Arrhythmias and conduction disturbances

Ventricular arrhythmias
Introduction

- PVCs are very common arrhythmias that can occur in healthy or diseased hearts with multiple features on ECG
- VT and VF are dangerous arrhythmias that can lead to sudden cardiac death
- Not all wide complex tachyarrhythmias arise from the ventricles
  - Distinguish between VT and SVT with aberrancy because the treatment and prognosis of each is very different
Mechanisms of ventricular arrhythmias

- **Impulse Formation Disorders**
  - Abnormal Automaticity
    - Discharge from a pathologic ectopic ventricular focus
  - Triggered beats
    - Afterdepolarizations (AD): Abnormal depolarizations of myocytes that interrupt phase 2, 3, or 4 of the AP
The Action Potential

During Phase 0, the cell depolarizes as the sodium channels are opened and positive sodium ions rapidly move into the heart.

- 90 mV

During Phase I, there is early rapid repolarization.

0 mV

During Phase II, this repolarization plateaus.

At Phase III there is a later phase of repolarization (primarily secondary to potassium and calcium ions).

30 mV

In Phase IV, the cell has again reached the resting membrane potential and it is during this time that the diastolic depolarization can occur in spontaneously excitable cells.
Afterdepolarization

- **Early afterdepolarization (EAD)**
  - occurs with abnormalities during phase 2 (interrupted due to augmented opening of Ca channels) or phase 3 (opening of Na channels)
Delayed afterdepolarization (DAD)

- begin during phase 4 - after repolarization is completed, but before another action potential would normally occur. Due to elevated cytosolic Ca concentrations (digoxin toxicity)
Mechanisms of ventricular arrhythmias

- **Impulse Conduction Disorders**
  - **Delayed conduction**
    - Delayed SA/AV nodal impulse allows initiation of inherent ventricular impulse
  - **Re-entry**
    - Creation of a circuit that leads to 2 or more depolarizations in surrounding tissue
The “Re-Entry” Mechanism of Ectopic Beats & Rhythms

Electrical Impulse

Cardiac Conduction Tissue

Fast Conduction Path
Slow Recovery

Slow Conduction Path
Fast Recovery

Tissues with these type of circuits may exist:

• in microscopic size in the SA node, AV node, or any type of heart tissue
• in a “macroscopic” structure such as an accessory pathway in WPW
1. An arrhythmia is triggered by a premature beat
2. The beat cannot gain entry into the fast conducting pathway because of its long refractory period and therefore travels down the slow conducting pathway only
3. The wave of excitation from the premature beat arrives at the distal end of the fast conducting pathway, which has now recovered and therefore travels retrogradely (backwards) up the fast pathway.
4. On arriving at the top of the fast pathway it finds the slow pathway has recovered and therefore the wave of excitation ‘re-enters’ the pathway and continues in a ‘circular’ movement. This creates the re-entry circuit.
Atrial Re-entry
• atrial tachycardia
• atrial fibrillation
• atrial flutter

Atrio-Ventricular Nodal Re-entry
• supraventricular tachycardia

Atrio-Ventricular Re-entry
• Wolf Parkinson White
• supraventricular tachycardia

Ventricular Re-entry
• ventricular tachycardia

Re-entry Circuits as Ectopic Foci and Arrhythmia Generators
Types

- Premature ventricular contraction (PVC)
  - Bigeminy, trigeminy, couplets, interpolated, monomorphic, multimorphic, fusion beat
- Idioventricular rhythm/ accelerated idioventricular rhythm
- Ventricular parasystole
- Ventricular tachycardia (VT)
- Torsades de pointes
- Ventricular flutter
- Ventricular fibrillation (VF)
Premature Ventricular Contractions (PVCs)

- **Epidemiology**
  - Very common; occur in healthy people & pts with cardiac disease

- **Etiology**
  - Cardiac: CAD, post-MI, MVP, CHF, rheumatic heart disease, congenital arrhythmias
  - Non-cardiac: acid-base disturbance, electrolyte abnormalities, meds, caffeine, anxiety
Premature Ventricular Contractions (PVCs)

- **Symptoms**
  - Palpitations, “skipped beats”
  - Chest or neck discomfort

- **Physical exam findings**
  - Presence of premature beat
  - Hypotension
  - Decreased or absent peripheral pulses (radial)
Premature Ventricular Contractions (PVCs)

QRS is wide and much different ("bizarre") looking than the normal beats. This indicates that the beat originated somewhere in the ventricles and consequently, conduction through the ventricles did not take place through normal pathways. It is therefore called a “ventricular” beat.

There is no p wave, indicating that the beat did not originate anywhere in the atria.

A "retrograde p-wave" may sometimes be seen on the right hand side of beats that originate in the ventricles, indicating that depolarization has spread back up through the atria from the ventricles.
Premature Ventricular Contractions (PVCs)

• In most cases, the heart circulates no blood (no pulse because of an irregular squeezing motion)

• PVC’s are sometimes described by lay people as “skipped heart beats”, often normal variant

![Diagram showing R on T phenomenon, Multifocal PVC's, and Compensatory pause after the occurrence of a PVC]
ECG Characteristics of PVCs

- Ectopic beat originating from ventricles occurring before next expected beat (premature)
- Usually not proceeded by P wave
- **Wide QRS**: at least > 0.12 sec, usually 0.16-0.2 with bizarre morphology
- Large T wave in the opposite direction of the major QRS deflection
ECG Characteristics of PVCs

- Full Compensatory Pause
  - Follows most PVCs
  - PVCs usually do not conduct retrograde to the atria, thus SA nodal rhythm not disturbed
  - When SA node discharges, the ventricles are still refractory from the PVC and don’t depolarize in response to the impulse
  - The interval between the first sinus beat and the PVC plus the interval between the PVC and the next sinus beat = 2 normal sinus intervals
ECG Characteristics of PVCs

- **Interpolated PVCs**
  - No compensatory pause
  - PVC occurs between 2 normal sinus beats
  - No change in the R-R interval
  - Usually seen when the HR is slow
ECG Characteristics of PVCs

- **Fusion beats**
  - Simultaneous activation of the ventricle from SV impulse and a PVC
  - Ventricular depolarization occurs simultaneously in two directions
  - QRS complex that has the characteristics of the PVC and the QRS complex of the underlying rhythm
ECG Characteristics of PVCs

- **Captured beats** *(Dressler beats)*
  - QRS complexes during a WCT that are identical to the sinus QRS complex.
  - Implies that the normal conduction system has momentarily "captured" control of ventricular activation from the VT focus.
ECG Characteristics of PVCs

- **R on T phenomenon**
  - PVC begins during mid/late T wave
  - Associated with vulnerable ventricles often predisposing to polymorphic VT or VF, especially in acute ischemia

![ECG waveform showing PVC](image)
PVC Patterns

- **Bigeminy**
  - PVC every other beat
  - “Rule of bigeminy”: often becomes self-perpetuating

- **Trigeminy**
  - PVC every 3rd beat
PVC Patterns

- **Couplets**
  - Two successive PVCs

- **Triplets**
  - Three successive PVCs
  - Rate <100 bpm
PVC Morphology

- **Monomorphemic**
  - PVCs originate from a single ventricular ectopic focus
  - Single wave morphology

- **Polymorphic**
  - PVCs originate from multiple ventricular ectopic foci
  - $\geq 2$ morphologies
PVC Morphology

- left vs right PVC's - best recognized in V₃
  - '+' in V₃ => LV origin; called RBBB pattern
    - usually monophasic R or qR in V₃
    - rS or QS in V₆
    - left rabbit ear taller than right in V₃; often opposite if true RBBB
  - ‘-” in V₃ => RV origin, LBBB pattern

- importance
  - LV more likely with HD
  - LV more likely to precipitate V-tach in acute MI
Premature ventricular contraction (PVC)

- **Cause:** enhanced automaticity in the ventricular conduction or muscle tissue
  - Electrolyte imbalance (K↑ or ↓, Mg ↓, Ca ↓)
  - Metabolic acidosis
  - Hypoxia
  - Drug intoxication (cocaine, amphetamines, tricyclic antidepressants)
  - Enlargement or hypertrophy of ventricular chamber
  - Increased sympathetic stimulation
  - Myocarditis
  - Caffeine or alcohol ingestion, Tobacco use
  - Irritation of ventricles by pacemaker or pulmonary artery catheter
  - Sympathomimetic drug (epi, isoproterenol)
PVC's are Dangerous When:

- They are frequent (> 30% of complexes) or are increasing in frequency
- The come close to or on top of a preceding T-wave (R on T)
- Three or more PVC's in a row (run of V-tach)
- Any PVC in the setting of an acute MI
- PVC's come from different foci ("multifocal" or "multiformed")

These dangerous phenomenon may preclude the occurrence of deadly arrhythmias:

- **Ventricular Tachycardia** ➔ The sooner defibrillation takes place,
- **Ventricular Fibrillation** ➔ the increased likelihood of survival
PVC Prognosis

Lown Classification

- Class 1: <30 PVC/hr
- Class 2: >30 PVC/hr
- Class 3: Multiform PVCs
- Class 4a: PVC couplets
- Class 4b: PVC triplets or greater
- Class 5: R on T

- Post MI PVCs and Lown’s class 3-5 are associated with ↑ risk for VT/VF and sudden death
Intervention

- Intervention:
  - A cardiac origin: drug to suppress ventricular irritability (procainamide, amiodarone and lidocaine)
  - Recently PVC: underlying heart disease or complex medical condition
  - Chronic PVC: frequent PVC or dangerous pattern
Idioventricular (Escape) rhythm

- Escape rhythm due to failure of SA/AVN ventricular activation or complete conduction block
- Inherent 20-40bpm takes over since it is no longer suppressed
- Regular wide QRS
- Etiologies
  - Post-MI, CM, digoxin toxicity
Accelerated Idioventricular Rhythm (AIVR)

- May result from accelerated ventricular focus that is faster than the prevailing sinus rate and takes over or can occur as escape rhythm (generally with 3\textsuperscript{rd} degree AV block)
- Usually 60-100 bpm (differentiates from VT)
- Regular wide QRS
- Usually self limited, rarely see progression to VT/VF
Accelerated Idioventricular Rhythm (AIVR)

Enhanced automaticity appears to be the likely electrophysiologic mechanism behind the genesis of AIVR. Enhanced automaticity generally is ascribed to phase-4 depolarization of the action potential of the myocardial cell. AIVR can occur in the His-Purkinje fibers or myocardium under certain abnormal metabolic conditions.

AIVR arises from subordinate or second-order pacemakers and manifests itself when the patient's prevailing sinus rate becomes lower than the accelerated rate (AIVR) of the otherwise suppressed focus. Sinus bradycardia combined with enhanced automaticity of the subordinate site is the common pathophysiology.

Several conditions, including myocardial ischemia (especially inferior wall ischemia or infarction), digoxin toxicity, electrolyte imbalance (eg, hypokalemia), and hypoxemia may accentuate the phase-4 depolarization in the subordinate pacemaker tissues of the atrioventricular (AV) junction or His-Purkinje system, thus increasing the rate of impulse generation. Frequently, when inferior wall ischemia is present, the subordinate pacemaker acceleration coexists with sinus node depression. The latter permits escape and domination of the pacemaker function, which may occur with AV junctional or ventricular rates of only 60-70 bpm. The ectopic mechanism also can begin after a premature ventricular complex or, as described above, when the ectopic ventricular focus simply can accelerate sufficiently enough to overtake the intrinsic rhythm.

The onset of AIVR is gradual (nonparoxysmal). The ventricular rhythm can be regular or irregular and, occasionally, can show sudden doubling, suggesting the presence of exit block. The ventricular rate, commonly 60-110 bpm, usually stays within 10-15 beats of the sinus rate; therefore, the control of the cardiac rhythm occasionally passes back and forth between these 2 competing pacemaker sites.

Fusion beats often develop at the onset and termination of arrhythmia, which occurs when the pacemakers are competing for control of ventricular depolarization. Because of the slow rate, capture beats also are common. Due to the slow rate and nonparoxysmal onset, precipitation of more rapid ventricular arrhythmias rarely is observed. Rhythm termination generally occurs gradually, while the underlying sinus rhythm accelerates or the AIVR slows down.
Causes and significance

- When all of the heart’s higher pacemakers fail to function or supraventricular impulse was blocked
- **Idioventricular rhythm may accompany 3rd-degree heart block**
- **Cause:**
  - Myocardial ischemia
  - Myocardial infarction (MI)
  - Digoxin toxicity, beta-adrenergic blockers, calcium antagonist, tricyclic antidepressant
  - Pacemaker failure
  - Metabolic imbalances
- **Continuous idioventricular rhythm: serious situation**
- **Slow rate and loss of atrial kick ➔ ↓ cardiac output ➔ death**
S/S and intervention

- Continuous idioventricular rhythm: due to ↓ cardiac output ➔ dizziness, light-headedness, syncope or loss of consciousness

- Tx: increase HR, improve cardiac output and establish normal rhythm
  - Atropine: increase HR
  - If hypotension or clinical instability ➔ pacemaker
  - Transcutaneous pacemaker in an emergency
  - Not to suppress the idioventricular rhythm ➔ never use lidocaine or other antiarrhythmic to suppress the escape beat
  - ECG monitor until restore hemodynamic stability
  - Bed rest
  - Education
Recognizing accelerated idioventricular rhythm

This rhythm strip illustrates accelerated idioventricular rhythm.

- **Rhythm**: regular
- **Rate**: ventricular — 80 beats/minute
- **P wave**: absent; sometimes inverted P wave follows the QRS complex
- **PR interval**: unmeasurable
- **QRS complex**: 0.18 second; wide and bizarre
- **T wave**: 0.48 second
- **QT interval**: usually prolonged
- **Other**: none

100 > **AIVR** > 30-40 beat/min
Idioventricular rhythm: 30-40 beat/min
**Ventricular tachycardia**

- Usually precedes ventricular fibrillation and sudden cardiac death.
- <30 sec → few or no symptoms
- Sustained → immediate Tx to maintain cardiac output
- **Cause:**
  - Myocardial ischemia
  - MI
  - Coronary artery disease
  - Valvular heart disease
  - Heart failure
  - Cardiomyopathy
  - Electrolyte imbalance (↓ K)
  - Drug intoxication: procainamide, quinidine or cocaine
- ↓ ventricular refilling time and drop of cardiac output → cardiovascular collapse
Notes on V-tach:

• Typical V-tach patient
  • MI with complications & extensive necrosis, EF<40%, d wall motion, v-aneurysm)
• V-tach complexes are likely to be similar and the rhythm regular
  • Irregular V-Tach rhythms may be due to:
    • breakthrough of atrial conduction
      • atria may “capture” the entire beat beat
      • an atrial beat may “merge” with an ectopic ventricular beat
        (fusion beat)

Fusion beat - note p-wave in front of PVC and the PVC is narrower than the other PVC’s – this indicates the beat is a product of both the sinus node and an ectopic ventricular focus

Capture beat - note that the complex is narrow enough to suggest normal ventricular conduction. This indicates that an atrial impulse has made it through and conduction through the ventricles is relatively normal.
Ventricular Tachycardia (VT)

Rate: 140-220 (200±50); at least 3 ectopic QRS in row
Rhythm: generally regular (may be slightly irregular)
P wave: no related P waves
P-R: none
QRS: normally wide and bizarre; (≥ 0.14 sec favors VT)

• Usually associated with MI or other organic HD; unusual in normals
• Often serious requiring quick treatment if sustained
• Mechanism? Reentry or rapid firing ectopic??
ECG diagnosis - clues to rule in VT

• Difficult to rule out SVT with aberrant ventricular conduction
  • use more leads whenever possible
• Unrelated P's (independent atrial activity) - rules out atria
• Presence of fusion beats suggests VT as contrasted to SVT
• LVT favored - monophasic pattern in R chest leads (V1 or MCL1) with taller left 'rabbit ear'
• Concordant positivity (all complexes positive) in V leads => favors LV ectopy (rule out WPW)
• Concordant negativity = favors RV ectopy (rule out LBBB)
• QRS interval > .14 sec (prior tracing available to rule out BBB)
Classification of Ventricular Arrhythmia by Clinical Presentation

- Hemodynamically stable
  - Asymptomatic
  - Minimal symptoms, e.g., palpitations
- Hemodynamically unstable
  - Presyncope
  - Syncope
  - Sudden cardiac death
  - Sudden cardiac arrest

ACC/AHA/ESC 2006 Guidelines for Management of Patients With Ventricular Arrhythmias and the Prevention of Sudden Cardiac Death
Classification of Ventricular Arrhythmia by Electrocardiography

- Nonsustained ventricular tachycardia (VT)
  - Monomorphic
  - Polymorphic
- Sustained VT
  - Monomorphic
  - Polymorphic
- Bundle-branch re-entrant tachycardia
- Bidirectional VT
- Torsades de pointes
- Ventricular flutter
- Ventricular fibrillation

ACC/AHA/ESC 2006 Guidelines for Management of Patients With Ventricular Arrhythmias and the Prevention of Sudden Cardiac Death
VT - S/S and intervention

- Weak or absent pulses
- Low cardiac output ➔ hypotension, conscious change, angina, heart failure or organ perfusion ↓
- Intervention:
  - Evaluation of consciousness, respiration and circulation
  - If pulseless ➔ immediate defibrillation
  - **Unstable P’t**: ventricular rate > 150 beat/min with S/S: hypotension, shortness of breath, chest pain or alternated consciousness ➔ immediate synchronized cardioversion
  - **Stable P’t with wide-complex VT** and no signs of heart failure
    - Monomorphimic: IV procainimide, amiodarone
    - Polymorphic: Magnesium + ventricular or atrial pacing
  - Chronic, recurrent episodes of VT unresponsive to drug therapy ➔ implantation cardioversion-defibrillator (ICD)
  - Education
Torsades De Pointes

- Rapid ventricular rate: 250~350 beat/min
- Character: QRS complex change back and forth, with amplitude of each successive complex gradually increasing and decreasing
- DDx: ventricular flutter: rapid, regular, repetitive beating of ventricle → single ventricular focus firing at a rapid rate of 250~350 beat/min → smooth and “sine-wave” appearance
Torsades De Pointes

- French term meaning "twisting of the points"
- Torsade de pointes occurs in the setting of delayed ventricular repolarization, evidenced by prolongation of the QT intervals or the presence of prominent U waves.
- Causes:
  - Electrolyte imbalances, including hypokalemia, hypomagnesemia, and less commonly, hypocalcemia, which prolong repolarization
  - Miscellaneous factors such as severe bradyarrhythmias, liquid protein diets, and hereditary long-QT syndromes

Table 12. Examples of Drugs Causing Torsades de Pointes*

Frequent (greater than 1%)
(e.g.,) hospitalization for monitoring recommended during drug initiation in some circumstances)
- Disopyramide
- Dofetilide
- Ibutilide
- Procainamide
- Quinidine
- Sotalol
- Ajmaline

Less frequent
- Amiodarone
- Arsenic trioxide
- Bepridil
- Cisapride
- Anti-infectives: clarithromycin, erythromycin, halofantrine, pentamidine, sparfloxacin
- Antiemetics: domperidone, droperidol
- Antipsychotics: chlorpromazine, haloperidol, mesoridazine, thioridazine, pimozide
- Opioid dependence agents: methadone
<table>
<thead>
<tr>
<th>Table 13. Risk Factors for Drug-Induced Torsades de Pointes</th>
</tr>
</thead>
<tbody>
<tr>
<td>• Female gender</td>
</tr>
<tr>
<td>• Hypokalemia</td>
</tr>
<tr>
<td>• Bradycardia</td>
</tr>
<tr>
<td>• Recent conversion from atrial fibrillation</td>
</tr>
<tr>
<td>• Congestive heart failure</td>
</tr>
<tr>
<td>• Digitalis therapy</td>
</tr>
<tr>
<td>• High drug concentrations (<em>exception: quinidine</em>), often due to drug interactions</td>
</tr>
<tr>
<td>• Rapid rate of intravenous drug administration</td>
</tr>
<tr>
<td>• Baseline QT prolongation</td>
</tr>
<tr>
<td>• Ventricular arrhythmia</td>
</tr>
<tr>
<td>• Left ventricular hypertrophy</td>
</tr>
<tr>
<td>• Congenital long QT syndrome</td>
</tr>
<tr>
<td>• Certain DNA polymorphisms</td>
</tr>
<tr>
<td>• Severe hypomagnesemia</td>
</tr>
<tr>
<td>• Concomitant use of 2 or more drugs that prolong the QT interval</td>
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<tr>
<td>• Combination of QT-prolonging drug with its metabolic inhibitor</td>
</tr>
</tbody>
</table>
Torsades De Pointes

Tx:

- **Removing or correcting causative factors** such as drug toxicity, electrolyte imbalance, or underlying bradycardia.

- In emergency settings a **temporary pacemaker** may be inserted to accomplish "overdrive" suppression of the arrhythmia by increasing the underlying heart rate and thereby decreasing ventricular repolarization time.

- **Intravenous magnesium sulfate** has proved highly useful for suppressing this arrhythmia.

- **Drug therapy** with isoproterenol or bretylium has been used in selected cases.

- Sustained episodes of torsade de pointes attempted **cardioversion**
Ventricular fibrillation

- VF ➔ chaotic, disorganized pattern of electrical activity ➔ multiple ectopic pacemaker ➔ no cardiac output ➔ sudden cardiac death

- Cause:
  - CAD
  - Myocardial ischemia
  - MI
  - Untreated VT
  - Underlying heart disease
  - Acid-base imbalance
  - Electric shock
  - Severe hypothermia
  - Drug toxicity (digoxin, quinidine and procainamide)
  - Electrolyte imbalance (↑↓ K, ↑ Ca)

- Completely ineffective contraction, cardiac output = 0 ➔ ventricular standstill and death
ECG

- Ventricular rhythm: no pattern or regularity
- P wave, QRS complex, PR interval, T wave: can’t be determined
- Coarse fibrillatory wave: greater chance of successful electrical cardioversion than small amplitude
S/S and intervention

- Full cardiac arrest, unresponsive, undetectable BP

- Intervention:
  - Access VF, confirm
  - Immediate defibrillation is the most effective Tx
    - Unsynchronized electrical shock at 200 J
    - Unsynchronized electrical shock at 200 to 300 J
    - Unsynchronized electrical shock at 360 J
  - CPR until defibrillator arrives
Intervention

- Intervention:
  - Drug: epi or vasopressin (for persistent VF)
  - Antiarrhythmia agent: amiodarone, lidocaine and Mg
  - AED- automated external defibrillator ➔ out-of-hospital setting
  - Rapid recognition of the problem and defibrillation
  - Education, CPR
Wide complex tachyarrhythmias

- QRS greater or equal to 0.12 sec and rate >100 bpm
- Not all are of ventricular origin
- Differential
  - Ventricular tachycardia
  - Supraventricular tachycardia with aberrancy (conduction block) or presence of an accessory pathway with antegrade conduction (WPW syndrome)
  - Artifact
VT vs SVT with Aberrancy

- Both manifest as wide complex tachycardias on ECG
- Distinguishing ECG findings:
  - SVT with aberrant conduction
    - QRS > 0.14
    - Rhythm onset with premature P wave
    - PR interval <100msec
    - P wave and QRS are linked
    - Vagal maneuver slows/terminates rhythm
  - Monomorphic VT
    - QRS >0.14 msec
    - AV dissociation with fusion or capture beats
    - Absence of RS complex in precordial leads
    - Extreme axis deviation
- If above findings fail to be detected, morphologic criteria used: if QRS in V1 does NOT look like typical R or L conduction block
<table>
<thead>
<tr>
<th>Characteristic</th>
<th>Ventricular tachycardia</th>
<th>Rate-related aberrancy</th>
<th>Preexcitation tachycardia</th>
</tr>
</thead>
<tbody>
<tr>
<td>AV dissociation (more QRS than P waves)</td>
<td>Rules in</td>
<td>Practically rules out</td>
<td>Practically rules out</td>
</tr>
<tr>
<td>Captured beats</td>
<td>Rules in</td>
<td>Rules out</td>
<td>Rules out</td>
</tr>
<tr>
<td>Fusion beats</td>
<td>Rules in</td>
<td>Rules out</td>
<td>Rules out</td>
</tr>
<tr>
<td>Rhythm (ventricular)</td>
<td>Regular, except torsade de pointes</td>
<td>Regular, except AF and MAT</td>
<td>Regular, except AF and MAT</td>
</tr>
<tr>
<td>QRS axis</td>
<td>Leftward shift supports diagnosis</td>
<td>Usually normal</td>
<td>Usually normal</td>
</tr>
<tr>
<td>QRS duration &gt; 140 msec</td>
<td>Strongly supports diagnosis</td>
<td>Almost never occurs</td>
<td>Rare</td>
</tr>
<tr>
<td>Remote myocardial infarction</td>
<td>Common</td>
<td>Usually not present</td>
<td>Usually not present</td>
</tr>
<tr>
<td>Normal heart</td>
<td>Uncommon</td>
<td>Common</td>
<td>Common</td>
</tr>
<tr>
<td>Response to carotid massage</td>
<td>None</td>
<td>May terminate if AV node–dependent</td>
<td>May terminate if AV node–dependent</td>
</tr>
<tr>
<td>Response to Valsalva maneuver</td>
<td>None</td>
<td>May terminate if AV node–dependent</td>
<td>May terminate if AV node–dependent</td>
</tr>
</tbody>
</table>

AF, atrial fibrillation; AV, atrioventricular; MAT, multifocal atrial tachycardia.
Physical findings highly suggestive of VT:

- Signs of AV dissociation, including:
  - Canon A waves in the jugular venous pulsations
  - Varying BP measurement from beat to beat
  - Varying intensity of $S_1$
WQRS tachycardia algorithm

1. presence of AV dissociation;
2. presence of an initial R-wave in lead aVR;
3. did the morphology of the WCT correspond to bundle branch (BBB) or fascicular block (FB)?
4. estimation of initial (Vi) and terminal (Vt) ventricular activation velocity ratio (Vi/Vt) by measuring the voltage changes on the ECG tracing during the initial 40 ms (Vi) and the terminal 40 ms (Vt) of the same bi- or multiphasic QRS complex.
**Step 1** A-V dissociation present?
- Yes
- No

VT diagnosed

**Step 2** Initial R wave in aVR present?
- Yes
- No

VT diagnosed

**Step 3** QRS morphology unlike BBB or FB?
- Yes
- No

VT diagnosed

**Step 4** $V_1/V_2 \leq 1$?
- Yes
- No

VT diagnosed

SVT diagnosed
Clinical importance

- Misdiagnosing VT as SVT can lead to fatal error

- Treating VT as SVT with verapamil, diltiazem, and adenosine can precipitate ventricular fibrillation, even if initially stable.

- All wide complex tachyarrhythmia should be considered VT until proven otherwise
Primary ABCD survey - Basic CPR and defibrillation

Check responsiveness
Activate emergency response system
Call for defibrillator
A Airway: open the airway
B Breathing: give 2 rescue breaths, each over 1 second.
C Circulation: give 30 chest compressions: 2 rescue breaths until defibrillator ready
D Defibrillation: assess for and shock VF/pulseless VT, x 1, if necessary.
(monophase 360 J or biphasic 150-200 J)

VF/pulseless VT? Yes

Defibrillate x1
(360 J monophasic, 150-200 J biphasic)

Immediately resume CPR x 2 minutes
30 chest compressions: 2 rescue breaths

Secondary ABCD survey - More advanced assessment and treatment

A Airway:
Place airway device as soon as possible
B Breathing:
Confirm airway device placement by exam plus confirmation device
Secure airway device; purpose-made tube holders preferred
Confirm effective oxygenation and ventilation
C Circulation:
Establish IV access
Identify rhythm and monitor
Administer drugs appropriate for rhythm and condition
D Differential diagnosis:
Search for and treat identified reversible causes

Epinephrine: 1 mg IV push, repeat every 3 to 5 minutes
OR Vasopressin: 40 U IV, single dose, one time only

Resume attempts to defibrillate
1 x 360 J (or equivalent biphasic) within 30 to 60 seconds

Consider antiarrhythmics: amiodarone(class IIb); lidocaine (Indeterminate); magnesium (class IIb) for torsade or hypomagnesemia; procaainamide (class IIb) for intermittent/recurrent VF/VT
Consider buffers for hyperkalemia, preexisting metabolic acidosis, some drug overdoses
Stable ventricular tachycardia (VT) monomorphic or polymorphic?

Monomorphic VT
Is cardiac function impaired?
  - Normal function
  - Medications: any one
    - Procainamide
    - Sotalol
  - Others acceptable
    - Amiodarone
    - Lidocaine

Polymorphic VT
Is QT baseline interval prolonged?
  - Normal baseline QT interval
    - Normal function QT interval
    - Medications: any one
      - Beta-blockers OR
      - Lidocaine OR
      - Amiodarone OR
      - Procainamide OR
      - Sotalol
    - Prolonged baseline QT interval
      - (suggests torsades)
  - Long baseline QT interval
    - Correct abnormal electrolytes
    - Therapies: any one
      - Magnesium
      - Overdrive pacing
      - Isoproterenol
      - Phenytoin
      - Lidocaine

Note! May go directly to cardioversion

Amiodarone
150 mg IV bolus over 10 minutes OR
Lidocaine
0.5 to 0.75 mg/kg IV push
Then use
Synchronized cardioversion
General Evaluation for Documented or Suspected VA

Resting Electrocardiogram

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

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Exercise testing is recommended in
- adult patients with ventricular arrhythmias who have an **intermediate or greater probability of having CHD** by age, gender, and symptoms to provoke ischemic changes or ventricular arrhythmias.
- patients with **known or suspected exercise-induced ventricular arrhythmias**, including catecholaminergic VT, to provoke the arrhythmia, achieve a diagnosis, and determine the patient’s response to tachycardia.
General Evaluation for Documented or Suspected VA

Ambulatory Electrocardiography

Ambulatory ECG is indicated when there is a need to clarify the diagnosis by detecting arrhythmias, QT-interval changes, T-wave alternans (TWA), or ST changes to evaluate risk, or to judge therapy.

ACC/AHA/ESC 2006 Guidelines for Management of Patients With Ventricular Arrhythmias and the Prevention of Sudden Cardiac Death
General Evaluation for Documented or Suspected VA

Left Ventricular Function and Imaging

Echocardiography is recommended in

- patients with ventricular arrhythmias who are suspected of having structural heart disease.
- for the subset of patients at high risk for the development of serious ventricular arrhythmias or SCD, such as those with dilated, hypertrophic, or RV cardiomyopathies, AMI survivors, or relatives of patients with inherited disorders associated with SCD.

ACC/AHA/ESC 2006 Guidelines for Management of Patients With Ventricular Arrhythmias and the Prevention of Sudden Cardiac Death
General Evaluation for Documented or Suspected VA

Left Ventricular Function and Imaging

Exercise testing with an imaging modality — (echocardiography or nuclear perfusion [single-photon emission computed tomography (SPECT)])—is recommended to detect silent ischemia in patients with ventricular arrhythmias who have an intermediate probability of having CHD by age, symptoms, and gender and in whom ECG assessment is less reliable because of digoxin use, LVH, greater than 1-mm ST-segment depression at rest, Wolf-Parkinson-White (WPW) syndrome, or Left Bundle Branch Block (LBBB).
Cardiac Electrophysiology Study

- Inducibility of VT
  - Reentrant (ischemic VT)
  - Triggered (idiopathic VT)
- Assessment of antiarrhythmic therapy via serial drug testing
- May lead to therapy with radiofrequency catheter ablation
Asystole

- AKA: ventricular standstill
- Result from a prolonged period of cardiac arrest without effective resuscitation
- DDx with VF
- Cause:
  - Hypovolemia
  - MI(coronary thrombosis)
  - Severe electrolyte imbalance(↑↓K)
  - Massive pulmonary embolism
  - Hypoxia
  - Drug overdose
  - Hypothermia
  - Cardiac tamponade
  - Tension pneumothorax

- No electrical activity, no contraction → cardiac output=0 → no perfusion for vital organ
ECG

- No electrical activity

**Warning**

*Recognizing asystole*

Characteristics of asystole:

- **Rhythm**: atrial rhythm — usually indiscernible; no ventricular rhythm
- **Rate**: atrial rate — usually indiscernible; no ventricular rate
- **P wave**: may be present
- **PR interval**: unmeasurable
- **QRS complex**: absent, or occasional escape beats
- **T wave**: absent
- **QT interval**: unmeasurable
- **Other**: absence of electrical activity in the ventricles results in a nearly flat line
Intervention

- Immediate Tx: CPR, oxygen and advanced airway control with intubation
- Check more than one ECG lead to confirm asystole
- IV Epi and atropine, vasopressin
- Consider terminating resuscitation if persist asystole.
Antiarrhythmic Therapy
Vaughn-Williams Classification

- Based on cellular properties of normal His-Purkinje cells
- Classified on drug’s ability to block specific ionic currents (i.e. Na⁺, K⁺, Ca+++) and beta-adrenergic receptors
- Advantages:
  - Physiologically based
  - Highlights beneficial/deleterious effects of specific drugs
Antiarrhythmic Agents
Vaughn-Williams Classification

- Class I - Na\(^+\) - channel blockers (direct membrane action)
- Class II - Sympatholytic agents
- Class III - Prolong repolarization
- Class IV - Ca\(^{++}\) - channel blockers
- Purinergic agonists
- Digitalis glycosides
Class I
Na+ Channel Blockers

- **IA** - Quinidine/Procainamide/Disopyramide
- **IB** - Lidocaine/Mexiletine/Phenytoin
- **IC** - Flecainide/Propafenone/Ethmozine

Affects Phase 0 of depolarization—the rapid inflow of sodium through the sodium channels.
Class IA - Na+ Channel Blockers
Procainamide/Quinidine/Disopyramide

- ECG changes
  - Increase PR, QRS (Diso: PR ↑ > QRS ↑ )
  - Toxicity: QTc increases by 30% or QT > 0.5 sec
  - Ca++ channel blockade / potent anticholinergic (Diso)
Arrhythmia-focused Therapy

- Class IB antiarrhythmics are very effective and very safe.
- Little or no effect on “normal” tissues.
- First line for ischemic, automatic arrhythmia's (Ventricular tachycardia)
- Not a lot of effect on normal conduction tissue – not a good medicine for reentry and atrial tachycardias.
Class IC
Flecainide/Propafenone/Ethmozine

- ECG changes
  - ↑ PR, QRS
  - ↑ QTc (Propafenone)
Arrhythmia –focused Therapy

- IC’s have a lot of side effects that make them appropriate for use only by experienced providers.
Class II Agents
Beta-blockers

- Propranolol
- Atenolol
- Metoprolol
- Nadolol
- Esmolol
- d,l-Sotalol
Arrhythmia-focused Therapy

- Beta-blockers are good for re-entry circuits and automatic dysrhythmias.
- Their effect of decreasing contractility may be limiting.
Class III
K⁺ - channel blockers

- Properties
  - Prolong repolarization
  - Prolong action potential duration
  - Contractility is unchanged or increased

- Agents
  - Amiodarone
  - Sotalol
  - Bretylium
Arrhythmia-focused Therapy

- Can be very powerful antiarrhythmics but limited indications for first-line use – beyond the spectrum of primary care providers
- Amiodarone: become a first-line medicine for a broad spectrum of arrhythmias, currently still high-risk
PULSELESS ARREST

1. BLS algorithm: Call for help, give CPR
   • Give oxygen
   • Attach monitor/defibrillator

2. Check rhythm
   Shockable rhythm?

3. VF/VT
   Give 1 shock
   • Manual biphasic: device-specific (typically 120 to 200 J)
     Note: if unknown, use 200 J
   • AED: device specific
   • Monophasic: 360 J
   Resume CPR immediately

4. Give 5 cycles of CPR*

5. Check rhythm
   Shockable rhythm?

6. Shockable

9. Asystole/PEA
   Resume CPR immediately for 5 cycles
   When IV/IO available, give vasopressor
   • Epinephrine 1 mg IV/IO
     Repeat every 3 to 5 min or
   • May give 1 dose of vasopressin 40 U
     IV/IO to replace first or second dose of epinephrine

10. Consider atropine 1 mg IV/IO
    for asystole or slow PEA rate
    Repeat every 3 to 5 min (up to 3 doses)
**Shockable**

6. **Give 1 shock**
   - Manual biphasic: device-specific (same or higher dose as first shock)
     Note: if unknown, use 200 J
   - Monophasic: 360 J
   - Resume CPR immediately
     When IV/IO available, give vasopressor
   - Epinephrine 1 mg IV/IO
   - Repeat every 3 to 5 min
     or
   - May give 1 dose of vasopressin 40 U IV/IO to replace first or second dose of epinephrine

7. Check rhythm
   - Shockable rhythm?
     - Give 5 cycles of CPR*

8. Give 1 shock
   - Manual biphasic: device-specific (same or higher dose as first shock)
     Note: if unknown, use 200 J
   - AED: device-specific
   - Monophasic: 360 J
   - Resume CPR immediately
   - Consider antiarrhythmics: amiodarone (300 mg IV/IO once, then consider additional 150 mg IV/IO once) or lidocaine (1 to 1.5 mg/kg first dose then 0.5 to 0.75 mg/kg IV/IO, maximum 3 doses or 3 mg/kg)
   - Consider magnesium, loading dose 1 to 2 g IV/IO for torsades de pointes

9. After 5 cycles of CPR, *go to Box 5 above

11. Check rhythm
    - Shockable rhythm?
      - If asystole, go to Box 10
      - If electrical activity, check pulse. If no pulse, go to Box 10
      - If pulse present, begin post-resuscitation care
        - Avoid:
          - hyperventilation
          - hypotension
          - hypo-/hyperglycemia
          - hypothermia

12. During CPR
    - Secure and search for contributing factors:
      - Hypovolemia
      - Hypoxia
      - Hydrogen ion (acidosis)
      - Hypo-/hyperkalemia
      - Hypoglycemia
      - Hypothermia
      - Toxins
      - Tamponade, cardiac
      - Tension pneumothorax
      - Thrombosis (coronary or pulmonary)
      - Trauma
    - Push hard and fast (100/min)
    - Ensure full chest recoil
    - Minimize interruptions in chest compressions
     - One cycle of CPR: 30 compressions then 2 breaths; 5 cycles approx. 2 min
    - Establish IV/IO access
    - Secure airway and confirm placement
    - After an advanced airway is placed, give 8 to 10 breaths/min without pausing compressions, avoid hyperventilation

* NOTE: After an advanced airway is placed, give about 2 min of CPR (instead of 5 cycles)
Pacemakers

- Surgical implantation of electrical leads attached to a pulse generator
- Over 175,000 implanted per year
The Pacemaker System

- Patient
- Lead
- Pacemaker
- Programmer
Pacemakers

1) Leads are inserted via subclavicle vein and advanced to the chambers on the vena cava (right) side of the heart

2) Two leads used, one for right atrium, other for right ventricle

3) Pulse generator containing microcircuitry and battery are attached to leads and placed into a “pocket” under the skin near the clavicle

4) Pulse generator sends signal down leads in programmed sequence to contract atria, then ventricles
Pacemakers

Pacemakers

- Pulse generator can sense electrical activity generated by the heart and only deliver electrical impulses when needed.
- Pacemakers can only speed up a heart experiencing bradycardia, they cannot alter a condition of tachycardia.
Internal Cardioverter Defibrillator (ICD)

The first full human implantation of an ICD occurred in February 1980. Since then, ICD technology has become extremely complex. Current ICDs not only have the ability to defibrillate, but also to pace, terminate ventricular tachycardia (VT) and provide back-up pacing for bradycardia.
Internal Cardioverter Defibrillator (ICD) (continued)

Dual chamber pacing with rate response is also now available.

The ICD is extremely expensive. The total cost with implanting may exceed $40,000.00.
Indications for ICD Implantation

The Class I indications for ICD implantation in which a broad consensus exists include:

- Cardiac arrest caused by VT or ventricular fibrillation (VF) not due to a transient or reversible cause
- Spontaneous sustained VT
- Syncope of undetermined origin with clinically relevant electrophysiologically inducible sustained VT or VF when drug therapy is ineffective or not tolerated or not preferred
Indications for ICD Implantation (continued)

• Non-sustained VT in patients with CAD, prior MI, LV dysfunction and electrophysiologically inducible VT or VF not suppressed by Class I anti-arrhythmic drugs.

_These indications are frequently updated as the results of outcome trials become available._
ACC/AHA/ESC 2006 Guidelines for Management of Patients With Ventricular Arrhythmias and the Prevention of Sudden Cardiac Death
Antiarrhythmic Drugs

♥ **Beta Blockers**: Effectively suppress ventricular ectopic beats & arrhythmias; reduce incidence of SCD

♥ **Amiodarone**: No definite survival benefit; some studies have shown reduction in SCD in patients with LV dysfunction especially when given in conjunction with BB. Has complex drug interactions and many adverse side effects (pulmonary, hepatic, thyroid, cutaneous)

♥ **Sotalol**: Suppresses ventricular arrhythmias; is more pro-arrhythmic than amiodarone, no survival benefit clearly shown

♥ **Conclusions**: Antiarrhythmic drugs (except for BB) should not be used as primary therapy of VA and the prevention of SCD
Non-antiarrhythmic Drugs

♥ **Electrolytes:** magnesium and potassium administration can favorably influence the electrical substrate involved in VA; are especially useful in setting of hypomagnesemia and hypokalemia

♥ **ACE inhibitors,** angiotensin receptor blockers and aldosterone blockers can improve the myocardial substrate through reverse remodeling and thus reduce incidence of SCD

♥ **Antithrombotic and antiplatelet agents:** may reduce SCD by reducing coronary thrombosis

♥ **Statins:** have been shown to reduce life-threatening VA in high-risk patients with electrical

♥ **n-3 Fatty acids:** have anti-arrhythmic properties, but conflicting data exist for the prevention of SCD
Therapies for VA

Ablation

Ablation is indicated in patients who are otherwise at low risk for SCD and have sustained predominantly monomorphic VT that is drug resistant, who are drug intolerant, or who do not wish long-term drug therapy.

Ablation is indicated in patients with bundle-branch reentrant VT.

Ablation is indicated as adjunctive therapy in patients with an ICD who are receiving multiple shocks as a result of sustained VT that is not manageable by reprogramming or changing drug therapy or who do not wish long-term drug therapy.

Ablation is indicated in patients with WPW syndrome resuscitated from sudden cardiac arrest due to AF and rapid conduction over the accessory pathway causing VF.
Sustained Monomorphous VT

Wide-QRS tachycardia should be presumed to be VT if the diagnosis is unclear.

Direct current cardioversion with appropriate sedation is recommended at any point in the treatment cascade in patients with suspected sustained monomorphic VT with hemodynamic compromise.
Acute Management of Specific Arrhythmias

Polymorphic VT

Direct-current cardioversion with appropriate sedation as necessary is recommended for patients with sustained polymorphic VT with hemodynamic compromise and is reasonable at any point in the treatment cascade.

Intravenous beta blockers are useful for patients with recurrent polymorphic VT, especially if ischemia is suspected or cannot be excluded.

Intravenous loading with amiodarone is useful for patients with recurrent polymorphic VT in the absence of abnormal repolarization related to congenital or acquired LQTS.
Polymorphic VT

Urgent angiography with a view to revascularization should be considered for patients with polymorphic VT when myocardial ischemia cannot be excluded.

Intravenous lidocaine may be reasonable for treatment of polymorphic VT specifically associated with acute myocardial ischemia or infarction.
Torsades de Pointes

Withdrawal of any offending drugs and correction of electrolyte abnormalities are recommended in patients presenting with torsades de pointes.

Acute and long-term pacing is recommended for patients presenting with torsades de pointes due to heart block and symptomatic bradycardia.
Torsades de Pointes

Management with intravenous magnesium sulfate is reasonable for patients who present with LQTS and few episodes of torsades de pointes. Magnesium is not likely to be effective in patients with a normal QT interval.

Acute and long-term pacing is reasonable for patients who present with recurrent pause-dependent torsades de pointes.

Beta blockade combined with pacing is reasonable acute therapy for patients who present with torsades de pointes and sinus bradycardia.

Isoproterenol is reasonable as temporary treatment in acute patients who present with recurrent pause-dependent torsades de pointes who do not have congenital LQTS.