Ventricular Arrhythmias

Ventricular arrhythmias are potentially life-threatening cardiac rhythm disturbances originating from the ventricles, ranging from benign premature ventricular contractions (PVCs) to fatal ventricular fibrillation (VF). This guide students with a comprehensive framework to evaluate, diagnose, and manage ventricular arrhythmias in the hospital setting, with case scenarios to apply the knowledge.

Introduction and Pathophysiology

Ventricular arrhythmias arise from abnormal electrical activity in the ventricular myocardium, often due to reentry, enhanced automaticity, or triggered activity. They can lead to hemodynamic instability, syncope, or sudden cardiac death (SCD) if untreated. The severity depends on the type, underlying cause, and patient's cardiac function.

The pathophysiology involves:

Reentry: Most common mechanism; requires a circuit with differing conduction properties (e.g., scar tissue in ischemic heart disease).

Automaticity: Abnormal pacemaker activity in ventricular myocytes (e.g., in electrolyte imbalances).

Triggered Activity: Afterdepolarizations causing ectopic beats (e.g., early afterdepolarizations in long QT syndrome).

Ventricular arrhythmias are classified by type, duration, and clinical impact, requiring urgent management in the hospital setting to prevent adverse outcomes.

Indications for Hospital Management

Severe Manifestations:

- Sustained Ventricular Tachycardia (VT):
- Persistent VT causing hemodynamic instability (hypotension, chest pain, syncope).
- Ventricular Fibrillation (VF):
- Chaotic rhythm leading to cardiac arrest; requires immediate resuscitation.
- Frequent PVCs/VT in Structural Heart Disease:
- Risk of deterioration into VF or cardiomyopathy.

Symptoms:

- Syncope:
  - Indicates significant arrhythmia burden or risk of SCD.
- Heart Failure Symptoms:
  - Dyspnea, fatigue, or worsening ejection fraction (EF) due to arrhythmia-induced cardiomyopathy.
- Palpitations with Hemodynamic Compromise:
  - Suggests need for acute intervention.

Underlying Conditions:

- Acute Coronary Syndrome (ACS):
  - VT/VF in the setting of myocardial infarction (MI).
- Electrolyte Imbalances:
  - Severe hypokalemia, hypomagnesemia, or hyperkalemia.
- Drug Toxicity:
  - Antiarrhythmic toxicity (e.g., amiodarone, sotalol) or QT-prolonging drugs.

Other:

- Need for EP Ablation or ICD Placement:
  - Patients with recurrent VT or high SCD risk.
- Monitoring Post-Resuscitation:
  - After VF arrest to assess for recurrence or underlying cause.

Evaluation

History:

- Symptoms:
  - Palpitations, syncope, chest pain, dyspnea, or cardiac arrest.
- Risk Factors:
  - History of MI, heart failure (HF), cardiomyopathy, family history of SCD, or inherited arrhythmia syndromes.
- Systemic Symptoms:
  - Fatigue, weight gain (HF exacerbation), or symptoms of electrolyte imbalance (e.g., muscle cramps).
- Medication History:
  - Use of QT-prolonging drugs (e.g., fluoroquinolones, antipsychotics), antiarrhythmics, or illicit drugs (e.g., cocaine).

Physical Exam:

- General:cSigns of HF (e.g., jugular venous distension, edema, crackles).
- Cardiac: Irregular rhythm, cannon A waves (AV dissociation in VT), murmurs (e.g., ischemic MR).
- Neurologic: Altered mental status or syncope (if hemodynamically unstable).
- Other: Signs of electrolyte imbalance (e.g., tetany in hypocalcemia), drug toxicity (e.g., tremor in amiodarone toxicity).

Initial Labs:

- Electrolytes:
  - Potassium, magnesium, calcium (hypokalemia, hypomagnesemia increase risk).
- Cardiac Biomarkers:
  - Troponin, BNP (to assess for ischemia or HF).
- Toxicology Screen:
  - If drug-induced arrhythmia suspected.
- ECG:
  - Identify VT morphology (monomorphic vs. polymorphic), QRS duration, QT interval.

Imaging/Other:

- Echocardiogram:
  - Assess EF, structural heart disease (e.g., dilated cardiomyopathy, hypertrophic cardiomyopathy [HCM]).
- Cardiac MRI:
  - Detect scar tissue (e.g., in ischemic VT) or infiltrative disease (e.g., sarcoidosis).
- Coronary Angiography:
  - Rule out ischemia in new-onset VT/VF.
- Electrophysiology Study (EPS):
  - To map arrhythmia substrate for ablation.
- Genetic Testing:
  - If inherited arrhythmia syndrome suspected (e.g., Long QT syndrome, Brugada syndrome).

Types of Ventricular Arrhythmias

Premature Ventricular Contractions (PVCs):

Ectopic beats from the ventricle; often benign but frequent PVCs (>10% burden) may lead to cardiomyopathy.

Non-Sustained Ventricular Tachycardia (NSVT):

≥3 consecutive ventricular beats at >100 bpm, lasting <30 seconds; concerning in structural heart disease.

Sustained Ventricular Tachycardia (VT):

VT lasting >30 seconds or requiring termination due to hemodynamic instability; monomorphic (single focus, often scar-related) or polymorphic (multiple foci, often ischemia or QT prolongation).

Ventricular Fibrillation (VF):

Chaotic, disorganized rhythm leading to cardiac arrest; requires immediate defibrillation.

Torsades de Pointes (TdP):

Polymorphic VT associated with prolonged QT interval; often triggered by drugs, electrolyte imbalances.

Causes of Ventricular Arrhythmias

Acquired Causes:

- Ischemic Heart Disease:
  - Scar-related reentry post-MI.
- Cardiomyopathies:
  - Dilated (DCM), hypertrophic (HCM), or non-ischemic (e.g., sarcoidosis, amyloidosis).
- Electrolyte Imbalances:
  - Hypokalemia, hypomagnesemia, hyperkalemia.
- Drug Toxicity:
  - QT-prolonging drugs (e.g., sotalol, fluoroquinolones), digoxin toxicity.

- Metabolic:
  - Hypoxia, acidosis, or thyroid dysfunction (e.g., hyperthyroidism).

Inherited Causes:

- Long QT Syndrome (LQTS):
  - Mutations in ion channel genes (e.g., KCNQ1, KCNH2, SCN5A) prolong QT interval, leading to TdP and VF.
- Brugada Syndrome:
  - SCN5A mutation; ST elevation in V1-V3, risk of VF, often triggered by fever or sodium channel blockers.
- Catecholaminergic Polymorphic VT (CPVT):
  - Mutations in RYR2 or CASQ2; exercise/stress-induced bidirectional VT, risk of VF.
- Arrhythmogenic Right Ventricular Cardiomyopathy (ARVC):
  - Desmosomal mutations (e.g., PKP2, DSG2); fibrofatty replacement of RV myocardium, VT risk.
- Short QT Syndrome (SQTS):
  - Mutations in potassium channel genes (e.g., KCNH2, KCNQ1); short QT interval (<330 ms), risk of VF and SCD.
- Early Repolarization Syndrome (ERS):
  - J-point elevation on ECG; associated with idiopathic VF, often in young males with no structural heart disease.
- Hypertrophic Cardiomyopathy (HCM):
  - Sarcomeric mutations (e.g., MYH7, MYBPC3); ventricular hypertrophy, risk of VT/VF due to fibrosis and ischemia.
- Dilated Cardiomyopathy (DCM) with Genetic Basis:
  - Mutations in LMNA, TTN, or SCN5A; dilated ventricles, risk of VT due to fibrosis and conduction abnormalities.
- Left Ventricular Non-Compaction (LVNC):
  - Mutations in sarcomeric genes (e.g., MYH7); trabeculated myocardium, risk of VT and SCD due to abnormal myocardial architecture.

Table: Common Causes of Ventricular Arrhythmias

Cause Type	Examples	Mechanism	Notes
Acquired	Ischemic heart disease, DCM	Scar-related reentry, fibrosis	Most common cause of VT in adults
Electrolyte	Hypokalemia, hypomagnesemia	Increased automaticity, afterdepolarizations	Correct rapidly to prevent TdP

Cause Type	Examples	Mechanism	Notes
Drug-Induced	Sotalol, fluoroquinolones	QT prolongation, TdP	Check medication list, ECG for QT interval
Inherited	LQTS, Brugada, CPVT, ARVC, SQTS, HCM, LVNC	Ion channel defects, reentry, fibrosis	Genetic testing, family screening

Management

General Principles:

- Identify and treat underlying causes (e.g., ischemia, electrolytes).
- Stratify risk for SCD to determine need for ICD or ablation.
- Acute management focuses on stabilization; chronic management on prevention.

Acute Management:

- Hemodynamically Unstable VT/VF:
  - Immediate Defibrillation: 200 J biphasic for VF or pulseless VT.
  - ACLS Protocol: CPR, epinephrine 1 mg IV q3-5min, amiodarone 150 mg IV bolus for refractory VF/VT.
- Stable Sustained VT:
  - Pharmacologic: Amiodarone 150 mg IV over 10 minutes, then 1 mg/min x 6 hours, or lidocaine 1-1.5 mg/kg IV bolus.
  - Synchronized Cardioversion: 100-200 J if medical therapy fails.
- Torsades de Pointes:
  - Magnesium Sulfate: 1-2 g IV over 5-10 minutes, even if magnesium levels normal.
- Correct Electrolytes:
  - Replete potassium to >4 mEq/L, magnesium to >2 mg/dL.
- Temporary Pacing:
  - Overdrive pacing to shorten QT interval if refractory.

Chronic Management:

- **PVCs/NSVT**:
  - Observation: if asymptomatic, low burden, and normal heart.
  - Beta-Blockers: Metoprolol 25-100 mg PO daily (reduce sympathetic drive).
  - Ablation: If symptomatic or high PVC burden (>10%) causing cardiomyopathy.

- **Sustained VT:**
  - Antiarrhythmics: Amiodarone 200-400 mg PO daily or sotalol 80-160 mg PO BID (if no HF).
  - Ablation: For scar-related VT or recurrent episodes despite medical therapy.
- **ICD**: For secondary prevention (post-VT/VF arrest) or primary prevention (EF ≤35%, high SCD risk).
- Inherited Arrhythmias:
  - **LQTS**: Beta-blockers (e.g., nadolol 40-80 mg PO daily), avoid QT-prolonging drugs.
  - B**rugada Syndrom**e: ICD for high-risk patients (syncope, family history of SCD); quinidine 300-600 mg PO daily for VT suppression.
  - **CPVT**: Beta-blockers (e.g., nadolol), flecainide 100-200 mg PO daily if breakthrough VT.
  - **ARVC**: Beta-blockers, ICD for high-risk patients (VT, family history of SCD), ablation if focal VT.
  - **SQTS**: ICD for high-risk patients (syncope, VF history); quinidine to normalize QT interval.
  - **ERS**: ICD for survivors of idiopathic VF; no specific medical therapy.
  - **HCM**: Beta-blockers (e.g., metoprolol), ICD for high-risk (e.g., family history of SCD, VT on Holter).
  - **Genetic DCM/LVNC**: ICD for EF ≤35% or VT history, beta-blockers, ablation if recurrent VT.

Indications for EP Ablation:

- Scar-Related VT:
  - Post-MI or non-ischemic cardiomyopathy with recurrent VT.
- Idiopathic VT:
  - Focal origin (e.g., RVOT, fascicular VT) with high success rate (>80%).
- Symptomatic PVCs:
  - High burden (>10%) causing cardiomyopathy or refractory symptoms.
- ARVC:
  - For focal VT refractory to medical therapy.
- HCM:
  - Rarely indicated; considered for monomorphic VT with identifiable focus.

Indications for ICD:

- Secondary Prevention:
  - Post-VT/VF arrest with reversible cause ruled out.

- Primary Prevention:
  - EF ≤35% due to ischemic/non-ischemic cardiomyopathy, NYHA class II-III, despite optimal medical therapy.
- Inherited Syndromes:
  - High-risk Brugada syndrome: (syncope, family history of SCD).
- LQTS with syncope: despite beta-blockers.
- ARVC with VT or family history of SCD.
- SQTS with VF history or syncope.
- ERS with idiopathic VF history.
- HCM with high-risk features: (e.g., VT, family history of SCD, massive LVH >30 mm).
- Genetic DCM/LVNC with EF ≤35%: or documented VT.

Table: Management Strategies for Ventricular Arrhythmias

Arrhythmia Type	Acute Treatment	Chronic Management	Notes
Unstable VT/VF	Defibrillation, ACLS	ICD placement	Amiodarone if refractory
Stable VT	Amiodarone 150 mg IV, cardioversion	Beta-blockers, ablation, ICD if high risk	Assess for underlying structural disease
Torsades de Pointes	Magnesium 1-2 g IV, pacing	Correct electrolytes, beta-blockers	Avoid QT-prolonging drugs
PVCs/NSVT	Observation if stable	Beta-blockers, ablation if symptomatic	Ablation for high burden (>10%)

Complications

Acute:

- Cardiac Arrest:
- VT/VF leading to SCD if untreated.
- Hemodynamic Collapse:
  - Hypotension, syncope, or shock due to impaired cardiac output.
- Thromboembolism:
  - Stasis in dilated ventricles (e.g., DCM, LVNC) increases stroke risk.

Chronic:

- Arrhythmia-Induced Cardiomyopathy:
  - Frequent PVCs/VT causing reduced EF, HF.

- Device-Related:
  - ICD infections, lead dislodgement, or inappropriate shocks.
- Drug Toxicity:
  - Amiodarone (pulmonary fibrosis, thyroid dysfunction), sotalol (TdP risk).

Other:

- Psychological Impact:
  - Anxiety or depression from ICD shocks or SCD fear.
- HF Exacerbation:
  - Recurrent VT worsening ventricular function, especially in DCM, HCM, or LVNC.

Key Pearls

Types: PVCs (benign if low burden), NSVT (concerning in structural disease), sustained VT/VF (life-threatening).

Causes: Ischemic heart disease, cardiomyopathies, electrolytes, inherited syndromes (e.g., LQTS, Brugada, CPVT, ARVC, SQTS, ERS, HCM, genetic DCM, LVNC).

Acute Treatment: Defibrillation for unstable VT/VF, amiodarone or cardioversion for stable VT, magnesium for TdP.

Ablation: Indicated for scar-related VT, idiopathic VT, symptomatic PVCs, or select cases in ARVC/HCM.

ICD: Secondary prevention post-arrest, primary prevention in EF ≤35% or high-risk inherited syndromes.

Monitoring: Assess for reversible causes (e.g., ischemia, electrolytes) before long-term therapy.

References

UpToDate: “Ventricular Arrhythmias: Diagnosis and Management” (2025). UpToDate Ventricular Arrhythmias

AHA: “Guidelines for the Management of Ventricular Arrhythmias and SCD” (2024). AHA Guidelines

ESC: “Management of Ventricular Arrhythmias in Structural Heart Disease” (2023). ESC Guidelines

NEJM: “Catheter Ablation for Ventricular Tachycardia: Advances and Outcomes” (2024). NEJM Ablation

Case Scenarios

Case 1: A 55-Year-Old Male with Syncope

- Presentation: A 55-year-old male with a history of MI (EF 30%) presents after a syncopal episode. He reports palpitations prior to collapse. Exam shows HR 180 bpm, BP 90/60 mmHg, no murmurs.
- Labs/ECG: ECG shows monomorphic VT, QRS 160 ms. Troponin normal, potassium 4.2 mEq/L.
- Diagnosis: Sustained VT (Scar-Related) → Monomorphic VT in ischemic heart disease, hemodynamic instability.
- Management: Admit to ICU. Synchronized cardioversion 100 J (stable VT). Start amiodarone 150 mg IV over 10 minutes, then 1 mg/min x 6 hours. Echocardiogram confirms EF 30%. Refer for ICD placement (secondary prevention). EP consult for ablation (scar-related VT). Beta-blocker (metoprolol 25 mg PO daily) on discharge.

Case 2: A 25-Year-Old Male with Palpitations During Exercise

- Presentation: A 25-year-old male presents with palpitations and near-syncope during a soccer game. He has a family history of SCD in his father. Exam shows HR 160 bpm, BP 110/70 mmHg, normal cardiac exam.
- Labs/ECG: ECG during episode shows bidirectional VT. Echocardiogram normal. Genetic testing reveals RYR2 mutation.
- Diagnosis: Catecholaminergic Polymorphic VT (CPVT) → Exercise-induced VT, family history of SCD, RYR2 mutation.
- Management: Admit for monitoring. Start nadolol 40 mg PO daily (beta-blocker). Flecainide 100 mg PO BID added for breakthrough VT. Refer for ICD placement (high-risk due to syncope, family history). Counsel on avoiding competitive sports. Family screening recommended.

Case 3: A 35-Year-Old Female with Cardiac Arrest

- Presentation: A 35-year-old female presents after cardiac arrest while taking fluconazole for a fungal infection. She was resuscitated with defibrillation. Exam post-arrest shows normal vitals, no murmurs.
- Labs/ECG: ECG shows QTc 520 ms, polymorphic VT (TdP). Potassium 3.2 mEq/L, magnesium 1.8 mg/dL. Genetic testing reveals KCNH2 mutation.
- Diagnosis: Long QT Syndrome (LQTS) → TdP triggered by fluconazole (QT-prolonging drug), KCNH2 mutation, hypokalemia.

- Management: Admit to ICU. Magnesium sulfate 2 g IV bolus, replete potassium to >4 mEq/L. Start nadolol 40 mg PO daily. Refer for ICD placement (secondary prevention, TdP arrest). Discontinue fluconazole, avoid QT-prolonging drugs. Family screening for LQTS.

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