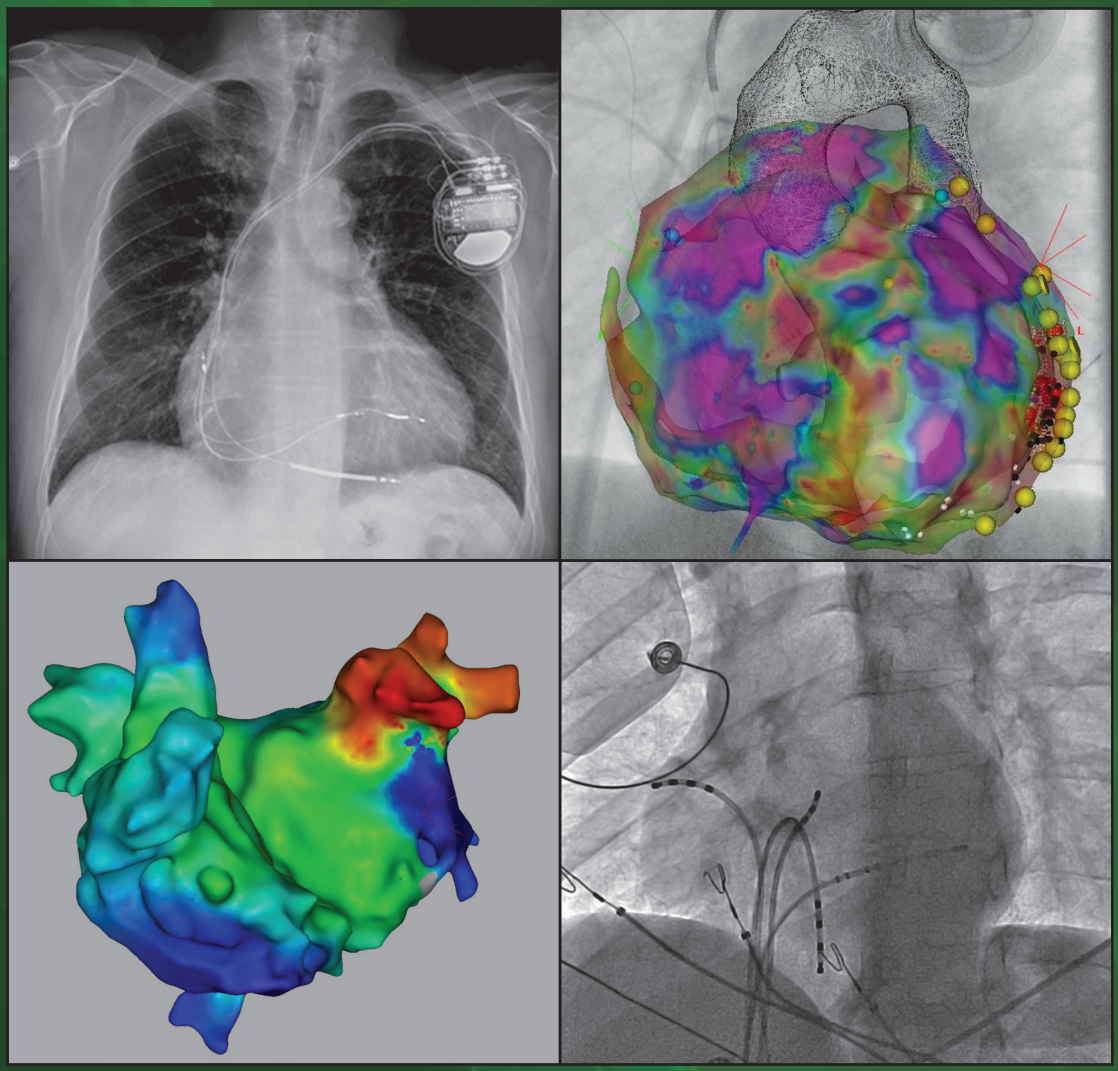
The CLINICAL CARDIAC ELECTROPHYSIOLOGY Handbook

SECOND EDITION



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CONTENTS

CHAPTER

1

Fundamentals

THE CARDIAC ACTION POTENTIAL

Myocardial cells/tissue contain 5 key electrical and mechanical properties.

- **Automaticity**: The ability to initiate an impulse or stimulus. In the absence of external stimulation, the pacemaker cells spontaneously depolarize. This property generates sinus rhythm at a rate appropriate to the body's needs.
- **Excitability**: The ability to respond to an impulse or stimulus. Myocardial cells respond to the impulse generated by the pacemaker cells of the cardiac conduction system through depolarization and repolarization.
- **Conductivity**: The ability to transmit impulses to other areas. While conductivity is much more efficient in the conduction system, the cells in the conduction system and the myocardium also have this property.
- **Refractoriness**: The property that governs the time following excitation until the tissue can be re-excited. This property prevents tissue from being re-excited too soon after the previous excitation, thereby protecting against dangerously rapid rates and reentrant arrhythmias.
- **Contractility**: The ability to respond to electrical stimulation with mechanical action.

The "Fast-Channel" Cardiac Action Potential

Characteristic of action potentials found in the atrial and ventricular myocardium, as well as the rapidly conducting His-Purkinje system—consisting of the bundle of His, right and left bundle branches, fascicular Purkinje cells, and endocardial Purkinje-cell conduction system.

These action potentials have a true resting potential, a rapid depolarization phase, and a prolonged plateau phase.

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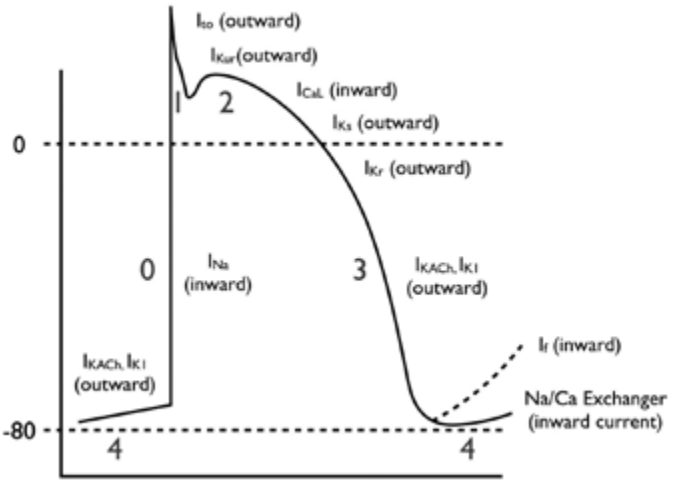


Depolarization

When the cardiac muscle cell is stimulated, the cell undergoes an electrical event called depolarization.

- **Phase 0**: **Rapid depolarization phase ("upstroke")**
  - With stimulation, the fast sodium (Na) channels are activated, resulting in a rapid increase in sodium membrane conductance (G_{Na}) and/or rapid influx of sodium ions (I_{Na}) into the cell.
  - The entry of positively charged sodium ions produces a rapid change in the electrical charge of the interior of the cell, providing the energy for rapid impulse propagation.

Repolarization

Almost immediately after depolarization, the inactivation of the fast sodium channels arrests Na^+ movement into the cell, allowing the cell to initiate the restoration of its (inactive) resting state.

- **Phase 1**: **Early repolarization**
  The early repolarization phase starts with the opening of rapid, outward potassium current (I_{to}). This currents result in rapid repolarization to about 0 mV.
- **Phase 2**: **"Plateau" phase**
  A "stable" membrane potential is observed resulting from a balance of the inward movement of calcium through L-type calcium channels ($I_{Ca,L}$) and the outward movement of K^+ through the delayed rectifier (rapid and slow components: I_{Kr} and I_{Ks}) and the inward rectifier (I_{K1}) potassium channels. The sodium–calcium exchange current ($I_{Na/Ca}$) and the sodium–potassium pump current ($I_{Na/K}$) also play minor roles in the maintenance of the current, and major roles in the maintenance of physiological intracellular sodium, potassium, and calcium concentrations.
- **Phase 3**: **Rapid repolarization at the conclusion of the plateau phase**
  Initially, the net negative change in membrane potential is driven by the inactivation of L-type Ca channels. The rapid delayed rectifier K^+ channel (I_{Kr}) and inwardly rectifying K^+ current (I_{K1}) activate, causing a more rapid net outward current, causing the cell to repolarize to baseline. This phase governs refractoriness

by controlling action potential duration (APD). I_{Na} is inactivated at voltages positive to –60 mV, so following the phase 0 upstroke, the cell cannot be reactivated until it returns to –60 mV during phase 3. The APD to –60 mV determines the "effective refractory period" (ERP) of fast-channel tissue.

- **Phase 4**: **Resting phase**
  The resting phase constitutes a steady, stable, polarized membrane (–90 mV in working myocardial cells). When membrane potential is restored to baseline, the delayed rectifier K^+ channels close. Voltage-regulated inward rectifiers (I_{K1}) remain open, regulating resting membrane potential.

Ventricular Action Potential

Compared to the atrium, the ventricular action potential has a:

- Longer duration
- Higher phase 2 (absent I_{Kur})
- Shorter phase 3, with faster repolarization

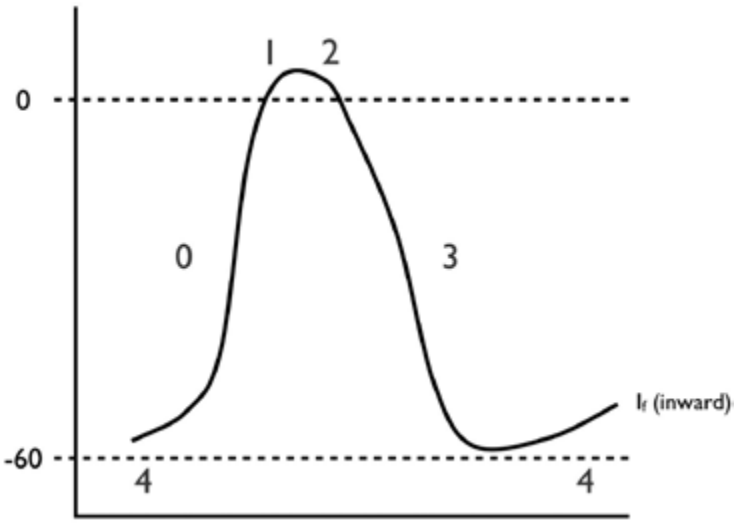
Action Potential of the His-Purkinje System

Compared to the myocardium, the action potential of the **His-Purkinje system** displays the following differences:

- More prominent early (phase 1) repolarization
- Longer plateau phase (phase 2)
- Automaticity: Spontaneous, phase 4 depolarization

The "Slow-Channel" Cardiac Action Potential

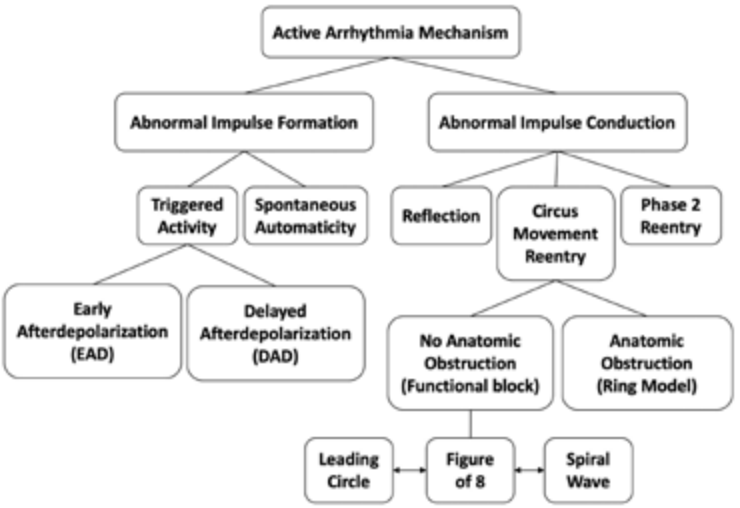
Characteristic of action potentials found in the sinoatrial (SA) and atrioventricular (AV) nodes. The key features of these action potentials are the property of automaticity, as well as the fact that the depolarization phase is slower, and with a shorter APD than "fast-channel" action potentials.



- **Phase 0**: **Rapid depolarization phase ("upstroke")**
  This phase is slower (conduction velocity of ~0.02 to 0.05 m/s) due to a smaller inward current that governs activation (generated by $I_{Ca,L}$, rather than I_{Na}).
- **Phase 1**: **Early repolarization phase**
  Early repolarization is not perceptible in slow-channel tissue, because I_{to} is small and partially inactivated by the relatively positive resting potential.
- **Phase 2**: **"Plateau" phase**
  In principle, this phase is similar to that of fast-channel tissue, except that because of the small phase 0 current, the transitions from phase 0 to phase 1 and subsequently to phase 2 are much less defined.
- **Phase 3**: **Rapid repolarization at the conclusion of the plateau phase**
  This phase has similar ionic mechanisms to fast-channel phase 3. Because $I_{Ca,L}$ is smaller and recovers much more slowly than I_{Na}, the main factor determining ERP in slow-channel tissue is slow, time-dependent recovery of $I_{Ca,L}$. Therefore, unlike fast-channel tissue, APD is not the main determinant of ERP and changes in APD have relatively little effect on ERP. The time-dependent recovery of $I_{Ca,L}$ results in reduced current at fast rates, greatly limiting the maximum follow frequency of the AVN. This is an important protective property that prevents excessively rapid ventricular rates during very rapid supraventricular tachyarrhythmias like atrial flutter (AFL) and atrial fibrillation (AF).
- **Phase 4**: **"Resting" phase**
  Automaticity results from the combination of: (1) spontaneous diastolic depolarization due to phase 4 I_f (a poorly selective, inward current carried mainly by Na and activating upon repolarization), activation of T-type Ca^{2+} current ($I_{Ca,T}$), and inactivation of delayed rectifier K^+ currents; and (2) the maximum negative "resting" potential ("maximum diastolic potential" or MDP) being closer to the depolarization threshold (–50 to –60 mV vs. –80 mV for fast-channel potentials).

ARRHYTHMIA MECHANISMS

General Classification of Arrhythmia Mechanisms



CHAPTER

2

Electrophysiology Study and Maneuvers

THE ELECTROPHYSIOLOGY STUDY

Indications

- Evaluation of sinus node function in patients in whom sinus node dysfunction is suspected (but not confirmed) as the cause of symptoms.
- Evaluation of symptomatic patients in whom His-Purkinje block is suspected as a cause of symptoms.
- Evaluation of narrow QRS complex tachyarrhythmias.
- Evaluation of patients with wide QRS tachycardia in whom the diagnosis is unclear after clinical analysis, and for whom the correct diagnosis is essential for patient care.
- Assessment of patients with Wolff-Parkinson-White syndrome.
- Investigation of unexplained syncope, including suspected structural heart disease and syncope that remains unexplained after evaluation.
- Assessment of patients surviving cardiac arrest without evidence of acute myocardial infarction (MI) or >48 h after the acute phase.
- Assessment of patients with palpitations and inappropriately rapid pulse in whom the cause is not documented.

Complications

- The incidence and type of complication depends on the procedure being performed (see Table 2.1).
  - Atrial tachycardia (AT) or atrial flutter has a complication rate of about 4%–5%.
  - Ablation of the atrioventricular (AV) junction has a complication rate of about 2%–3%.
  - Modification of the AV junction for atrioventricular nodal reentrant tachycardia (AVNRT) has a complication rate of about 3%–4%.
  - Ablation of an AP has a complication rate of about 2%–4%.
  - Ablation of ventricular tachycardia (VT) has a complication rate of about 5%–8%.

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Table 2.1 Complications Associated with Various Diagnostic and Therapeutic Procedures

Complication	Diagnostic	Ablation
Death	<0.1%	0.3%
Access site complication	0.2%	0.6%
Embolism (systemic or cerebral)	<0.1%	0.2%–0.5%
Myocardial ischemia or infarction	<0.1%	0.1%–0.2%
AV block necessitating pacemaker	<0.1%	0.5%–2.0%
Pericardial effusion	<0.1%	0.3%–2.0%
Tamponade	<0.1%	0.2%–0.7%
Pericarditis/chest pain	<0.1%	<1.0%
Venous thrombosis	0.5%–1%	0.5%–1%
Major bleeding	<0.1%	0.2%–0.7%
Pacemaker lead dislodgment	<1%	<1%
Pneumothorax	0.1%	0.1%
Total	1%	3%

STANDARD CATHETER PLACEMENT

High Right Atrium

- Catheters used include Josephson or Cournand shape.
  - Quadripolar catheters are used for simultaneous stimulation and recording.
- Preferred position is high posterolateral wall at the junction of the superior vena cava (SVC) as this site approximates the sinoatrial (SA) node exit site.
  - Right atrial appendage (anterior) may be used if it is difficult to place.
  - Technique: In the anteroposterior projection, advance the catheter to the HRA and torque catheter posteriorly.
- While recording, keep in mind:
  - The EGM corresponds to the depolarization wavefront arriving at the atrial cells of the superior RA.
  - The timing of the EGM is close to the onset of the P wave on the surface ECG.

Right Ventricle

- Catheters used include Josephson or Damato shape.
  - Catheters are quadripolar for simultaneous stimulation and recording, or bipolar (capture inferred from ECG).
- Preferred position is in the right ventricular apex (RVa).
  - Right ventricular outflow tract (RVOT) is used in difficult cases or if dual-site pacing is required.
  - Technique: In anteroposterior or RAO projection, advance catheter to RVa.
- While recording, keep in mind:
  - EGM corresponds to the depolarization of the RV after the signal has exited the His-Purkinje.
  - Timing of the EGM is close to the onset of the QRS on the surface ECG.

His Bundle Catheter

- Catheters used include Josephson or Cournand shape.
- Usual location is approximately 1–2 o'clock on the tricuspid valve (TV) in LAO projection where there is a large His EGM.
  - Technique: Position the catheter across the anterior-superior TV in antero-posterior or RAO projection, then gently withdraw the catheter with clockwise torque to assure septal contact.
- While recording, keep in mind:
  - Atrial EGM corresponds to depolarization of the cells located in the low RA near the atrioventricular node (AVN).
  - His EGM corresponds to depolarization of the proximal His-Purkinje after the EGM exits the AVN.
  - Ventricular EGM corresponds to the depolarization of the ventricular cells adjacent to the node.
    - The timing of the EGM is close to the surface QRS onset as the septum is one of the first areas activated.
- Caveats
  - An inadequate atrial EGM results in:
    - Underestimation of the HV interval due to the recording of a right bundle potential
    - Misinterpretation of the position of the AVN and aortic root

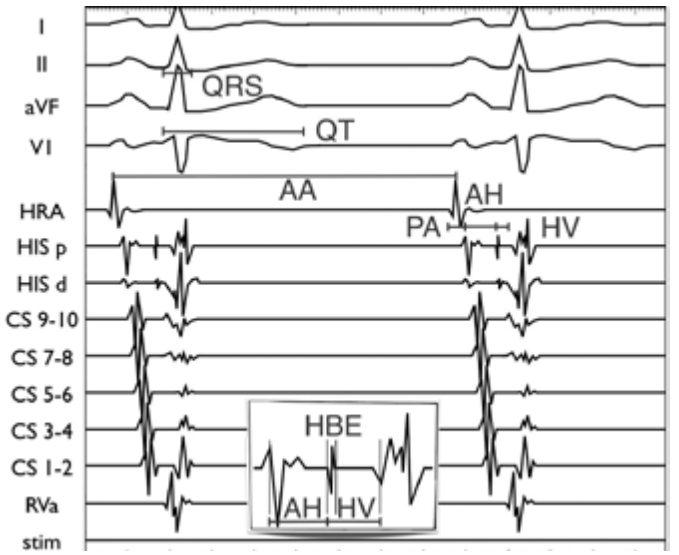
Coronary Sinus

- Multipolar catheter (8–10 electrodes) is utilized to record the LA activation sequence.
- Positioning depends on which approach is used.
  - Inferior approach
    - The catheter is positioned across the TV and deflected inferiorly. It is then withdrawn with clockwise torque until equal atrial and ventricular EGMs are recorded. Once the coronary sinus (CS) ostium is engaged, release the deflection while continuing to apply clockwise torque to allow the tip to turn superiorly and follow the course of the vein.

- Superior approach
  - Position the catheter across the TV and withdraw towards the IVC while applying clockwise torque until equal atrial and ventricular EGMs are recorded.
- Difficulties in positioning the catheter may include:
  - Failure to cannulate the CS ostium
  - Failure to advance catheter distally in the CS due to a valve
  - Recurrent catheter dislodgement; consider using a support sheath (SR0 or SL2) or switching to a superior approach

- While recording, keep in mind:
  - Atrial EGM corresponds to the depolarization of the LA cells adjacent to the mitral annulus.
  - Ventricular EGM corresponds to the depolarization of the LV cells adjacent to the mitral annulus.
  - Caveats
    - If the CS catheter position is too proximal, a false chevron pattern with midline activation will be observed due to the mid CS bipoles being positioned over the CS ostium and thus activating early.
    - If the CS position is too distal, there will be a reverse chevron pattern with the distal and proximal CS being activated before the mid-CS due to anterior and posterior septal activation.

MEASUREMENT OF BASIC CONDUCTION INTERVALS



Conduction Intervals
PA: 25–55 ms AH: 55–125 ms HBE: <30 ms HV: 35–55 ms
Sinus Node Function
Maximum SNRT: ≤1.5 s CSNRT: <550 ms Maximum TRT: ≤5 s SACT: 50–115 ms
Refractory Periods
Atrial ERP: 180–330 ms AV Nodal ERP: 250–400 ms (anterograde) AV Nodal FRP: 330–550 ms Ventricular ERP: 180–290 ms

Cycle Length

- Cycle length (CL) is the length of time between each successive heartbeat as measured in milliseconds.

CHAPTER

10

Ventricular Tachycardia

UNDERSTANDING AND MANAGING VENTRICULAR TACHYCARDIA (VT)

General Information

- Tachyarrhythmia of ventricular origin (originates distal to the bifurcation of the bundle of His) at a rate >120 bpm
- **Non-sustained:** ≥3–5 beats in duration but self-terminates within 30 seconds.
- **Sustained:** ≥30 seconds or requires termination due to hemodynamic instability within 30 seconds.
- **Complex ventricular ectopy:** >10 premature ventricular complexes (PVCs)/hour, couplets, triplets, or non-sustained VT
  - Complex ventricular ectopy confers an increased risk of death if found in association with a structurally abnormal heart; there is minimal risk for a normal heart.

Epidemiology and Clinical Features

- Tolerability depends on the rate, cardiac function, and peripheral compensation.
- **Asymptomatic** (with or without electrocardiogram (ECG) changes)
  - Usually due to a slower VT (rate <200 bpm)
- Potential symptoms attributable to ventricular arrhythmias include:
  - **Palpitations**: Usually paroxysmal
  - **Presyncope**: Dizziness, light-headedness, feeling faint, "greying out"
  - **Syncope**: A sudden loss of consciousness with loss of postural tone with spontaneous recovery may be associated with myoclonic jerks mimicking seizure.
  - Chest pain, dyspnea, and/or fatigue are usually related to underlying heart disease.
- Sudden cardiac death

Anatomy and Physiology (Mechanism)

- Pathophysiologic mechanisms of VT (see Table 10.1)

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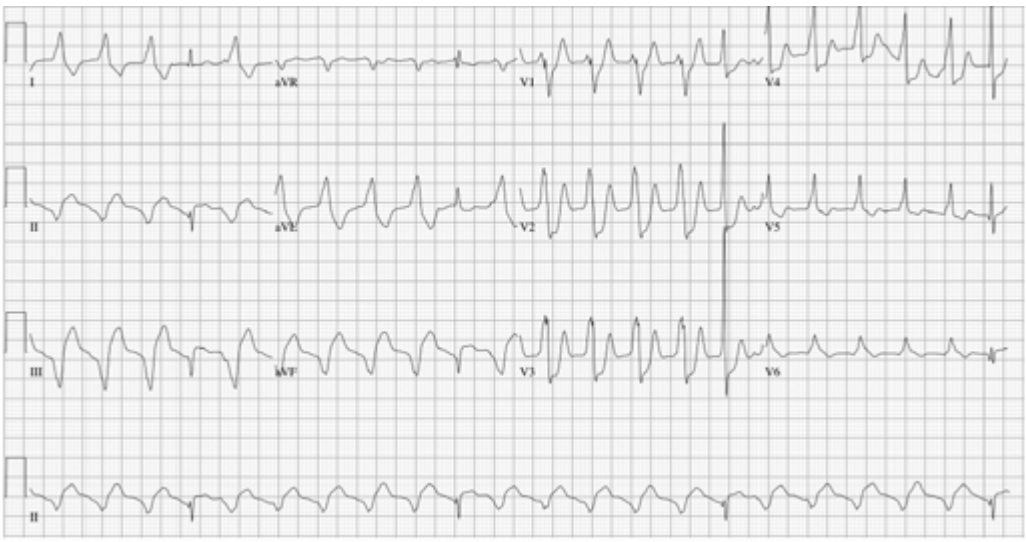
Table 10.1 Mechanisms of Ventricular Tachycardia

	Reentry	Abnormal Automaticity	Triggered Activity
VT morphology	Monomorphic	Monomorphic or polymorphic	Monomorphic or polymorphic
Onset/ termination	Abrupt	"Warm-up/cool-down"	"Warm-up/cool-down"
Inducible at EPS	Inducible • Programmed stimulation	Not inducible	Inducible • Initiated by adrenergic activation and rapid rates • Terminated by verapamil, diltiazem, and/or adenosine
Etiology	Underlying heart disease with myocardial scarring (permanent substrate) or acute ischemia	Metabolic changes • Ischemia, hypoxemia • ↓ Mg, ↓ K • Acid-base disturbances	Pause-dependent • Phase 3 (early afterdepolarization [EAD]) Catecholamine-dependent • Phase 4 (delayed afterdepolarization [DAD])
Risk	Permanent substrate	Reversible substrate	Permanent (genetic or heart disease) or reversible (e.g., due to drug or electrolyte imbalance) substrate

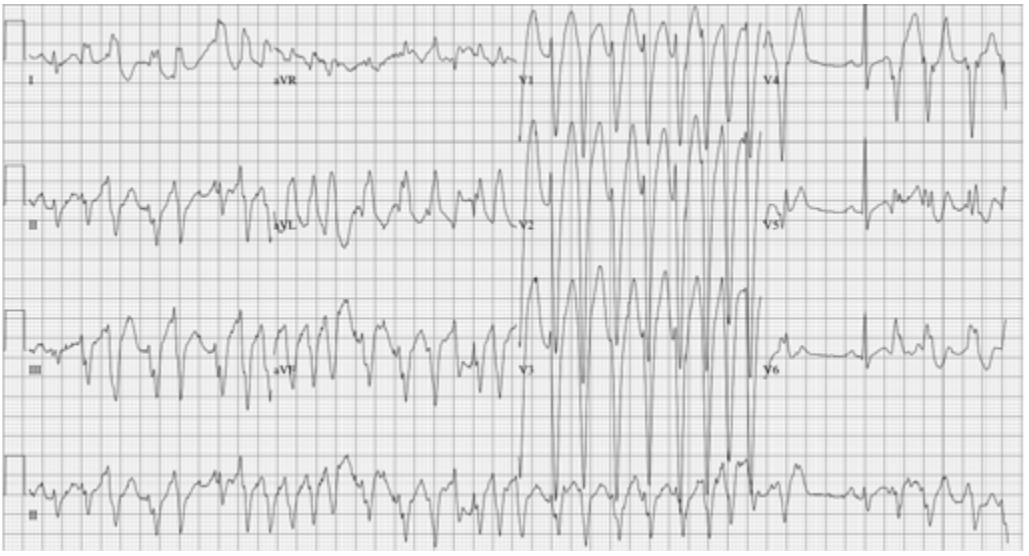
Classification

Monomorphic VT



- Single QRS morphology
- Etiology and classification:
  - Reentrant VT
    - Scar-related: Slow conduction from myocardial fibrosis or scar
      - Old myocardial infarction (MI)
      - Dilated cardiomyopathy (DCM)
      - Arrhythmogenic right ventricular cardiomyopathy (ARVC)
      - Congenital heart disease with surgical scar (e.g., Tetralogy of Fallot)
    - Reentry within the conduction system
      - Fascicular VT (left posterior fascicular VT most common)
      - Bundle branch reentry (ischemic cardiomyopathy or non-ischemic DCM with associated His-Purkinje disease)
  - Enhanced automaticity
    - Primary (idiopathic) VT
      - Outflow tract VT (75%): Right ventricular outflow tract (RVOT)-VT, left ventricular outflow tract (LVOT)-VT, aortic cusp VT
      - Non-outflow tract VT: Papillary muscle, mitral annular, tricuspid annular
      - Acute post MI or surgery (myocardial injury)
  - Triggered activity
    - Acute post MI (usually arising near the His-Purkinje system)

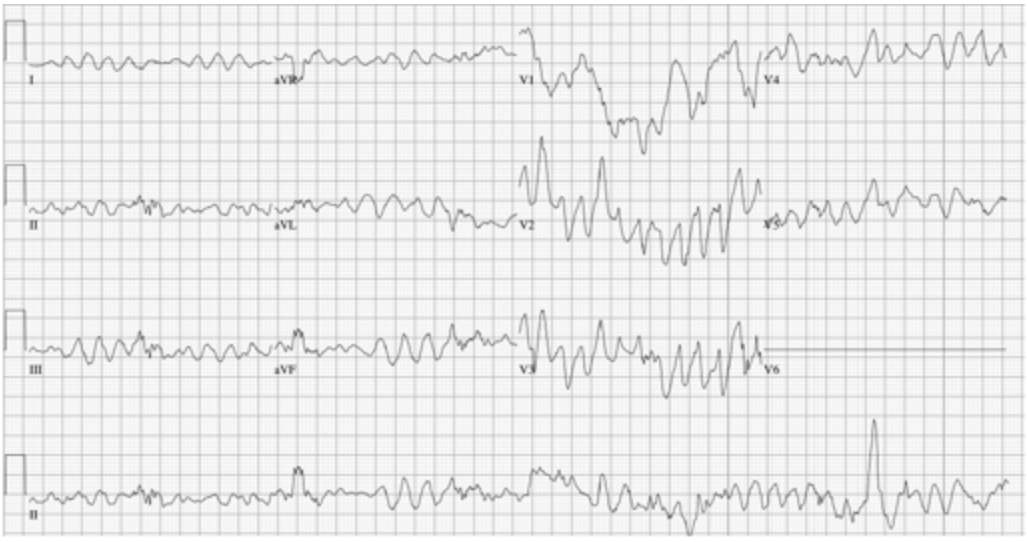
Polymorphic VT



- Unstable VT with beat-to-beat QRS morphology variation (cycle length [CL] between 180 and 600 ms)

- Etiology and classification:
  - Normal baseline QT
    - Pathophysiology
      - Can be due to reentry (e.g., acute MI, often degenerates to ventricular fibrillation [VF]), delayed afterdepolarization (e.g., catecholaminergic polymorphic VT (CPVT))
      - Related to conditions of high sympathetic tone
    - Etiology
      - Acute ischemia (multiple reentrant circuits; abnormal automaticity)
      - Channelopathies: Catecholaminergic polymorphic VT, Brugada syndrome, idiopathic polymorphic VT (PMVT)/VF
  - Prolonged baseline QT
    - Torsades de pointes
      - Defined as a PMVT with a QRS amplitude and cardiac axis rotation over a sequence of 5–20 beats.
      - Usually it is not sustained but recurs if the underlying cause is not corrected.
    - Pathophysiology
      - Due to early afterdepolarization
      - **Typical variant:** Initiated by "short-long-short" coupling intervals (pause-dependent, typical for drug-induced)
      - **Short coupled variant:** Initiated by "normal-short" coupling (induced by stress or startle, typical for congenital syndromes, adrenergic dependent)
    - Etiology
      - Acquired prolonged QT: Drugs (class Ia, III antiarrhythmic drug [AAD], phenothiazines, tricyclic antidepressant [TCA]), low Mg, or K
      - Congenital prolonged QT
  - Short QT syndrome

Ventricular Fibrillation (VF)



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" . . . a quick source for information in the EP lab or on the wards, as well as a guide to learning—offering the ability to quickly review the essential components before a case or to reinforce new notions and practices encountered during a case."

— From the Preface

New and updated sections in the *Second Edition*:

- Ablation energy and biophysics, including pulsed field ablation
- AF ablation techniques adjunctive to pulmonary vein isolation
- Alternatives to cardiac resynchronization therapy
- His bundle pacing
- ICD discrimination algorithms
- Intracardiac echocardiography
- Left atrial appendage occlusion

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