Premature Ventricular Contraction

- Groups of pacemaker cells throughout the conducting system are capable of spontaneous depolarization.
- The rate of depolarization decreases from top to bottom: fastest at the sinoatrial node; slowest within the ventricles.
- Ectopic impulses from subsidiary pacemakers are normally suppressed by more rapid impulses from above.
- However, if an ectopic focus depolarizes early enough — prior to the arrival of the next sinus impulse — it may “capture” the ventricles, producing a premature contraction.
- Premature contractions ("ectopics") are classified by their origin — atrial (PACs), junctional (PJC s) or ventricular (PVCs).
Premature Ventricular Contraction

ECG Features:
- Broad QRS complex ($\geq 120$ ms) with abnormal morphology.
- Premature — i.e. occurs earlier than would be expected for the next sinus impulse.
- Discordant ST segment and T wave changes.
- Usually followed by a full compensatory pause.
- Retrograde capture of the atria may or may not occur.

Classification
- **Unifocal** — Arising from a single ectopic focus; each PVC is identical.
- **Multifocal** — Arising from two or more ectopic foci; multiple QRS morphologies.
Premature Ventricular Contraction

- Arise within an ectopic focus within the ventricular
- No preceding P wave, T wave opposite direction of the QRS,
- Multiform PVCs: different contours; Multifocal PVCs: different origin
- Pattern:
  - Bigeminy: one normal QRS followed by PVC;
  - Trigeminy: 2 sinus QRS followed by PVC;
  - Quadrigeminy: 3 sinus QRS followed by PVC
  - Couple: two consecutive PVCs.
  - Triplet: three consecutive PVCs.

Causes:
- CAD, CHF, electrolytes imbalance, hypokalemia, anxiety, stress, caffeine, alcohol

Treatment:
- Treat the cause, Lidocaine, Bretylium
Premature Ventricular Contraction

**Causes:**
- Anxiety
- Sympathomimetics
- Beta-agonists
- Excess caffeine
- Hypokalemia
- Hypomagnesaemia
- Digoxin toxicity
- Myocardial ischemia
- CHF,
- Caffeine, alcohol

**Treatment:**
- Treat the cause, Lidocaine, Bretylium
Different PVCs Rhythm
What do you see??
Ventricular Tachycardia

Clinical Significance

• Ventricular tachycardia may impair cardiac output with consequent hypotension, collapse, and acute cardiac failure. This is due to extreme heart rates and lack of coordinated atrial contraction (loss of “atrial kick”).

• The presence of pre-existing poor ventricular function is strongly associated with cardiovascular compromise.

• Decreased cardiac output may result in decreased myocardial perfusion with degeneration to VF.

• Prompt recognition and initiation of treatment (e.g. electrical cardioversion) is required in all cases of VT.
Ventricular Tachycardia

- Ventricular Tachycardia (VT) is a broad complex tachycardia originating in the ventricles.

Classification according to:

1. Morphology
   - Monomorphic: a rise from one focal point, giving same shape
Ventricular Tachycardia

Classification according to:

1. **Morphology**
   - Polymorphic and Torsades de pointes: a rise from different focal points, giving different shapes
Ventricular Tachycardia

Classification according to:

2. Duration

- Sustained = Duration > 30 seconds or requiring intervention due to hemodynamic compromise.
- Non-sustained = Three or more consecutive ventricular complexes terminating spontaneously in < 30 seconds.
Ventricular Tachycardia

Classification according to:

3. Clinical Presentation

• Hemodynamically stable.
• Hemodynamically unstable — e.g. hypotension, chest pain, cardiac failure, decreased conscious level.
Ventricular Tachycardia

Mechanisms:
1. **Reentry**
   - Commonest mechanism.
   - Develops due to abnormal myocardial scarring usually due to prior ischemia or infarction.

2. **Triggered Activity**
   - Occurs due to early or late after-depolarisations.
   - Examples include Torsades and digitalis toxicity.

3. **Abnormal Automaticity**
   - Accelerated abnormal impulse generation by a region of ventricular cells.
Ventricular Tachycardia

**Features suggestive of VT**
- Very broad complexes (>160ms).
- Absence of typical RBBB or LBBB morphology.
- Extreme axis deviation ("northwest axis") — QRS is positive in aVR and negative in I + aVF.
- Rate: 150 – 250 bpm
- Rhythm: mostly regular
- P wave usually hidden in the QRS

**Causes:**
- CAD, CHF, MI, electrolytes imbalance, digoxin, quindine, thyroid medications,

**Treatment:**
- consider cardioversion or defibrillation
Ventricular Fibrillation

- Ventricular fibrillation (VF) is the most important shockable cardiac arrest rhythm.
- The ventricles suddenly attempt to contract at rates of up to 500 bpm.
- This rapid and irregular electrical activity renders the ventricles unable to contract in a synchronized manner, resulting in immediate loss of cardiac output.
- The heart is no longer an effective pump and is reduced to a quivering mess.
- Unless advanced life support is rapidly instituted, this rhythm is invariably fatal.
- Prolonged ventricular fibrillation results in decreasing waveform amplitude, from initial coarse VF to fine VF and ultimately degenerating into asystole due to progressive depletion of myocardial energy stores.
Ventricular Fibrillation

Causes

• Myocardial ischemia / infarction
• Electrolyte abnormalities
• Cardiomyopathy (dilated, hypertrophic, restrictive)
• Long QT (acquired / congenital) causing TdP → VF
• Drugs (e.g. verapamil in patients with AF+WPW)
• Environmental – electrical shocks, drowning, hypothermia
• Pulmonary embolism
• Cardiac tamponade
• **Treatment**: immediate electrical defibrillation and start CPR, consider Epinephrine 1mg repeated 3-5 minutes
Asystole

- Rate: no atrial or ventricular activity
- No distinguished ECG waves or intervals; flat line
- Treatment: start CPR (see app-6)
Cessation of CPR???

• The decision to stop CPR should be tailored according to the specifics of the individual case and is based on clinical judgment.
• The decision is best made by the Team Leader in consultation with other team members.
• Always maintain initial resuscitation efforts until adequate information is available to make the call to discontinue.
Cessation of CPR???

- Reasons to cease CPR generally include:
- ROSC: resuscitation guidelines require 2 min of CPR post defibrillation prior to checking for ROSC; may be identified by an upsurge in ETCO2)
- pre-existing chronic illness preventing meaningful recovery (i.e. nursing home resident with dementia, disseminated cancer)
- acute illness preventing recovery (i.e. 100% burns, non-survivable injuries, catastrophic TBI with no brain stem reflexes)
- no response to ACLS after 20min of efficient resuscitation in absence of ROSC, a shockable rhythm or reversible causes
Questions and answers