Diagnosis of Ventricular Arrhythmia & Management of Wide Complex Tachycardia
Ventricular Arrhythmias

Uniform Ventricular Extrasystoles

Multiform Ventricular Extrasystoles

Left bundle branch block
Nonsustained VT
Tachycardia

Regular

Narrow Complex
- Sinus Tachycardia
- PSVT
- Atrial Flutter

Broad Complex
- Ventricular Tachycardia
- SVT with Aberrancy
- Preexcitation

Irregular

Narrow Complex
- Atrial Fibrillation
- Atrial Flutter with varying Block
- Multifocal AT

Broad Complex
- Polymorphic
- Torsade De Pointes
- Preexcited AF

Rule: Wide QRS tachycardia is VT until proven otherwise
Wide-Complex Tachycardia

Ventricular or Supraventricular With Aberrant Condition?
WIDE COMPLEX TACHYCARDIA

85% Ventricular Tachycardia

15% SVT with Aberrancy (functional or preexisting)

5% Preexcited Tachycardias over an accessory pathway
Diagnosis of Wide Complex Tachycardia

- A wide QRS tachycardia is VT until proven otherwise

- This is especially in patients with history of heart disease
<table>
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<tr>
<th></th>
<th>VT</th>
<th>SVT aberrant</th>
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<tbody>
<tr>
<td>AV dissociation</td>
<td>+</td>
<td>−</td>
</tr>
<tr>
<td>QRS width ≥ 0.14</td>
<td>+</td>
<td>−</td>
</tr>
<tr>
<td>QRS extreme axis</td>
<td>+</td>
<td>−</td>
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<tr>
<td>QRS morphology</td>
<td></td>
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</tr>
<tr>
<td>RBBB V₁</td>
<td>[Diagram]</td>
<td>[Diagram]</td>
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<tr>
<td>RBBB V₆</td>
<td>[Diagram]</td>
<td>R/S &lt; 1</td>
</tr>
<tr>
<td>LBBB initial rV₁₂</td>
<td>[Diagram]</td>
<td>R/S &gt; 1</td>
</tr>
<tr>
<td>Fusions, captures</td>
<td>+</td>
<td>−</td>
</tr>
<tr>
<td>Concordant pattern</td>
<td>+</td>
<td>−</td>
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</tbody>
</table>

VT: ventricular tachycardia; SVT: supraventricular tachycardia; RBBB: right bundle-branch block; LBBB: left bundle-branch block.
ECG features of VT

Captured beats or fusion beats

if present, pathognomonic of VT

Rarely seen because rate of VT must be slow, usually < 120 bpm
VT: right axis, V6 RS < 1
SVT with aberrancy: Normal axis RBBB, V6 RS > 1

Diag of Ventricular Arrhythmias & WCT
1 APRIL 2006
Preexcited AF
WPW paths & associated rhythms

FIG. 65-34. WPW paths and associated rhythms. (From Watanabe Y, Dreifus LS: Cardiac arrhythmias, New York, 1977, Grune & Stratton.)
VT degenerate to VF
Torsades de Pointes
(a type of Polymorphic VT)

Rate : Atrial not discernible; ventricular rate extremely rapid and irregular
Morphology: Abnormal looking & constantly changing QRS complexes .
Gradually shifting electrical axis (twisting of points)
Sinus rhythm shows prolong QT
Onset: Often starts as a short cycle following a long cycle
Long QT - QT alternans - Torsades
Torsades de Pointes

Management:

- Discontinuation of offending drugs
- Magnesium sulfate
- Overdrive pacing
Wide Complex Tachycardia

Artifact VT
Tachycardia Algorithm

- Assess responsive
- Call for help/defibrillator
- Assess ABCs
- Administer oxygen
- Establish IV

- ECG monitor
- Assess vital signs
- Review history
- Perform physical examination
- Do 12 Lead ECG

Unstable, with serious signs or symptoms
ie: Heart Failure, SBP<90, in shock

Yes
Immediate synchronised Cardioversion

No

- Narrow Complex Tachycardia
- Wide Complex Tachycardia
- Polymorphic VT
Ventricular Tachycardia

- Hemodynamically unstable
  - Synchronized cardioversion

- Once rhythm has converted, provide antiarrhythmic therapy
Wide Complex Tachycardia

1. Wide Complex Tachycardia
   - Adenosine 6mg rapid iv push
   - Note: Blood Pressure low proceed to immediate synchronised cardioversion
   - Adenosine 12mg rapid iv push
   - Lignocaine 50-100 mg IV push
   - If still VT, synchronised cardioversion
   - Amiodarone 150 mg IV push over 10 mins

2. Polymorphic VT
   - If suspect VT
     - Lignocaine 50-100 mg IV push
   - Amiodarone 150 mg IV push over 10 mins

Medications:
- Magnesium
- Consider overdrive pacing

Correct abnormal electrolytes
Treat ischemia if present
Hypotension
Shock
Pulmonary Edema
Hypotension / Shock

Shock :
Inadequate cellular perfusion and inadequate oxygen delivery for existing metabolic demand
Mean Arterial Pressure:

Cardiac Output \times Total Peripheral Resistance

Stroke Volume \times Heart Rate

Venous Return (Blood Volume)

Contractility

MAP falls if

Heart rate
Blood volume
Contractility
Peripheral vascular resistance
Clinical Manifestation of Shock

Compensatory Mechanism (Adrenergic Mediators)
- Tachycardia
- Diaphoresis, anxiousness, nausea, vomiting, diarrhoea
- Cool and moist skin
- Oliguria

Decompensating
- Altered mentation
- Myocardial ischaemia
Hypotension / Shock Algorithm

- Assess danger / responsiveness
- Call for help / defibrillator
- Assess ABCs
- Administer Oxygen
- Establish IV
- ECG monitoring
- Assess vital signs
- Record history
- Perform physical examination
- Do 12 Lead ECG
- Do portable CXR

What is the nature of the problem

- Volume / Vascular resistance problem
- Rate Problem
- Pump Problem
<table>
<thead>
<tr>
<th>Rate Problem</th>
<th>Pump Problem</th>
<th>Volume Problem</th>
<th>Vascular Resistance Problem</th>
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<tr>
<td>Bradycardia</td>
<td>Intracardiac</td>
<td>Haemorrhage</td>
<td>Sepsis</td>
</tr>
<tr>
<td>Tachycardia</td>
<td>a) Myocardium MI Myocarditis</td>
<td>Dehydration GI Loss Renal Loss</td>
<td>Spinal Injury</td>
</tr>
<tr>
<td></td>
<td>b) Valvular Pap muscle dysfunction</td>
<td></td>
<td>Adrenal insufficiency</td>
</tr>
<tr>
<td></td>
<td>c) Rupture Chordae VSR</td>
<td></td>
<td>Anaphylaxis</td>
</tr>
<tr>
<td></td>
<td>Extra cardiac Cardiac Tamponade Pulmonary Embolism</td>
<td>Hypotension / Shock Algorithm</td>
<td></td>
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What is the blood pressure?

- **SBP<70 mmHg**
  - Signs and symptoms of shock
  - **IV Dopamine** 5-20 μg/kg/min
    - Add **IV Noradrenaline** 0.5 to 30 μg/min if Dopamine>20μg/kg/min

- **SBP 70-100 mmHg**
  - Signs and symptoms of shock
  - **IV Dopamine** 2-20 μg/kg/min

- **Systolic BP 70-100 mmHg**
  - No signs and symptoms of shock
  - **IV Dobutamine** 2-20 μg/kg/min
Volume / Vascular resistance problem

Fluid / blood transfusion
Consider vasopressor
(vascular resistance problem)
Cause-specific intervention

Hypotension / Shock Algorithm
Pulmonary Oedema

Cardiogenic pulmonary oedema: Increased pulmonary venous pressure (LVEDP $\geq 18$ mmHg) due to altered pump function, rhythm or excess fluid.

Non-cardiogenic pulmonary oedema: normal pulmonary venous pressure due to altered permeability of alveolar or pulmonary capillary endothelium.
Acute Pulmonary Oedema Algorithm

- Assess danger / responsiveness
- Call for help / defibrillator
- Assess ABCs
- Sitting position / legs dependent
- Administer oxygen
- ECG monitoring
- Establish IV

- Assess vital signs
- Review history
- Perform physical examination
- Do 12 Lead ECG
- Do portable CXR

Volume problem
Rate Problem
Pump Problem
Pump Problem

What is the blood pressure?

First line Actions
- Frusemide IV 0.5-1.0mg/kg
- Morphine IV 1-3 mg
- Nitroglycerin S/L
- Oxygen / Intubation CPAP/PEEP

Second line Actions
- Nitroglycerine IV or nitroprusside IV or dobutamine IV if SBP > 100 mmHg
- Dopamine of BP < 100 mmHg

Third line Actions
- Thrombolytic therapy (if not in shock)
- Angioplasty
- IABP, Surgery (valves, CABG)
Management of Acute Pulmonary Oedema (I)

1. Loop diuretics
   Immediate decrease in venous tone
   (increase in renal capacitance)
   Increase in renal water excretion, reaches a peak in 30 minutes
   Rapid onset of actions (5 to 10 minutes)

2. Morphine:
   Central sympatholytic effect causing peripheral vasodilation
   ↓ central venous return (↓ preload)
Management of Acute Pulmonary Oedema (2)

3. Nitrates
   - Potent venodilatory (low dose)
   - Dilate arterial resistance vessel (high dose)

4. Positive pressure ventilation
   - Useful in non-cardiogenic pulmonary oedema
   - Respiratory insufficiency
Search for underlying causes of HF

event precipitating acute decompensation
  eg : AMI, salt overload,
  medication non-compliance,
  NSAIDs, arrhythmia
Scenario

55 years old man presented after 2 hours of severe substernal chest pain and discomfort in his left arm

Non responsive

Vital signs BP 50/30, pulse 40/min, RR 16/min
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Inferior & RV infarct
• Is there a rate problem?
• Is there a pump problem?
• Is there a volume problems?
• How would you treat this patient?
Definitive treatment with a transvenous pacemaker depends on the type of block

- Sinus or first-degree needs no pacing
- Second-degree type I (usually nodal) possibly needs temporary pacing
- Second-degree type II (usually infranodal) has unpredictable need for pacing
- Third-degree (usually infranodal) generally needs pacing
**Scenario**

62 year old Indian man  
Complained of chest tightness, giddiness and sweating

Cold and clummy, conscious but drowsy  
Raised JVP  
HR 48/min,  BP 84/40 mmHg  
Heart S1 S2 S4 gallop  
Systolic murmur at left parasternal edge  
Lungs were clear  
Liver 3 cm

ECG as shown  
Raised CKMB and troponin T
Inferior, lateral & RV infarct
Complicated by complete heart block and hypotension

RV pump failure, suboptimal heart rate
Congested right heart
Diminished LV filling and low C.O
No significant pulmonary congestion
Treatment

Oxygen
IV fluid infusion
IV dopamine, IV atropine
May need temporary cardiac pacing
Avoid over zealous use of diuretics
Avoid vasodilators
Aspirin, plavix, s/c heparin
± revascularization