Topic Overview: Cardiac Module
Sub-Module: C5- Structured Approach to Arrhythmias and Bradyarrhythmias

Last updated July 30 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 bradyarrhythmias
4. Identify pre-malignant bradyarrhythmias (indications for cardiac pacing)
5. Rehearse transcutaneous cardiac pacing

A: STRUCTURED APPROACH TO THE PATIENT WITH AN ARRHYTHMIA

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?

Applying the "Stable or Unstable" rule?

Frequently, you will be asked by senior clinicians 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:

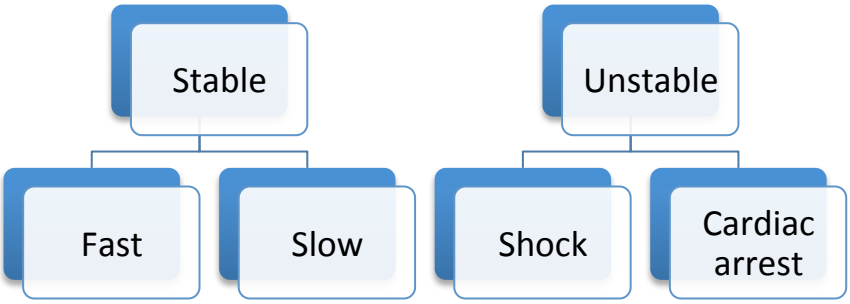
1. STABLE –CO adequate to perfuse end organs

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

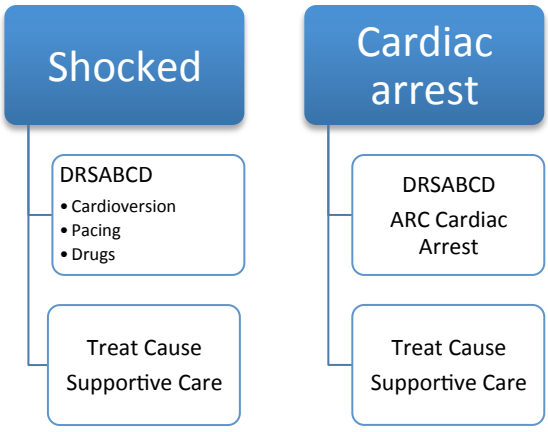
3. CARDIAC ARREST – No CO



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:



B: BRADYARRHYTHMIAS

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

Stable patients

If the patient is haemodynamically stable, there is no acute treatment required in the emergency department irrespective of the arrhythmia although the patient may still require admission or cardiology intervention. *However*, two steps need to be followed.

1. Firstly, determine the underlying *cause* of the slow heart rate and correct if able to be optimised, such as electrolytes, drug side effects and cardiac conditions.
2. Secondly, appraise the likelihood the arrhythmia will evolve into a malignant arrhythmia likely to cause haemodynamic instability .

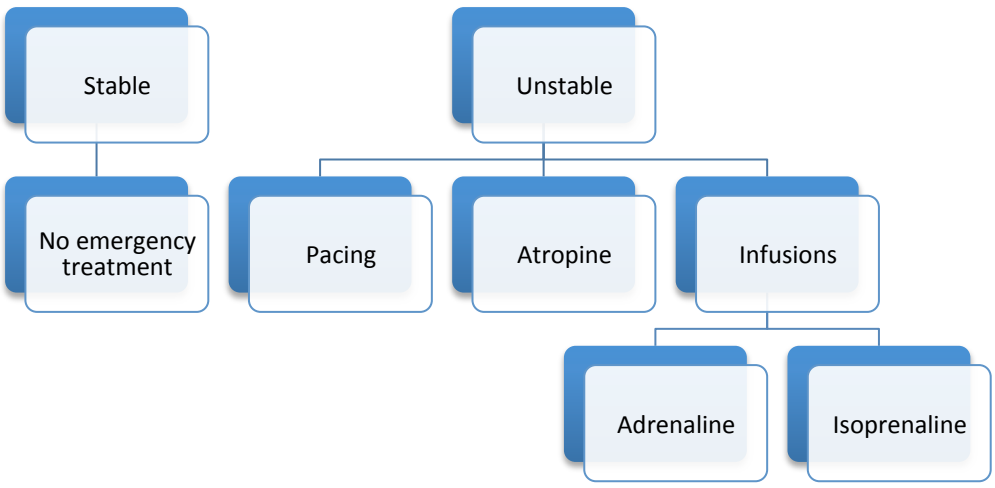
Unstable patients

Treatment options fall into three categories:

1. Pharmacological
2. Non- Pharmacological (Cardiac Pacing) and/or
3. Supportive (e.g. correction of causes and contributors)

Specific treatment is determined by the underlying rhythm as described in the following section. As general statements:

1. Atropine can be very useful in treating some bradycardias, but it should be noted that its effect may be only temporary. Repeated doses may be needed or a different drug may need to be used to maintain the effect.
2. Infusions and/or pacing will have a more lasting effect, until definitive management is available.



Heart Block

An important group of bradyarhythmias is Heart Block however not heart bocks manifests as bradycardia.

First Degree Heart Block

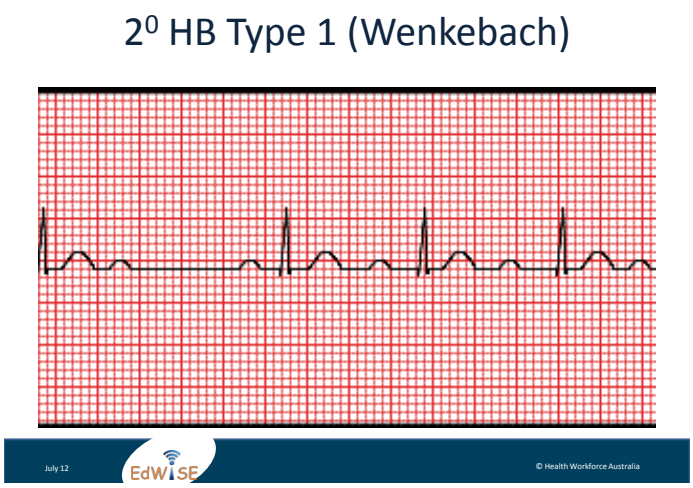
The identifying ECG feature is a PR interval greater than 0.20 seconds. It occurs as a result of degeneration of the conduction system, usually due to advancing age. The site of the block is above the atrioventricular node. It does not generally cause instability.

2nd Degree HB Type 1 (Wenkebach)

The ECG feature is a gradual increase in the PR (or PQ interval if no R wave) interval until a P wave is not followed by a QRS complex.

Essentially, the conduction of the sinus P wave to the AVN is increasingly slow until one P wave is not conducted to the ventricle – hence not temporally linked to a QRS complex. When present, the ECG rhythm strip (Lead II) reveals a gradually increasing PR interval culminating in a dropped QRS complex. The frequency of dropped QRS complexes is described as a cluster ratio of p waves followed by QRS complexes. For instance, a 5:4 cluster means that for every 5 P waves, only 4 are followed by a QRS complex. Note also, that the PR/PQ interval following the dropped beat is the shortest. Exercise tends to reduce the frequency of the block through the AV node.

Type 1 second degree heart block is generally well tolerated although suggests an underlying cardiac cause.

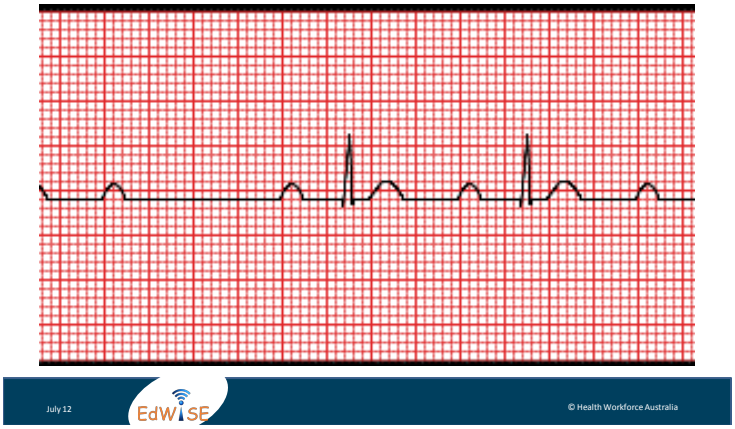


2nd Degree HB Type 2 (Mobitz)

Type 2 (or , Mobitz) second degree heart block heart is a more sinister arrhythmia as it is more likely to progress to Type 3 or complete heart block with subsequent cardiovascular consequence.

The ECG feature is a random occurrence of non-conduction of the P wave. Hence the QRS complexes do not occur in clusters with irregular dropping of a QRS complex. There is no variation in the PR/PQ interval in the beats before the non-conducted P wave. The location of the conduction problem is in the His bundle or the Purkinje fibres.

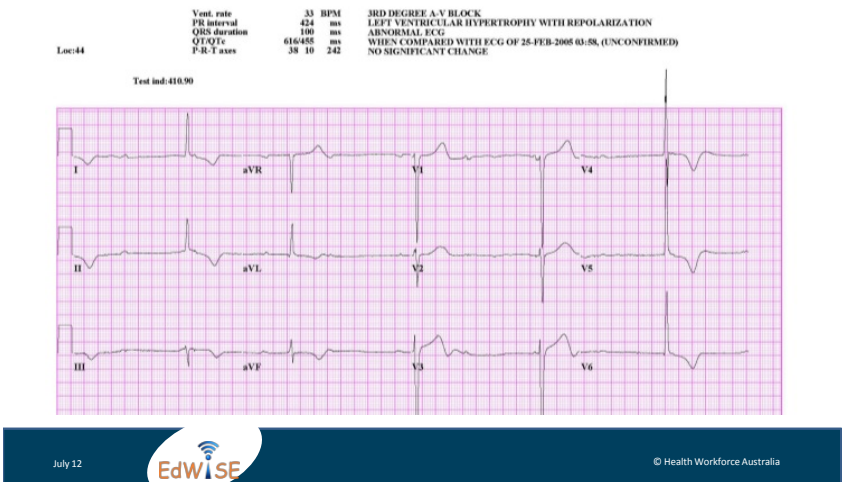
2^0 HB Type II (Mobitz)



3rd Degree HB (complete heart block)

In this arrhythmia the P waves and QRS complexes are present but uncoupled, that is complete atrio-ventricular dissociation. The QRS complexes reflect an "escape" rhythm generated from the nodal area or within the ventricles. Hence the terminology used is a nodal escape rhythm or an idioventricular rhythm. Both are slow reflecting the ntive rate of the ventricular pacing tissue, approximately 30 bpm. This is a dangerous arrhythmia and if the cerebral perfusion is inadequate, it leads to syncope or presyncope. Stokes Adams attacks refer to the loss of consciousness secondary to complete heart block. CHB requires urgent treatment regardless of apparent haemodynamic stability.

Complete Heart Block



Summary – Key points

- Use atructured clinical approach to arrhythmias – both fast and slow
- Apply the "Stable or unstable" rule - this the most important question at the bedside

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

Acknowledgements

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