general:
- VT is defined as three more VEB at a rate greater than 130bpm and may exceed 300bpm
- VT lasting over 30 seconds is sustained

monomorphic VT
- most common form of VT
- most commonly associated with myocardial infarction
- most common mechanism is re-entry secondary to inhomogenous activation of the myocardium and slow conduction through scar tissue
- AV dissociation is present in 76% of cases

polymorphic VT and torsades de pointes
- has QRS complexes at 200bpm or more which change in amplitude & axis so that they appear to twist around the baseline
- torsades de pointes usually has a prolonged QT during sinus rhythm; however, polymorphic VT may be associated with a normal QT interval in settings such as myocardial ischaemia and post-cardiac surgery

mechanisms of ventricular tachycardia:
- (i) abnormalities in impulse generation
  - involves enhanced automaticity (ectopic pacemaker activity) or triggered activity (action potentials that result from after depolarisations
  - (ii) abnormalities in impulse conduction (re-entry)
  - re-entry is a phenomenon in which a normally propagating impulse reenters previously excited tissue after its refractory period if over and excites it again
  - several forms of re-entry have been described including circus movement re-entry, phase 2 re-entry & reflection

predisposing conditions:
- (i) channelopathies:
  - diseases in which there are abnormalities of proteins forming ion channels
  - most hereditary channelopathies so far described involve mutations in genes that encode for Na and K channels.
- examples include: Long-Nielsen syndrome (a long QT syndrome associated with deafness), Romano-Ward syndrome (a long QT syndrome not associated with deafness), & Brugada syndrome
  - (ii) other primary electrophysiological defects:
    - WPW
    - catecholamine-sensitive polymorphic VT
  - (iii) drugs that prolong the QT interval:
    - www.qtdrugs.org is an up-to-date list of all such drugs
    - examples include: Ic antiarrhythmics, antibiotics such as clarithromycin and erythromycin, complex anti-arrhythmics, severe hypoxia and non-synchronised DC cardioversion
  - (iv) electrolyte abnormalities:
    - hypokalaemia prolongs the QT & increases risk of arrhythmia
    - hyperkalaemia increases excitability & can precipitate arrhythmia
    - hypomagnesaemia is associated with prolonged QT & increases risk of arrhythmia
    - hypocalcaemia increases that QT interval and predisposes to VT
  - (v) hypothermia:
    - hypothermia lengthens the QT interval and predisposes to VT
    - also causes J waves (also known as Osborn waves)
  - (vi) structural heart disease:
    - LV dysfunction
    - coronary artery disease and myocardial infarction
    - hypertrophic cardiomyopathy

causes of long QT
- Genetic:
  - mutations in the genes for the ion channels that regulate the QT interval
- Environmental:
  - drugs that prolong the QT interval
  - electrolyte imbalances
  - systemic conditions
- Drugs:
  - QT-prolonging medications
- Structural:
  - cardiomyopathies
- Cardiac:
  - myocardial ischemia
- Non-cardiac:
  - hypothermia

factors facilitating proarrhythmia with antiarrhythmic drugs
- DC shock is indicated if a patient is haemodynamically unstable
- drug therapy is indicated for haemodynamically stable monomorphic VT:
  - (i) amiodarone: may terminate VT but is negatively inotropic
  - (ii) sotalol and procainamide are more effective than lignocaine
  - but are associated with significant myocardial depression
  - (iii) lignocaine is traditionally indicated
- NB: using two antiarrhythmic drugs is discouraged because of potential for a proarrhythmic effect
  - magnesium is recommended for torsades de pointes
  - electrical storm is a highly lethal phenomenon with recurrent episodes of VF occurring in the context of an acute AMI. The mechanism seems to be excessive sympathetic activity and recent studies have shown that iv beta blockers can be effective therapy