wide complex tachycardia [created by Paul Young 14/10/07]

differentiation of VT from wide complex SVT

criteria for diagnosis of VT using the 4-step Brugada algorithm:
(i) Is RS complex present in any lead?
- if not the rhythm is VT
(ii) Is the RS duration >100ms in any lead?
- if yes then the rhythm is VT
(iii) Is there AV dissociation?
- if yes then the rhythm is VT
(iv) Is the rhythm morphologically consistent with SVT?
- if not the rhythm is VT
  - if p waves are not visualised, a Lewis lead (arm electrode positioned on the parasternal area) or an oesophageal lead can be used

accelerated idioventricular rhythm (AIVR)

ecg:
- wide QRS with a rate of 60-110bpm
- sinus rhythm is often only slightly slower than the arrhythmia so the dominant rhythm may be intermittent AIVR and sinus rhythm
- fusion beats are therefore common

clinical:
- commonly encountered in inferior AMI
- occasionally causes haemodynamic deterioration usually due to loss of atrial systole
- can be overriden by increasing the atrial rate with pacing or atropine

ventricular fibrillation

general:
- always causes haemodynamic collapse, loss of consciousness & death if not immediately treated
- of patients resuscitated from VF, 20-30% have sustained an acute myocardial infarction & 75% have coronary artery disease

ecg:
- ECG shows irregular waves of varying morphology & amplitude

clinical:
- VF is usually associated with IHD; other causes include cardiomyopathy, anti-arrhythmics, severe hypoxia and non-synchronised DC cardioversion

treatment:
(i) cardioversion
(ii) if DC cardioversion fails amiodarone is most widely used 2nd line agent

RBBB

- activation of the right ventricle is delayed

ecg:
- QRS >120ms
- RSR in V1
- broad S-wave in the left ventricular leads especially I & V6

clinical:
- a normal variant but may occur with massive pulmonary embolism, right ventricular hypertrophy, ischaemic heart disease and congenital heart disease

LBBB

- in LBBB the interventricular septum is activated from right to left

ecg:
- QRS >120ms
- M-shaped in V6
- Q waves never seen in left ventricular leads

clinical:
- LBBB is associated with heart disease such as coronary artery disease, cardiomyopathy or left ventricular hypertrophy

factors facilitating proarrhythmia with antiarrhythmic drugs

Toxic blood levels due to excessive dose or reduced clearance from old age, heart failure, renal disease or hepatic disease
Severe left ventricular dysfunction. Ejection fraction less than 35%
Pre-existing arrhythmia or arrhythmia substrate
Digoxin therapy
Hypokalaemia or hypomagnesaemia
Bradycardia
Combinations of anti-arrhythmic drugs and concomitant drugs with similar toxicity

ventricular tachycardia

general:
- VT is defined as three of 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 75% 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 assoicated 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: Lange-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, antipsychotics such as haloperidol, tricyclic antidepressants, antihistamines such as terfenadine, opiate agonists such as methadone, enterokinetic agents such as cisapride, droperidol and domperidone
(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

Acquired
- Drugs
  - Class IA anti-arrhythmic drugs:
    - Quinidine, procainamide
  - Class III anti-arrhythmic drugs:
    - Amiodarone, sotalol
  - Tricyclic antidepressants
  - Macrolide antibiotics
  - Phenothiazines
  - Anti-histamines
  - Cisapride
- Myocardial ischaemia/infarction
- Hypokalaemia
- Cardiomyopathy
- Acute myocarditis
- Mitral valve prolapse
- Acute cerebral injury
- Hypothermia

Idiopathic
- Familial: 90%
  - Linked to a DNA marker on the short arm of chromosome 11
  - Autosomal dominant in most cases.
  - Some cases linked to congenital deafness and autosomal recessive
- Sporadic: 10%
  - Non-familial related to new gene mutation.

treatment:
- 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 occuring 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