodium channel
  - Reduction of sodium current available during Phase 0 of action potential
- Autosomal dominant
- ST elevation in anterior precordial leads
  - Cove
  - Saddleback
- ECG can be dynamic
- In children temperature spike may uncover
  - Treat aggressively
- Syncope
  - 2 year risk of SCD approximately 30%
- ICD recommended
Most common in Asia
Typical patient young male age 30 to 50 who is otherwise healthy.
Wolff Parkinson White Syndrome

- Termed Pre-excitation because some conduction occurs via the Kent bundles in addition to the normal pathway; because conduction via the Kent bundles is faster than via the AV node the ventricles are pre-excited

- This produces a “delta wave” on the EKG
  - Short PR
  - Wide QRS

Delta Wave of Pre-excitation Syndrome

- 60 to 70% of WPW shows evidence in SR

- Left sided accessory pathway: Positive delta wave in V1
- Right sided accessory pathway: Negative delta wave in V1
Conduction over an accessory pathway

**Manifest accessory pathway**

- PR ≤ 120 ms
- Delta wave present
- QRS ≥ 120 ms
- Repolarization abnormal

**Concealed accessory pathway**

- PR normal
- Delta wave absent
- QRS normal
- Repolarization normal

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**Conduction over an accessory pathway**

- Conduction over AV connection (no complete conduction)
- Fusion of conduction over AV connection and delta wave over AV node (QRS normal)
- Delta wave present
- More of ventricle activated via the AV connection (QRS exit width)
- Maximal preexcitation
- Ventricle activated only by AV connection (assembles PVC)
Arrhythmias of WPW (CMT)

Orthodromic SVT

Antidromic SVT

Atrial fibrillation

Orthodromic Tachycardia

Negative P' in lead 1 = left sided accessory pathway

Positive P' in lead 1 = right sided accessory pathway
Antidromic Tachycardia

WPW and Atrial Fibrillation

- Mechanism of Action
  - Development of Atrial Fibrillation in WPW
  - Refractory period of accessory pathway
- Danger
Wide QRS Irregular Tachycardia:  
Atrial Fibrillation with antidromic conduction in patient with accessory pathway – Not VT

Treatment for Atrial Fib in WPW

- Atrial Fib with antegrade conduction over accessory pathway

Slow conduction over accessory pathway:
- Amiodarone
- Procainamide
- Flecainide
- Sotalol
- Propafenone
Evaluation

Syncope: A Transient Loss of Consciousness

- Neurocardiogenic (most common)
  - Vasovagal
  - Absence of prodrome points to cardiac arrhythmia or central neurodegenerative disorder with autonomic failure
- Malignant Syncope:
  Syncope that occurs with little or no warning and results in significant injury or damage to property.
- Clinical application: Prodrome with vasovagal

- Cardiovascular origin common
  - Underlying structural heart disease
  - Transient ischemia
  - Wolff-Parkinson-White syndrome
  - Long QT syndrome
  - Brugada syndrome
  - Catecholaminergic ventricular tachycardia
  - Clinical application: Arrhythmias: Fast on and off
  - Clinical application: Seizure slow offset

- High rate of mortality
Goals for Patient Evaluation

• #1: Determine if patient at increased risk for death
• Identify cause of syncope
  – Improve quality of life
  – Prevent injury to self and others

* Mechanism of syncope unexplained in 40% of cases

• ECG / Monitoring
  – Gold standard for diagnosing an arrhythmogenic cause of syncope is to have ECG documentation of the rhythm disturbance at the time of syncope
• Echo
• Stress
• Tilt Table
  – Value in malignant syncope or structurally normal heart
General Considerations for SCD

- CPR / AED
  - Practice example success story
- Post resuscitative efforts
  - Induced mild hypothermia
Clinical Considerations

- **Reversible Causes**
  - Electrolyte abnormalities
  - Volume depletion
  - Hypemia / hypoxia
  - Mechanical factors

- **Obstructions to Cardiac Output**
  - HOCM / Aortic stenosis
    - Murmur assessment
  - Pulmonary embolus
    - Identification of risk factors
  - Tamponade
    - Beck’s Triad
  - Tension Pneumothorax
    - Barotrauma
    - Lung sounds

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Antiarrhythmic Therapy in Ventricular Arrhythmias and Sudden Death

- **Beta Blockers**
  - Suppresses ventricular arrhythmias
  - Reduces incidence of SCD

- **Amiodarone**
  - Suppresses ventricular arrhythmias
  - No definite survival benefit
  - Complex drug interactions and many adverse side effects

- **Sotalol**
  - Suppresses ventricular arrhythmias
  - No definite survival benefit
  - More pro-arrhythmic than amiodarone
Antiarrhythmic Therapy

- Antiarrhythmics (excluding beta blockers) not used as primary therapy for prevention
- Patients with VT who do not meet criteria for ICD
  - Beta-blocker first line
  - Amiodarone or sotalol if not effective
- Patients with ICD with recurrent VT
  - Sotalol or
  - Combination of amiodarone and beta-blocker

Patients Not Requiring Antiarrhythmics

- Because of proarrhythmias or exacerbations of existing arrhythmias antiarrhythmic therapy not indicated for:
  - Asymptomatic atrial ectopy and unsustained SVT
  - Asymptomatic ventricular ectopy without runs of VT
  - Simple ventricular ectopy in AMI with no hemodynamic compromise
  - Asymptomatic unsustained VT with no structural heart disease
  - Asymptomatic WPW without known SVT
  - Mildly symptomatic simple atrial or ventricular ectopy
Antiarrhythmic Medications Effecting the Action Potential

- **Class I** – Fast sodium channel blockers
  - IA: Quinidine, Procainamide, Disopyramide
  - IB: Lidocaine, Mexiletine, Tocainide
  - IC: Flecainide, Propafenone

- **Class III** – Potassium channel blockers
  - Amiodarone, Ibutilide, Dofetilide, Sotalol

- **Class IV** – Calcium channel blockers
  - Verapamil, Diltiazem

Class II ???

Class IV: Calcium Channel Blockers

Class III: K⁺ Channel Blockers

Class I

Na⁺ Channel Blockers

Marked prolongation of refractory period (prolong QT interval).

Slow conduction (widen QRS).
Some prolongation of refractory period (prolong QT interval).
Arrhythmias with ACS

• V-fib early in ACS
  • Increase hospital mortality
  • No increase in long term mortality

• Lidocaine prophylaxis
  • Decrease V-fib
  • Increase mortality

• Beta-blockers prophylaxis
  • Decrease V-fib

• Correction of potassium and magnesium

Monomorphric VT

• DC cardioversion with sedation if unstable
• IV procainamide
  • Stable VT
  • Caution with CHF or severe LV dysfunction

• IV amiodarone
  • Hemodynamically unstable
  • Refractory to shock

• TTVP for pace termination
• Lidocaine if ischemia
• Class III: Calcium channel blockers in wide complex of unknown origin; especially if myocardial dysfunction
Polymorphic VT

- DC cardioversion with sedation when unstable
- IV beta-blockers if ischemia suspected
  - Improve mortality
- IV amiodarone in absence of abnormal repolarization
- Urgent angiography to exclude ischemia
- Lidocaine may be reasonable if ischemia suspected

Incessant VT – VT Storm

- Class I
  - Revascularization and beta-blocker
  - Followed by IV amiodarone or procainamide
    (if due to acute ischemia)

- Class IIa
  - IV amiodarone or procainamide followed by VT ablation
  - Important to understand substrate to target treatment
Management Options for VT Storm

- Shock may beget shock
  - Spontaneous calcium release
- Reprogram ICD (stop pacing)
- IV Beta Blockers
  - Amiodarone / Lidocaine
- Revascularization
- IABP
- Deep sedation / general anesthesia
- Sympathectomy
  - Thoracic epidural anesthesia (T1-T2)
  - Other approach
- Catheter ablation

Other Pharmacotherapy

♥ Electrolytes:
  ✓ Magnesium and potassium especially if hypomagnesemia and hypokalemia
♥ ACE inhibitors, angiotensin receptor blockers and aldosterone blockers
  ✓ Reverse remodeling and thus reduce incidence of SCD
♥ Antithrombotic and antiplatelet agents
  ✓ Reduce coronary thrombosis and therefore SCD
♥ Statins
  ✓ Reduce life-threatening arrhythmias in high-risk patients
♥ n-3 Fatty acids
  ✓ Anti-arrhythmic properties, conflicting data for the prevention of SCD
ICD Therapy

- Reduction in mortality compared to drug therapy
  - 25-55% reduction in mortality compared to drugs
- Primary prevention
- Secondary prevention
- Assumed Criteria
  - Receiving optimal medical therapy
  - Expectation of survival with good functional status for > 1 year
  - Meets specific criteria for disease state

ICD Device

- Pulse Generator
  - Single chamber, dual chamber, or biventricular pacing
  - Back up pacing
  - Antitachycardia pacing
  - Implanted subcutaneously – same as pacemaker
- Defibrillator lead
  - Detects arrhythmias
  - Delivers therapy
  - Defibrillator lead capable of pacing and defibrillating
  - Placed in right ventricle
ICD Rhythm Detection

• **Heart Rate**
  - Monitors ventricular rate and delivers therapy when rate exceeds programmed tachycardia detection rate
  - Defined rate boundaries
    • Tachycardia zones

• **Sudden Onset**
  - Detects sudden shortening of cycle length

• **Interval stability**
  - Looks for variability in cycle lengths
  - Differentiates regular from irregular rhythms

• **Morphology**
  - Measures width of electogram
  - Only treats if width is greater than programmed value

Therapy: ATP-Antitachycardia Pacing

BURST

RAMP
Therapy: Cardioversion Shock

- Delivers shocks from 0.1 to 30 joules synchronized on the R wave

Therapy: Defibrillating Shock

- Delivers high energy (20-34 joules) unsynchronized shock for VF
Complications Related to Pacemaker / ICD

- Complication of subclavian vein stick
  - Pneumothorax
  - Hemothorax
  - Subclavian artery puncture
  - Air embolism
  - Bleeding
  - Brachial plexus injury
    - Pain or paresthesias in arm, hand, finger

- Complications related to pulse generator
  - Pocket erosion
  - Pocket hematoma
  - Infection
  - Generator migration
  - Generator malfunction
  - Premature battery depletion

Complications Related to Pacemaker / ICD

- Complications Related to Leads
  - Perforation of RV, subclavian veins
  - Lead dislodgement
    - Twiddlers Syndrome
  - Insulation breaks, lead fracture
  - Diaphragmatic stimulation
  - Venous thrombus
  - Pulmonary embolus

- Other Problems
  - Shocks for non VT rhythms
  - Failure to deliver therapy when needed
  - Ineffective therapy
  - High defibrillation thresholds
  - Device deactivation
Ablation

- Ischemic / Scar VT
- Bundle Branch Reentrant VT
- Idiopathic VT (focal origin)
- Accessory pathway in WPW

- Treatment of choice in idiopathic VT
- Adjunct to ICD when patient receiving multiple shocks
  - Incessant drug refractory VT
Ablation Overview

Ablation

- Radiofrequency Ablation
  - Ischemic heart disease: PREFER STABLE SCAR
    - Scar Tissue: Monomorphic
    - Ischemia: Polymorphic
  - BBB reentry
  - Idiopathic VT
    - Single morphology of PVCs

- Direct Surgical Ablation or Resection of Arrhythmogenic Focus
  - VT refractory to drugs, ICD, ablation
  - Pre-op or intraoperative mapping / scar based approach

Ischemic:
Endocardial Ablation
Non Ischemic:
Can do Epicardial Ablation
Ablation for Ischemic VT

- Endocardial and epicardial mapping to identify scarred areas
  - Challenges of epicardial mapping
    - Epicardial fat
    - Coronary arteries
- Non invasive imaging (MR) pre ablation to help locate area of scar
- Two strategies for ablation:
  - The first strategy involves induction of ventricular tachycardia, mapping and targeted ablation. *In those patients who have stable VT, it is possible to map the heart during stable VT, identify the critical circuit, and eliminate it; but only about 10% of patients actually have hemodynamically stable VT.*
  - The second strategy involves substrate mapping in sinus rhythm and linear ablation to eradicate regions of abnormal myocardium with the potential to facilitate reentry
  - Most centers now use a combination of these two approaches.

Complications of Ablation

- Perforation
- Tamponade
- Venous thrombosis
- Pulmonary embolism
- Pneumothorax
- Infection – sepsis
- Bleeding
- Damage to coronary arteries
Other Treatments

- Left cervicothoracic sympathetic ganglionectomy
  - Long QT syndrome

- Revascularization
  - Obstructive heart disease (left main or proximal LAD)

Final Case Study: ECG 1
Final Case Study: ECG #2

Final Case Study: ECG #3
Final Case Study: ECG #4

Final Case Study: ECG #5
Final Case Study: ECG #6

Find Joy in Our Work

Thank you!

Handouts available at
www.cardionursing.com