Topic Overview: Cardiac Module
Sub-Module: C6- Structured Approach to Arrhythmias and Tachyarrhythmias
Last updated August 3 2012

This handout supports the simulation session of the same name comprising a presentation followed by a team based pause-and-discuss simulation scenario. Use this document as preparation for the session and reflection of the simulation.

Topic Objectives

1. Use a structured approach to rapid assessment of cardiac arrhythmias
2. Apply the 'unstable' versus 'stable' decision tree to early management
3. Review common tachyarrhythmias
4. Identify pre-malignant tachyarrhythmias (indications for synchronised electrical cardioversion)
5. Perform electrical and chemical cardioversion
6. Rehearse synchronised electrical cardioversion

A: STRUCTURED APPROACH TO THE PATIENT WITH AN ARRHYTHMIA (Repeated from C5)

What is the structured clinical approach to arrhythmias?

Arrhythmias occur commonly, varying between patients and types of arrhythmias in their potential to cause serious morbidity and death. While the consequences of inappropriate or delayed treatment can be substantial assessment is complicated by the range of presentations and available treatment options. A methodical approach is assessment is essential. This involves three steps and questions:

Structured clinical approach to arrhythmias

1. Apply the "unstable v stable" rule – Is the patient hemodynamically compromised?
2. Use a methodical approach to diagnose the rhythm – What is the arrhythmia?
3. Seek the underlying cause and contributors – What is causing the arrhythmia?

The foremost clinical question when assessing a patient with an arrhythmia is not "What is the definitive cardiac rhythm?" but "Is the cardiac arrhythmia causing hemodynamic instability?"

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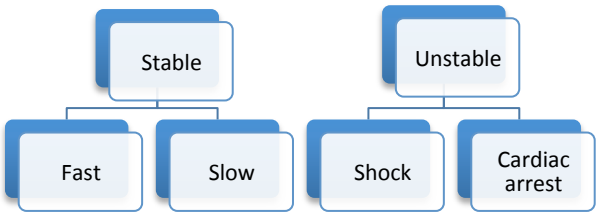
Applying the "Stable or Unstable" rule?

Frequently, senior clinicians will ask you if the patient is stable or unstable. How is stability evaluated? This decision is made on clinical evidence of reduced cardiac output (CO) readily available at the bedside which classifies CO into one of three functional categories:

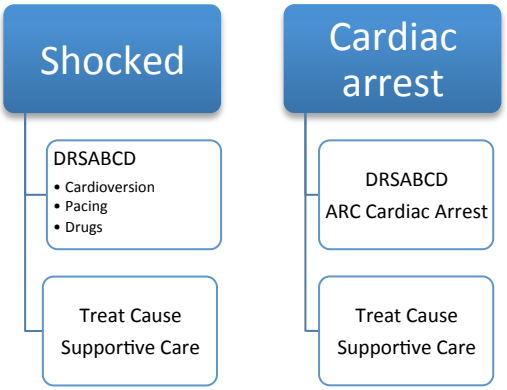
1. **STABLE** –CO adequate to perfuse end organs
2. **UNSTABLE** – SHOCK - CO inadequate to perfuse end organs. Criteria:
   - ❑ Altered level of consciousness, confusion
   - ❑ Chest pain
   - ❑ Tachypnoea, dyspnoea from acute left ventricular failure
   - ❑ BP< 90mmHg or more than 20% fall from baseline
   - ❑ Heart rate > 150 bpm
3. **CARDIAC ARREST** – No CO

The unstable patient has evidence of inadequate end-organ perfusion subsequent to reduced cardiac output. The most obvious are the brain and the heart but also affected include kidneys, liver, GIT and other tissues. The symptoms and signs suggestive of reduced cardiac output to the brain and heart are listed as criteria in the box above.



Management of the Unstable Patient

Management is determined by the Cardiac Output in one of three functional categories previously described. Patients in cardiac arrest need to be resuscitated according to the Australian Resuscitation Councils guidelines whereas unstable patients generally need a combination of pharmacological therapy and either DC cardioversion or pacing for tachydysrhythmias and bradydysrhythmias respectively as shown in the following diagram:



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B: TACHYARRHYTHMIAS

To reiterate, assessment is guided by the structured approach as follows:

1. Apply the "unstable v stable" rule
2. Use a methodical approach to diagnose the rhythm
3. Seek the underlying cause and contributors

Tachyarrhythmias - Approach to the stable patient.

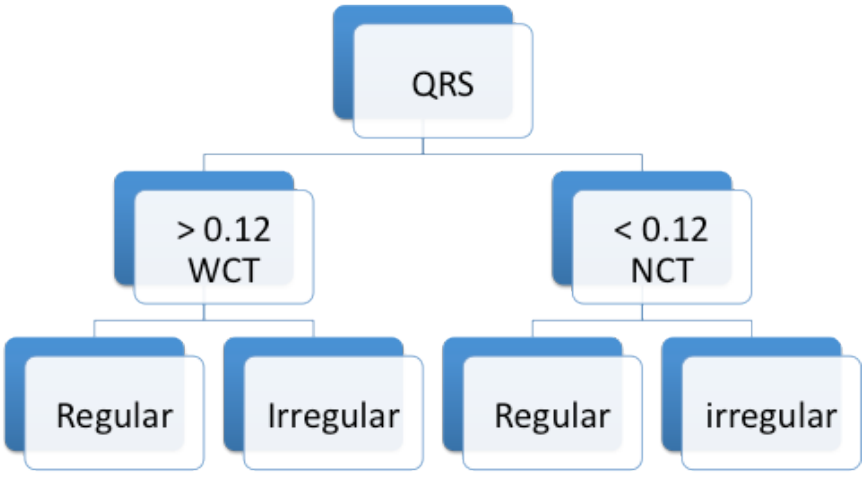
There is a group of tachyarrhythmias where the patient is not haemodynamically compromised initially. Hence there is some time available to:

- Review the arrhythmia systematically
- Look for the underlying causes
- Determine the best course of action

Note that in general, any rate greater than 150/min requires urgent treatment even if the patient does not have symptoms or signs consistent with a low cardiac output (hence classified as being unstable). In the elderly or those at risk of cardiac embarrassment, dropping this "alert threshold" to 120/min is recommended.

A methodical approach to diagnosing rhythm: The ECG decision tree

1. **Narrow or Wide Complex?** The first question allows us to categorise the arrhythmia into wide or narrow complex. If the QRS interval is less that 0.12 sec (three small squares in a standard rate ECG) then it is a narrow complex tachycardia (NCT). If the QRS is wider than 0.12 seconds then it is a wide complex tachycardia (WCT).
2. **Regular or Irregular?** The next question, that helps us to diagnose the tachyarrhythmia, is whether the rhythm is regular or irregular.



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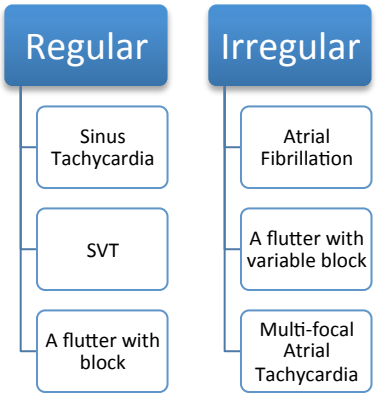
Narrow Complex Tachycardias (NCTs)



Regular NCTs:

This includes: Sinus Tachycardia, Supraventricular Tachycardia (SVT) and Atrial flutter with regular atrioventricular blockade.

Atrial flutter is technically a supraventricular tachycardia. Making it a separate entity is related to its acute management. In distinguishing atrial flutter with a regular block from other SVTs, the key is the rate. Atrial flutter with regular block occurs at predictable rates, which are fractions of the atrial rate of 300/min.

- Atrial flutter with 1:1 conduction from atrium to ventricle occurs at a rate of 300/min
- Atrial flutter with 2:1 blockade has a ventricular rate of 150/min
- Atrial flutter with 3:1 blockade has a ventricular rate of 100/min

Hence if the rate for NCTs is exactly any of the above with no beat-to-beat variation, then atrial flutter rather than other SVTs needs to be considered as the arrhythmia in question.

Irregular NCTs:

The commonest arrhythmia in this group is atrial fibrillation with a rapid ventricular rate. Not infrequently, the monitor or the 12 lead ECG will capture saw-tooth waves that are fleeting in appearance. In this situation, the arrhythmia is often termed “flutter-fibrillation”. It has implication for treatment as the patient is managed as per atrial fibrillation with the concern for anticoagulation factored in (see later).

Atrial flutter can also be conducted irregularly due to inconsistent block at the atrio-ventricular node. This arrhythmia is called atrial flutter with variable block.

Supraventricular Tachycardias (SVT)

This group contains a number of arrhythmias distinguished by the ventricular rate and the effect of the drug Adenosine. The P waves are often difficult to see and hence using this as a clinical pointer is unrealistic.

AVNRT – atrioventricular node reentry tachycardia

AVRT – atrioventricular reentry tachycardia with an accessory pathway.

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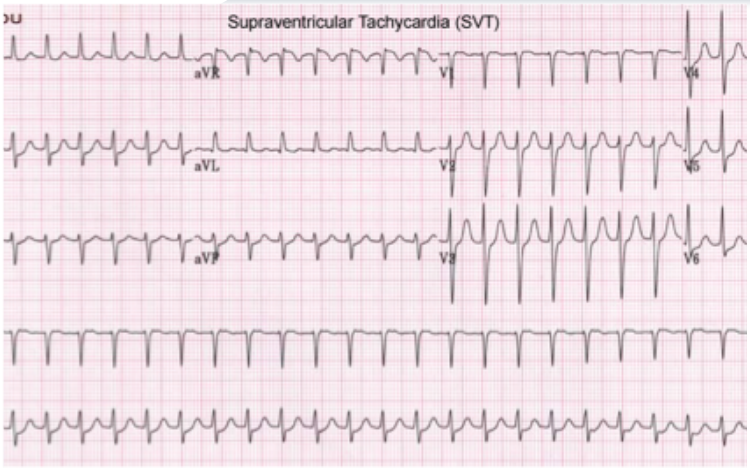

Rhythm	Origin	P wave	Atrial rate	Ventricular rate	Effect of adenosine
AVNRT	AV node	Within QRS	180-250	180-250	Stops
AVRT with accessory pathway Orthodromic conduction	AV node	RP interval is less than PR interval	150-250	150-250	Stops
Atrial tachycardia	Atria	Precedes QRS	120-250	75-200	Temporary AV node blockage
AV junctional tachycardia	AV node	RP interval is less than PR interval	60-100	60-100	Decreases the rate

Management of SVTs

Unstable patient

- Synchronised cardioversion with sedation

Stable patient

- Vagal manoeuvre
  - Valsalva manoeuvre in the supine patient is most effective
  - Carotid sinus massage is not recommended in elderly patients or those with a carotid bruit. Success rate is 20-25%
  - Can be done after drug treatment
- Adenosine: this is the first line drug used for NCT
  - Adenosine acts at the AVN and is said to convert > 90% of re-entrant SVT
  - It has no sustained anti-arrhythmic effects
  - Recurrence may occur in up to 25% of patients
  - No negative inotropic effects hence it is safe to use in patients with chest pain or hypotension
  - Safe in pregnancy
  - Adenosine has a half life of 10secs and effect up to 20 sec
  - A second dose can be given if no effect is present in 2 mins
  - Dose: 6mg or 0.1mg/kg IV rapid IV bolus
  - No proven effect if more than 2 doses or > 20mg in total
  - Effect: It may reveal atrial flutter waves
  - Note that at least 50% of patients experience chest pain and flushing. These effects are transient
- Verapamil
  - 2.5-5.0mg IV over 2mins, 3mins in older patients
  - It may be repeated, 5-10mg at 15mins up to 20 mg
  - Effect: Verapamil can induce hypotension. This can be treated with calcium chloride 500-1000mg IV every 10 mins
  - Contraindications: hypotension, presence of heart failure, chronic pulmonary lung disease, Beta-blocker use
- Beta-blockers can be used instead of verapamil although the contraindications are similar.
  - Metoprolol is commonly used
  - Dose: 5mg IV every 5 mins up to 15 mins
  - Contraindications: VT, hypotension, Severe cardiac failure, history of asthma or chronic pulmonary lung disease, Calcium channel blocker use

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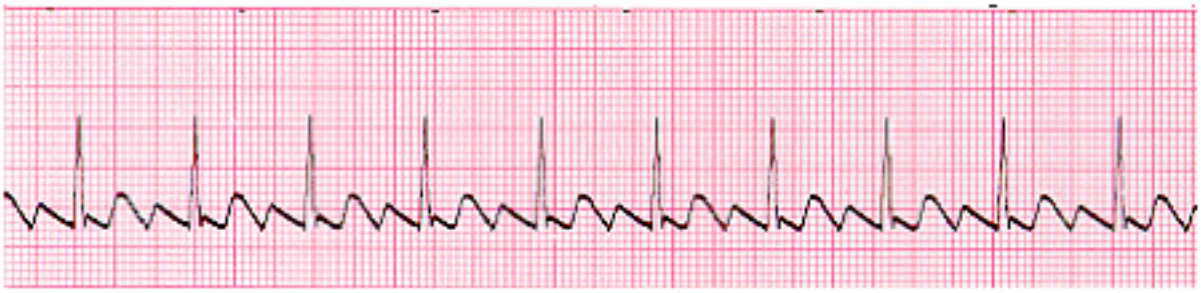
Narrow complex tachycardia with irregular rate

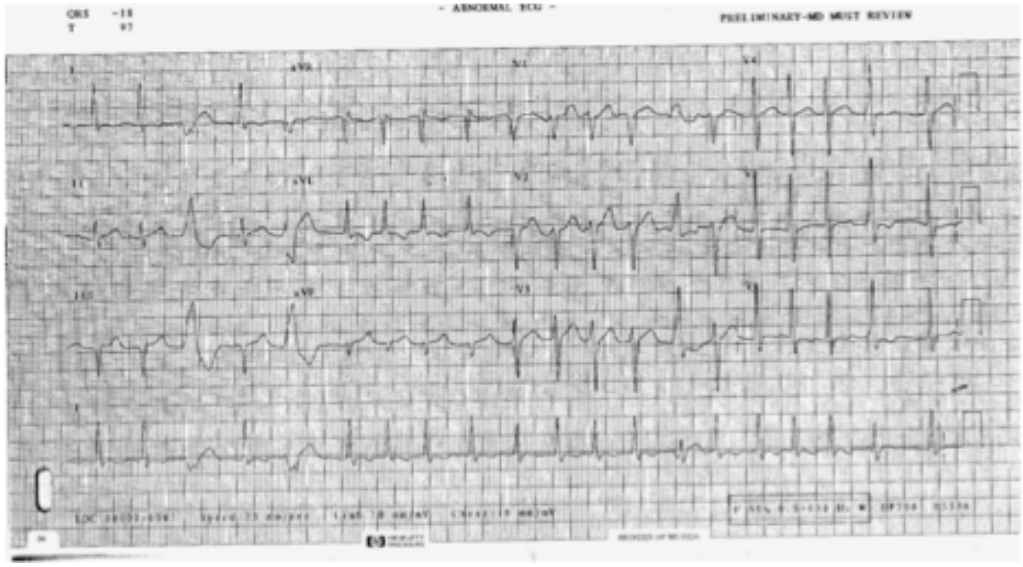
Atrial fibrillation is the commonest in this category. Atrial flutter with variable block may present as a NCT with irregular rate. The distinguishing features are as follows:

- In AF, the P wave is absent. In atrial flutter, look at lead II for the negatively orientated saw tooth wave
- In AF, the atrial frequency is 400-600/min. The ventricular response is between 75-180/min
- In Atrial flutter, the atrial frequency is 250-350/min. The ventricular response is a ratio of 300 depending on the block. Hence 2:1 block has a ventricular rate of 150/min. A 3: 1 block has a ventricular response of 100/min
- Adenosine will temporarily slow both of these rhythms. In Atrial flutter, the saw-tooth wave may become more evident in lead II



Classification of Atrial Fibrillation: AF is categorized into groups that have implications for management:

1. New onset
2. Paroxysmal: short bursts of sudden onset. Spontaneous reversion to sinus rhythm within 7 days and usually within 24 hours
3. Persistent: lasts for more than 7 days, requires either drugs or electrical cardioversion
4. Permanent: due to underlying cause especially structural heart disease and this group is refractory to cardioversion



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Management of AF

Unstable patient:

- Synchronised cardioversion with PSA
    - Biphasic waveform, use 200J
    - If AF has been present for > 48 hours, there is a risk of thromboembolism if cardioversion occurs without anticoagulation

Stable patient: The considerations of treatment are:

- Rate control
- Rhythm control by cardioversion (drugs or electricity)
- Prevent thromboembolic event

Rate control:

- Magnesium is an inexpensive agent which is nearly as effective as amiodarone, with fewer side effects. Dose: 2g bolus over 10-20minutes.
- If patient has no cardiac failure and no bypass tract, the first line drug is a B-Blocker or non-dihydropyridine calcium channel blocker such as verapamil. Amiodarone can be used as a second line drug.
- If there is cardiac failure, digoxin or amiodarone is recommended. Digoxin has a slow onset with a mean time of 11 hours prior to achieving rate control.

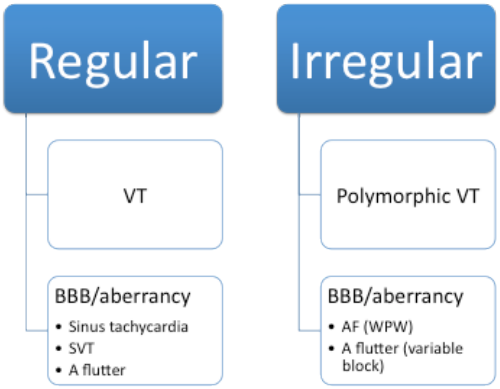
Special circumstance – suggest early cardiology input

- In pregnant patients with AF who are unstable, electrical cardioversion is used. If stable, B Blocker or digoxin may be used.
- Amiodarone is used in patients with acute myocardial ischaemia with LV dysfunction.
- Do not use digoxin or verapamil in patients with a known pre-excitation syndrome. Consult a cardiologist, who might advise one of the following:
    - Metoprolol 5mg IV every 5 mins up to 15 mgs (Contraindications: Hypotension, severe cardiac failure, asthma or COPD)
    - Amiodarone 5mg/kg IV over 30mins followed by 1.2g over 24 hours
    - Digoxin 0.5mg IV if not on digoxin normally

Rhythm Control

- Primarily with Synchronised cardioversion with sedation and analgesia.
- Some drugs are effective in controlling rhythm including sotalol and flecanide but should be used with caution.
- Discussion with a senior is advised

Wide Complex Tachycardia



WCTs are approached in the same way as NCTs.
Regular - The arrhythmias in this group can present in patients who seem stable.

Aberrancy:

Supraventricular tachycardias with abnormal ventricular conduction present as WCTs. The deviation from the normal conduction is also called aberrant conduction. It can be due to a bundle branch block or accessory pathway

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between the atria and ventricles.

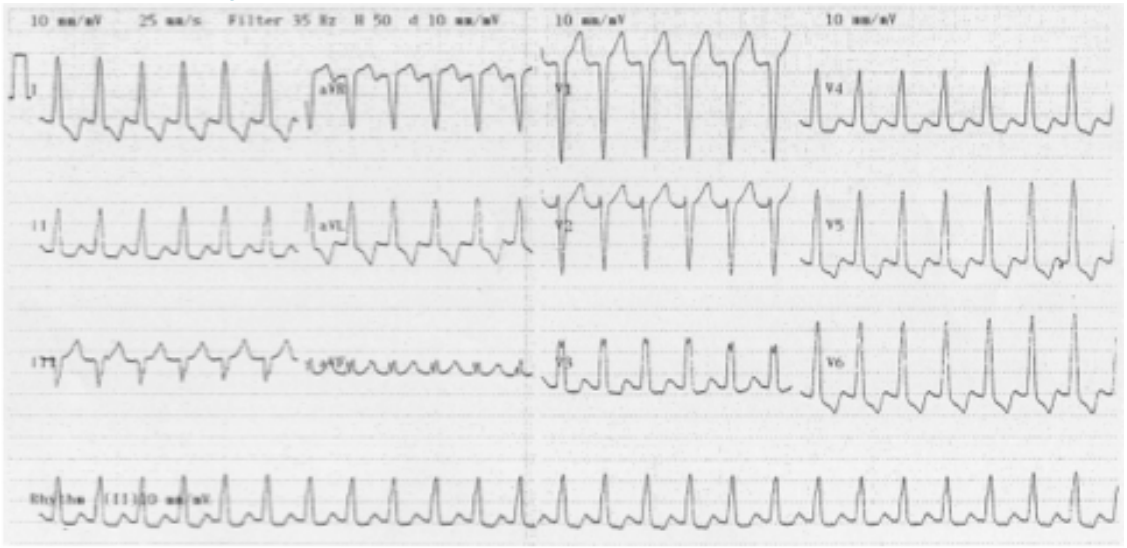
Atrial flutter

Atrial flutter is one of the supraventricular tachycardias. A rate of 150/min would suggest atrial flutter with 2:1 block and 300/min would be consistent with a 1:1 block.

Important note:

If there is doubt it is advisable to consider the regular WCT to be VT. This is important when choosing the drugs to be used in treatment. There are many 12 lead ECG features to support VT rather than SVT with aberrant conduction. However, in the ED, this approach is generally less safe than adopting the initial perspective of assuming VT until there is certainty of aberrancy.

SVT with Aberrancy



This is an ECG of SVT with aberrant conduction. Note that in patients with normal QRS complexes, when SVT occurs, the SVT may be too fast for the ventricular conducting system's ability to depolarize and repolarize. Hence it causes a rate-related bundle branch block, producing a WCT. There are many ECG criteria to distinguish this from the other WCT – VT. As mentioned before it is better to approach the patient as though this is VT, unless there are previous ECGs showing aberrant conduction. The following is a helpful guide as the ECG features are easier to find (Atlas of Emergency Medicine, accessed through http://proxy14.use.hcn.com.au/content.aspx?aid=6008102)

ECG Features consistent with SVT with aberrancy:

- Tachycardia (usually >120 bpm) with a wide QRS complex
- No "capture" or "fusion" beats or AV dissociation as seen with ventricular tachycardia
- QRS morphology consistent with one of the bundle branch block patterns
- Ventricular rates of 140 to 160 should prompt consideration of atrial flutter with a 2:1 block.

ECG Features consistent with VT

- Tachycardia (usually >120 bpm) with a wide QRS complex
- Evidence of AV dissociation is present such as P waves appearing periodically in the T wave or baseline
- "Capture" beats occur if atrial depolarization occurs prior to the intrinsic firing of the ventricle
- "Fusion" beats occur if atrial depolarization passes through the AV node at the same time as the intrinsic ventricle depolarization, producing a QRS that appears to be different or narrower than the other VT QRS

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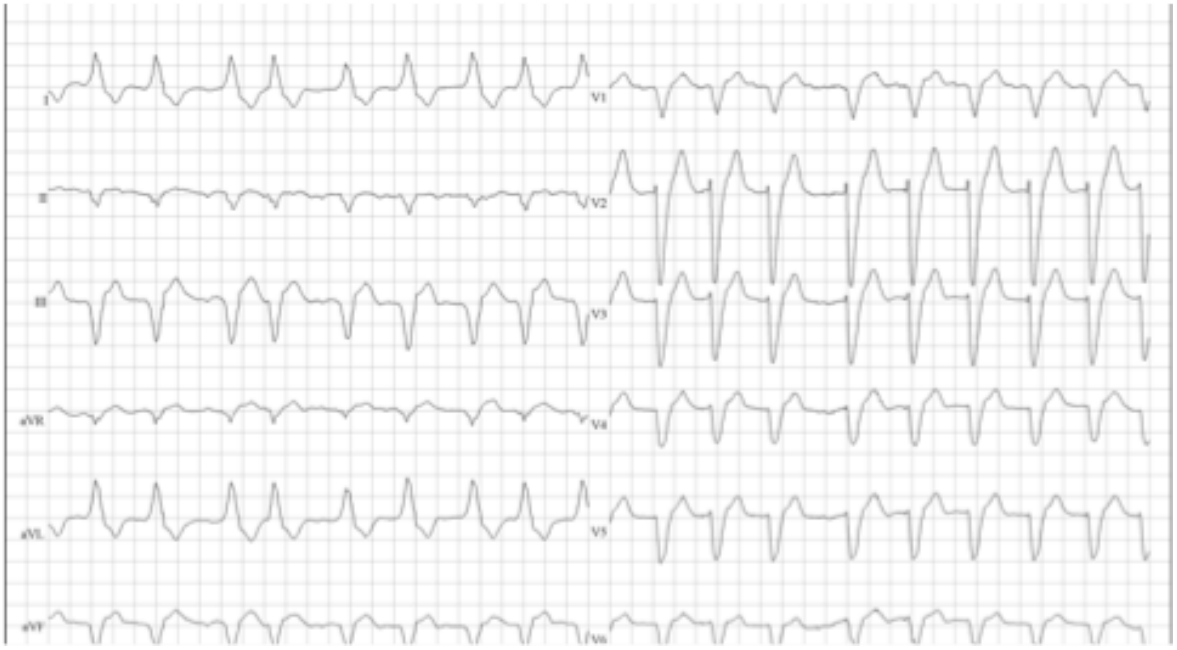
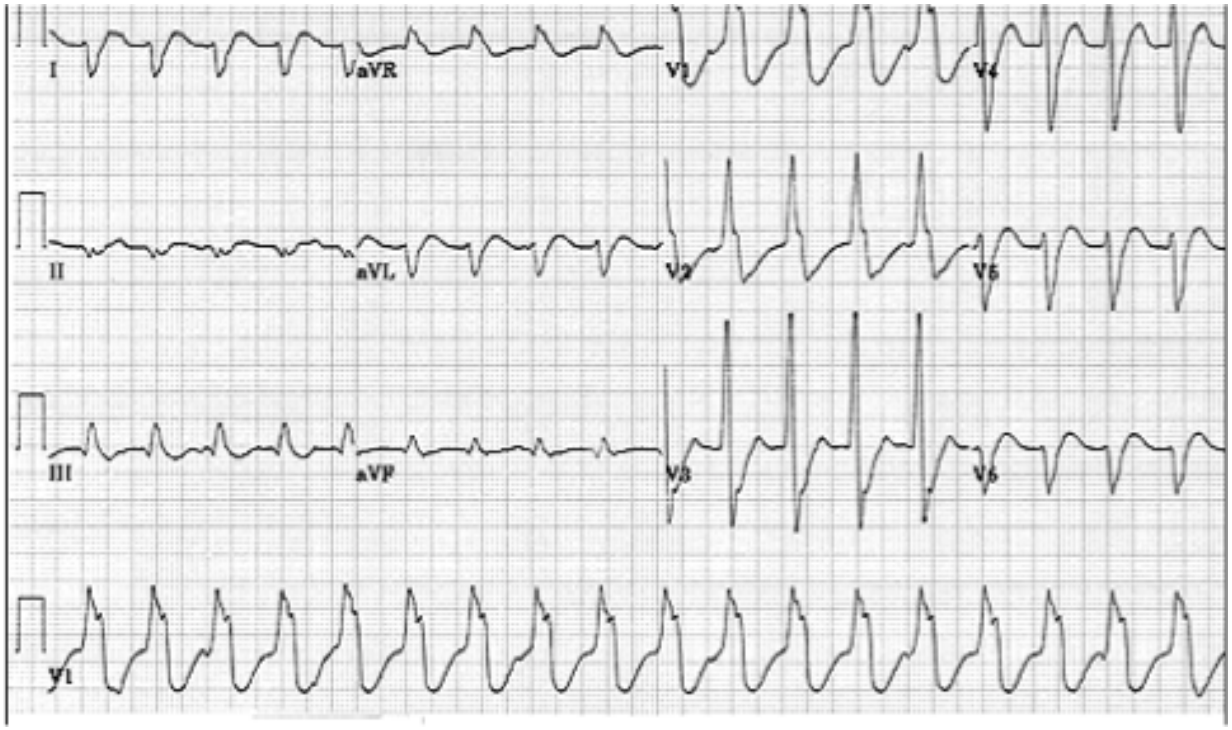
complexes

- QRS axis, between 180 to 270 degrees
- Precordial concordance which refers to the precordial QRS complexes being all up-going or all down-going
- V1 has a completely up-going QRS complex
- V4-6 have a down-going QRS complex



This is a 12 lead ECG of AF with aberrant conduction. The left bundle branch block was evident in a pre-existing ECG in sinus rhythm in this patient. This arrhythmia is unlikely to deteriorate into ventricular fibrillation.
An irregularly irregular or chaotic R-R interval, even if subtle, strongly suggests atrial fibrillation or flutter as the culprit SVT. In contrast, the R-R interval of ventricular tachycardia is almost never chaotic.



Ventricular Tachycardia

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The 12 lead ECG features are:

- QRS > 0.12 sec
- Regular rhythm, minor beat to beat variation may occur

Causes of VT

- Severe heart disease most commonly IHD
- Hypertrophic obstructive cardiomyopathy
- Mitral valve prolapse
- Drug toxicity
- Use of sympathomimetic drugs

Note that the risk of developing VT is increased by hypoxia, alkalosis, and electrolyte abnormalities.

Ventricular tachycardia is often thought to occur in patients who are unconscious. This is not always the case and hence there is the clinical term of "conscious VT", these usually have a weak pulse. However, this may not last long before haemodynamic compromise occurs and early action to treat the VT is important. It can deteriorate into pulseless VT or VF.

1st line:

-amiodarone 150mg over 10mins repeat X1 in 10 mins. This can be followed by an infusion of 900mg over 24 hours.

-Note: amiodarone is the antiarrhythmic of choice in setting of AMI, LV dysfunction or unknown cardiac function

2nd line

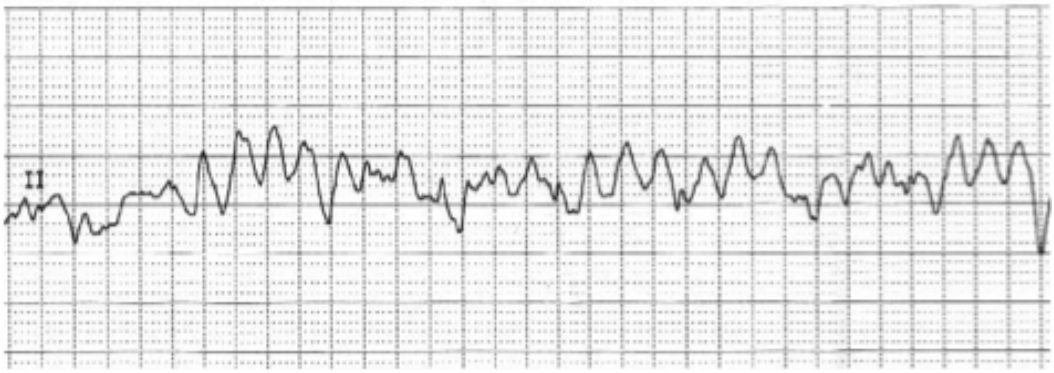
-Lignocaine: 1-1.5mg/kg IV every 5 mins up to 300mg/h

-Note: it may be prudent to go to cardioversion if patient is refractory to amiodarone. This should be discussed with a cardiologist prior to the introduction of the 2nd line drug.

Unstable tachyarrhythmias

The following are tachyarrhythmias that are likely to lead to cardiac arrest. Hence there is an emergent need to treat. In general, 80% of cardiac arrests are due to tachyarrhythmias originating from the ventricles. Hence, the following are sometimes called the Malignant Ventricular Arrhythmias:

- Ventricular Tachycardia: when > 3 ventricular ectopic beats occur at a rate of > 100/min
- Ventricular Fibrillation
- Poly morphic VT - Torsades de Pointes



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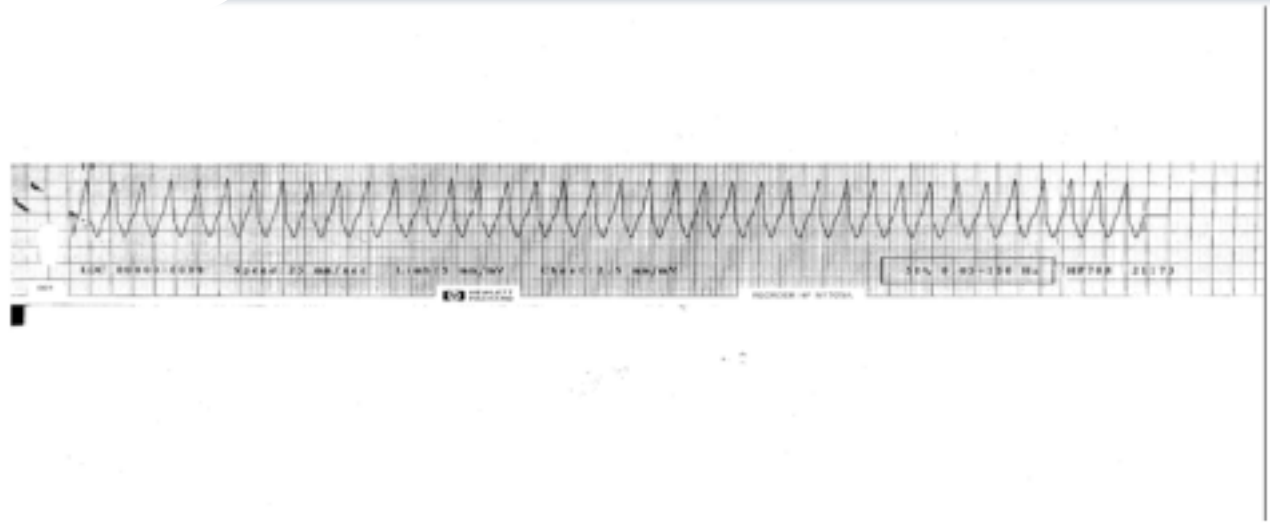

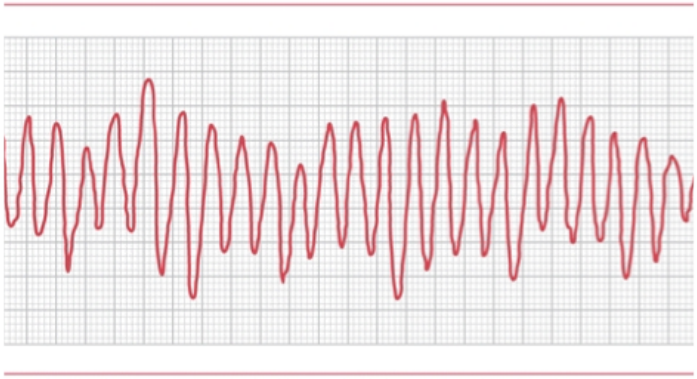

Torsades de Pointes (TdP)

This is a polymorphic ventricular tachycardia, which is associated with prolonged QT. The QRS complexes have different amplitudes and widths within a single lead. TdP may occur in short runs of 5-15sec at a rate of 200-240/min.

Torsades de pointes is triggered by a prolonged QT interval due to the following causes:

- Severe myocardial disease
- Congenital long QT syndrome
- Drugs: (www.torsades.org; www.azcert.org) There are many drugs in common use that will increase the QT interval and hence predispose to this arrhythmia
- More at risk if there is renal insufficiency, electrolyte disturbances, when more than one drug in this group is given concurrently

The likely drugs are categorized into likelihood of causing TdP. The following 2 categories are highlighted here:

- Prolongation of the QT interval AND cause TdP when used as directed
    - Amiodarone, flecainide, sotalol
    - Chlorpromazine, thioridazine
    - Droperidol, haloperidol
    - Clarithromycin, erythromycin, moxifloxacin
    - Methadone
- Prolongation of the QT interval but MAY cause TdP when used as directed
    - Roxithromycin
    - Clozapine, escitalopram, lithium, venlafaxine
    - Ondansetron

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Treatment of Torsades de Pointe

TdP, have a high risk of degrading to pulseless arrest and must be treated immediately

- Unstable patient: treat as for VF: defibrillate
- "Stable" patient: there is no consensus (therapeutic guidelines). Here are a few ground rules:
- Never give amiodarone, flecainide, or sotalol in this situation
- Correct electrolyte imbalance. Keep serum K+ in the 5.0-5.5 mmol/L range
- Stop the offending drug
- If there is an underlying bradycardia, give Atropine 0.5mg IV and repeat as required
- Transvenous pacing is probably the safest but not readily available
- Magnesium sulphate: 1-2grams IV over 60-90sec followed by infusion of 1-2gm/hr. Not likely to be helpful if normal QT
- Isoprenaline: 20mg IV repeat if required. Infusion at 1-4mcg/min. NB arrhythmogenic
- Lignocaine 75-100mg/IV over 1-2 mins followed by an infusion 4mg/min for 1 hour then 1-3mg/min

The Unstable Patient

Haemodynamic instability refers to a low cardiac output when the patient is in a particular rhythm. Cardiac arrest is when there is no cardiac output. The following features suggest the cardiac output is not adequate to support end-organ function. The symptoms and signs are due to inadequate cerebral and cardiac perfusion. These are readily discernable at the bedside. Hence:

- Altered level of consciousness: confusion, reduced responsiveness to verbal stimuli
- Chest pain: patient complains of persistent chest discomfort suggestive of ischaemic pain
- Hypotension: Systolic BP< 80mmHg
- Heart rate > 150/min. This is a Red Flag number. A rate above 150/min is likely to deteriorate if unheeded. 80% of rhythms that lead to cardiac arrest are tachyarrhythmias. The implication is that there is an urgency to treat! If the patient is elderly this red flag number can be reduced to 120/min.

ELECTRICAL CARDIOVERSION

Electrical cardioversion is the synchronised electrical therapy to reset the normal activity of the heart

Equipment:

- Defibrillator required, most EDs have biphasic defibrillators.
- Turn on the device.
- Positioning of the paddles/pads: antero-apical position is one paddle to the right of the upper half of the sternum below the clavicle and one to the left of the left nipple in the axilla. This is the preferred position for a supine patient
- Paddles: paddles or pads have a conducting gel or gel pad. These are firmly applied to the chest wall. Good contact with the skin ensures shock efficiency (watch out for hair, patches, jewelry, pacemakers, etc). Outcomes are better with larger paddles/electrodes i.e. 12cm better than 8cm

Drugs for sedation:

- For cardiac arrest, defibrillation is part of the immediate resuscitation process and sedation is not needed.
- For elective cardioversion, anaesthesia and procedural monitoring is required
- Equipment for airway management is essential. This includes IV access. Note that patient explanation and informed consent are both required.
- Sedating agents and analgesia are given intravenously and together it is called Procedural Sedation and Analgesia (PSA). There is a choice of drugs. The common combination is midazolam and fentanyl. Though there are many other choices which should be used by skilled practitioners.
- Continuous cardiac monitoring are maintained throughout the procedure and afterwards

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Midazolam:

Short acting benzodiazepine for minimal sedation. Maximum dose is 0.1mg/kg IV

- Peak effect within 2-3 mins. Duration of action is up to 1 hours
- Retrograde amnesia is 20-30 mins
- If given with an opiate, it can increase the risk of respiratory depression
- Side Effects: hypotension if patient is hypovolaemic

Fentanyl:

Short acting opiod. Maximum dose is 1-3microgram/kg given over 3-5mins. (see below)

- Onset is < 1min and peaks in 2-3mins
- Duration is 30mins to 1 hours
- Used alone as single minimal sedation.
- In combination with midazolam for moderate to deep PSA
- SE: rigid chest syndrome is rare characterised by respiratory muscle spasms leading to respiratory depression or apnoea. Usually if doses are in the >5mcg/kg are given in fast IV bolus. Not reversible with antagonists, intubation and rapid sequence induction is required. Usually happens when the line is flushed quickly.

Cautions

- With a low cardiac output state the drugs will take longer to work. Give each drug at least 5 minutes to take effect
- Do not give maximum dose as a bolus. Start off small and work your way up to the maximum dose, if needed

Personnel

Prepare for deterioration, with staff readily available for intubation and cardiac arrest protocols. Senior staff should be aware and present for this high risk procedure.

Procedure:

- Check that the defibrillator is in synchronised mode
- Select energy level: monophasic defibrillators:
  - 50J for PSVT and atrial flutter
  - 100J for VT and atrial fibrillation
- Energy level for biphasic:
  - 200J for cardiac arrest
  - 100J for VT and AF
  - 50J for PSVT and atrial flutter.
- Commence PSA – slowly
- Apply the paddles and charge
- Check that no personnel is in contact with the patient or the trolley and call out for everyone to stand clear
- Discharge the shock
- Review the outcome with a full reassessment of the patient.

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In Summary

Through the presentation, the cases and the scenario we have:

- Discussed the importance of a structured approach to assessment and management of patients with tachyarrhythmias
- Outlined the need to assess the ECG for QRS width and regularity
- Reviewed the indications and appropriate use of electrical and chemical cardioversion
- Highlighted the use of care when administering sedative medications to this patient population
- Looked at how we work in multidisciplinary teams. It is important to make our lives, and the treatment of our patients easier. To do this we need to be aware of non-technical skills and how we can use them to treat patients more efficiently.
- Remember to treat WCT as VT if there is any doubt.

References and Further Reading

- eTG complete March 2012. Accessed through www.use.hcn.com.au
- Australian Resuscitation Guidelines. Accessed through www.resus.org.au
- ECGpedia. Accessed through www.en.ecgpedia.org
- Tintinalli's Emergency Medicine. Accessed through http://proxy14.use.hcn.com.au/
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- www.torsades.org

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