
supraventricular tachycardia [created by Paul Young 09/10/07]
general
- SVTs are any tachycardia that require atrial or AV nodal tissue for their initiation & maintenance
- SVTs are usually narrow complexes but may be wide complex in the setting of bundle branch block or pre-excitation
- in critical illness the continuing arrhythmogenic and chronotropic factors make rate control of SVTs difficult
- digoxin often results in poor rate control in critical illness (although it may have minor inotropic and vasopressor effects that are beneficial)
- magnesium provides effective rate control and may revert SVT
- amiodarone may be useful for rate control
- cardioversion is indicated in unstable patients but is more likely to maintain SR if loading with an anti-arrhythmic such as amiodarone has been performed prior
classification
AV node dependent (junctional tachycardias):
(i) AV nodal re-entrant tachycardia (re-entry within the AV node)
(ii) AV re-entry tachycardia (re-entry includes accessory pathway between atria and ventricles)
(iii) accelerated idio-nodal rhythm (increased automaticity of AV node)
AV node independent:
(i) atrial flutter (re-entry confined to atria)
(ii) atrial fibrillation (multiple re-entry circuits confined to atria)
(iii) unifocal atrial tachycardia (usually due to increased automaticity)
(iv) multifocal atrial tachycardia (increased automaticity or triggered activity)
(v) others (sinus node re-entry tachycardia)
AV nodal re-entrant tachycardia
general:
- re-entry is confined to the AV node with antegrade conduction to the ventricles usually occurring over the slow (alpha) pathway and retrograde conduction over the fast (beta) pathway
ecg:
- regular narrow complex tachycardia of 140-220bpm with abrupt onset and termination
- p waves are not usually observed as they are buried in the QRS complex
clinical:
- AVRNT is a common arrhythmia that is usually not associated with structural heart disease
treatment:
(i) vagal manoeuvres slow conduction through the AV node & may 'break' the tachycardia
(ii) adenosine the drug of choice if carotid sinus massage fails
(iii) verapamil has been used but causes hypotension
(iv) sotalol, amiodarone & flecainide may all be used
(v) rapid atrial pacing is effective
(vi) cardioversion may be used
AV re-entrant tachycardia
general:
- the re-entry pathway consists of the AV node and accessory pathway which bypasses the AV node
- the accessory pathway may be evident during sinus rhythm with the ECG showing pre-excitation; however, in 25% of cases the accessory pathway conducts only retrogradely so pre-excitation doesn't occur
- orthodromic AVRT with antegrade nodal and retrograde accessory pathway circuit is the most common regular SVT in patients with an accessory pathway
ecg:
- similar to AVNRT; however, the accessory AV pathway is further from the AV node and retrograde p waves may be evident
clinical:
- AVRT is similar to AVNRT although antegrade conduction over the accessory pathway may be very rapid if AF occurs in WPW
treatment:
- acute treatment is the same as for AVNRT except that verapamil should be avoided because of the risk of blocking the AV node leading to rapid conduction down the accessory pathway
accelerated idionodal rhythm
general:
- increased automaticity of the AV junction above its usual inherent discharge rate of 40-60bpm is the usual cause of this rhythm
ecg:
- there are narrow complexes on the ECG at regular rate (60-130bpm) often with independent atrial activity
- AV dissociation may be present or there may be synchronisation of the two pacemakers giving isorhythmic dissociation with QRS either fixed just after the QRS or oscillating to and fro across the QRS in a rhythmic manner
clinical:
- may be observed in normal persons but is often associated with structural heart disease, especially following inferior AMI
- digoxin intoxication is another important cause
treatment:
- in most cases no treatment is required as the rhythm is transient
- treatment is otherwise directed to the underlying cause
atrial fibrillation
general:
- most common arrhythmia requiring treatment
- incidence increases with age
- AF is common in:
(i) CCF (40%)
(ii) post CABG (25-50%)
(iii) critical illness (15%)
ecg:
- atrial activity is chaotic (300-600bpm) with irregular depolarisations
- ventricular response is irregularly irregular with most atrial responses not conducted to the ventricle so that the ventricular rate is 100-180bpm
clinical:
- more common in patients with underlying heart disease (particularly those with a dilated left atrium)
- causes include:
(i) ischaemic and valvular heart disease
(ii) hypertension
(iii) cardiac failure
(iv) thyrotoxicosis
(v) alcohol abuse
- AF is associated with adverse haemodynamic effects, risk of systemic embolism and risk of tachycardiomyopathy
treatment:
- goals of treatment are ventricular rate control, anticoagulation +/- conversion to sinus rhythm
atrial flutter
general:
- atrial rate is 250-350 (usually close to 300)
- due to a re-entry circuit lying within the right atrium
- divided into two subtypes:
(i) type I flutter is slower with an atrial rate of 240-320 and is readily entrained with overdrive pacing
(ii) type II fluuter is faster with an atrial rate of 340-430 and is not terminated by pacing
ecg:
- flutter waves are best seen in V1, or aVF but II and III may also be useful
- AV conduction block at 2:1 is usually present
- 1:1 conduction occurs rarely (usually in association with sympathetic overactivity or class I antiarrhythmics)
clinical:
- may occur in ischaemic heart disease, cardiomyopathy, rheumatic heart disease, thyrotoxicosis and after cardiac surgery
treatment:
- no drug will reliably terminate atrial flutter although increasing AV block will slow the rate (options include digoxin, diltiazem, beta blockers, sotalol and amiodarone)
- flecainide and procainamide are occasionally effective at terminating atrial flutter; however, may lead to 1:1 block
- synchronised cardioversion with 25-50J is a reliable treatment option
- rapid atrial pacing will terminate type I atrial flutter in most patients
multifocal atrial tachycardia
general:
- irregular narrow complex tachycardia with varying p wave morphologies associated with chronic lung disease
ecg:
- rate is usually 100-130bpm with at least 3 p wave morphologies in the same ECG trace and an irregular rate
- most p waves are usually conducted to the ventricles with narrow complexes
clinical:
- occurs most often in critically ill elderly patients with chronic lung disease and cor pulmonale
- theophylline has been implicated as a precipitating cause
treatment:
- treatment should correct underlying cause (eg cardiorespiratory failure, electrolyte disturbance, acid base disturbance & theophylline toxicity)
unifocal atrial tachycardia
general:
- sometimes called ectopic atrial tachycardia
ecg:
- p wave morphology is often abnormal but monomorphic atrial rate is often 130-160bpm and may exceed 200bpm
- AV block is common
clinical:
- digoxin toxicity is the most common cause especially when AV block is present
- other causes include myocardial infarction, chronic lung disease & metabolic disturbance
treatment:
- if applicable digoxin is stopped and toxicity is treated; otherwise it may be used to control ventricular rate
- blockers or amiodarone are alternatives
- overdrive pacing may be ineffective but may slow ventricular rate by increasing AV block
- synchronised cardioversion may be required but should be avoided with digoxin toxicity