ACUTE CARDIAC CARE
CAIRNS BASE HOSPITAL

RHYTHM INTERPRETATION
WORKBOOK
1.0 Introduction

Approximately ninety percent (90%) of coronary patients will experience some sort of arrhythmia, particularly in the first 24 hours. (Nat Heart). Prompt and effective management is therefore vital especially if haemodynamic compromise or ischaemic myocardium is caused. Medical/surgical nurses caring for these patients therefore need to be able to recognize and appropriately respond to potentially lethal arrhythmias that their patients experience. This course will provide an understanding of the principles of monitoring, systematic approach to rhythm interpretation and an introduction to the strategies that can be used to manage arrhythmias.

1.1 Learning Outcomes

- Explain the correct procedure for routine continuous electrocardiogram (ECG) monitoring.
- Demonstrate analysis of an ECG rhythm strip using a systematic approach.
- Demonstrate the ability to recognize sinus rhythm.
- Interpret a 12 Lead ECG and recognize the significance of the changes identified
- Implement appropriate nursing management strategies for a patient with an arrhythmia

1.2 Foundation knowledge

It is assumed that participants undertaking this course will already have a basic understanding of anatomy and physiology in relation to cardiology. At this stage it may be useful to access a current and relevant anatomy and physiology textbook and review the anatomy and physiology of the heart and cardiovascular system.

1.3 Resources to complete the course

You may find the following text useful. It is assumed that you have prior knowledge of the anatomy and physiology of the heart. If you need to review these you will find these texts relevant:

- Wesly,K., Huszar’s Basic Dysrhythmias and Acute Coronary Syndromes: Interpretation and Management. 4th edition 2011, St Louis, Elsevier Morsby Jems;
2.0 Monitoring a rhythm

An ECG shows the precise sequence of electrical events occurring in the cardiac cells. Electrodes placed on the skin of the chest and extremities sense the electrical activity and transmit them to the monitor. [1, 2]

2.1 Continuous monitoring lead placement

Each ECG recording electrode provides the view of the electrical activity that it "sees" from its particular position on the body surface. A monitoring lead or ECG lead provides a particular view of the heart's electrical activity between two points or poles. When the electrical activity is not detected, a straight line is recorded. This line is called the baseline or isoelectric line. A waveform (deflection) is movement away from the baseline in a positive (upward) or negative (downward) direction. Each lead consists of a positive pole and a negative pole. The direction in which the electrical current flows will determine how the waveforms appear on the ECG tracing. [1]

It will depend on how many leads you have on your monitoring system as to how many views you can continuously monitor. Before reviewing the continuous ECG monitoring systems you use, it is important to review the standard possible views of the heart. The leads we need to monitor will depend on which view of the heart we want.

- 3-lead monitoring systems uses 3 electrodes to display leads I, II and III
- 5-lead monitoring systems uses 5 electrodes to display leads I, II, III, aVR, aVL, aVF and a V lead. The C1 lead indicates the position of the chest electrode for monitoring lead V1 and C6 indicates the position of the chest lead for monitoring lead V6. [3]
Continuous ECG monitoring is standard for patients at high risk of arrhythmias.[2] Two (2) techniques for continuous monitoring are hardwire monitoring more commonly found in critical care units and telemetry (portable) monitoring used in step down or acute care settings. [2] Telemetry allows patients to be more active. [1]

It is also important to note that one can monitor additional alarms without viewing the lead on the monitor e.g. ST segment analysis can occur on all leads available even though one may only be able to monitor 2 or 3 ECG tracings.

**Activity 1 – short answers**
Identify the continuous monitoring system used in your unit and identify the following:

a) What type of lead configuration does your unit use for continuous monitoring (i.e. 3, 5, 6, 12 or other)?

b) Identify how the leads are positioned correctly for that system.

c) What choice of leads (i.e. views of the heart) do you have available to monitor?

d) How many ECG leads can you monitor at once?

e) What are the best leads for monitoring the PQRST?

f) Discuss appropriate alarm parameters used in your Unit.

g) Identify how to print a rhythm strip

### 2.2 Cardiac Electrophysiology and the Cardiac Cycle
In order to interpret cardiac rhythms, it is important to understand the electrophysiology of the heart and its relationship to the mechanical activities.[1]

To distinguish between the electrical and mechanical events that occur in the heart.
- The surface ECG is representational of the electrical activity in the heart.
- Haemodynamic monitoring provides data on the mechanical activity of the heart.[1]
2.2.1 Action Potential

As you will have identified from your reading the action potential is an electrical signal that proliferates along the membrane of a muscle cell. The action potential mirrors the electrical activity in a cell in the same way that the ECG reflects the electrical activity of the entire heart.

The excitation stages (depolarization), recovery (repolarisation) and rest (excitability) are known as the action potential. The cells action potential is due to changes of electrolyte concentration across the cell membrane. The important electrolytes that play a key role in maintaining membrane stability are K+, Na+ and Ca++. Problems with impulse formation and conduction can lead to arrhythmias. Action potentials causing arrhythmias occur with electrolyte imbalance, hypoxia, ischemia, acidosis, etc. Conduction disturbance of impulses may also result in arrhythmias. Additionally, an understanding of cellular physiology is essential for gaining an understanding of the pharmacological management of arrhythmias.

The diagram below illustrates the phases of the action potential.

Figure 2 – Phases of action potential
To understand fully the response of cardiac cells to electrical stimulation, one must first understand the electrical and mechanical properties of cardiac tissue. The properties of cardiac tissues are *excitability, conductivity and automaticity*. Knowledge of these properties and the underlying physiology of cardiac cells in relation to normal conduction and contraction is vital to understanding both normal and abnormal rhythms.

**Activity 2 – short answers**

Review 3 patients on cardiac monitoring in your unit and answer the following questions:

(a) Identify the reason each patient is on cardiac monitoring.

(b) What condition/s does your patient have that predisposes them the development of cardiac arrhythmias?

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<thead>
<tr>
<th>Patient</th>
<th>Rationale</th>
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<tr>
<td>One</td>
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<tr>
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<td>Three</td>
<td>a)</td>
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<td>b)</td>
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</table>
2.3 Electrocardiogram

The ECG is a graphic representation of the electrical activity of the heart. The ECG complex consists of:

- P wave
- QRS complex
- T wave
- U wave

To understand the graphic representation, we need to look at the ECG paper that it is printed on, the baseline, artifact and waveform produced.

2.3.1 ECG Paper

The grid on ECG paper is standardised so that there are thin lines every 1 millimeters (mm) and a thick line every 5 mm. The ECG paper, on the horizontal axis refers to time. When the paper is running through the printer at 25 mm/sec, each small square (thin lines) = 0.04 seconds and the large squares (thick lines) = 0.20 seconds. [5,6]

The vertical axis of the paper refers to voltage or amplitude of the waveform. The amplitude is measured in millimeters (mm), therefore each small vertical box is equal to 1 mm. The voltage is measured in millivolts (mV). The electrocardiographic machine sensitivity must be calibrated so that 1mv of electrical signal will produce a deflection measuring exactly 10mm tall. Located to the right side of many ECGs is calibration box for easy reference, standard calibration will produce a box 2 large squares and exactly 1 cm in height. [8]


Activity 3 – short answers

1. When might you need to alter the voltage calibration (size of waveforms) to promote easier interpretation of the rhythm or ECG?

2. How should you document changes to the calibration on the 12 ECG recording?
3. When may you be required to alter the sweep speed calibration when monitoring?

2.3.2 Waveform, Segments and Intervals

The ECG complex is made up waveforms, segments and intervals.

A waveform is a deflection from the baseline that represents a cardiac event. The deflection may be in either a positive (upward) or negative (downward) direction. The waveforms represent electrical activity (depolarisation) or recovery (repolarisation) of the cells of a chamber of the heart. In the normally functioning heart mechanical contraction follows the electrical contraction. Therefore, the P wave will precede atrial systole and the QRS will precede ventricular systole.

The segments are the lines between waveforms and are named by the waveforms that precede and follow it. For example PR segment is the part of the baseline between the end of the P wave and the

An interval (e.g. PR interval) is the distance, measured in time, occurring between two cardiac events and includes a waveform and a segment.
The intervals include a waveform and a segment, for example PR interval includes the P wave and the PR segment. P Wave

The P wave is the first deflection in the cardiac cycle and is caused by depolarisation of the atria. Normal P waves are small, rounded and positive in lead II, no taller than 2.5 mm (2 ½ small boxes) or wider than 0.11 seconds (2 ½ small boxes). There should be one P wave preceding each QRS complex. A P wave of normal size, shape and direction indicates that the electrical impulse originated in the SA node and that normal depolarisation of the atria has occurred. If the P wave is abnormal, it indicates that the sinus impulse travelled through altered or damaged atrial tissue, or that the impulse responsible for the P wave originated in a site outside the SA node.

QRS complex

The Q, R and S waves represent ventricular depolarisation. These are grouped together and termed the QRS complex. The QRS complex represents ventricular depolarisation. The QRS complex is normally narrow and should be less than 0.12 seconds (less than 3 small squares wide).
- The first downward deflection of the QRS complex is termed a Q wave.
- The first upward deflection is termed an R wave.
- The downward deflection that follows the R wave is termed an S wave if it descends below the baseline.
- QRS complex can be predominantly positive (upright), predominantly negative (inverted) or equiphasic (equally positive and equally negative).


T Wave

The T wave is reflective of the later stage of ventricular repolarisation. Normal T waves are upright, rounded and slightly asymmetrical. They are positive in Lead II, with amplitude less than 5 mm (5 small squares high) and duration of 0.10-0.25 seconds or greater (approximately 6 small squares wide). Abnormal T waves may be:
- Tall and peaked, biphasic, flattened or inverted.

A Good Rule of Thumb

The normal T wave is always in the same direction as the QRS. The normal T wave is never more than two thirds the height of the R wave.
- Symmetrical T waves almost always indicate some pathology such as ischemia or electrolyte imbalance.

U Wave

The U wave is thought to represent the final stage of repolarisation of the ventricles. It is not always present. The onset of the U wave is identified as the first deviation on the baseline or the downward slope of the T wave. It is normally upright but may be inverted or flattened. Abnormalities resulting in tall U waves include:
- Hypokalaemia, cardiomyopathy and left ventricular hypertrophy.
2.3.3 Intervals & Segments

Evaluating specific intervals and segments provides additional information about electrical activity of the heart.

The **P-R interval** is the time required for the electrical impulse to leave the SA node and travel through the atria, AV node, bundle branches, and Purkinje network.\(^2\) It is measured from the beginning of the P wave as it leaves the baseline to the beginning of the QRS complex and usually lasts 0.12 - 0.2 second.\(^5\) Short PR intervals indicate the impulse was generated somewhere other than the SA node and is associated with junctional arrhythmias.\(^1\) Prolonged PR intervals may represent a conduction delay due to digoxin toxicity or conduction tissue disease.\(^1\)

**Q-T interval**

QT interval measures the time from ventricular depolarization and repolarisation and the length varies according to heart rate.\(^1,\,2\) It extends from the beginning of the QRS complex to the end of the T wave.\(^2\) It lasts for 0.36-0.44 second usually but can vary according to age, sex and heart rate.\(^1\)

Abnormality may indicate myocardial problems – increases the risk of life threatening arrhythmias such as torsades de points.\(^1,\,2\)

**T-P segment**

T-P segment is the portion of the ECG from the end of the T wave to the beginning of the P wave. It is usually isoelectric and particular useful when determining any deviation from the baseline of the ST segment.\(^6\)

**ST Segment**

Represents early ventricular repolarisation and lasts form the end of the QRS complex to the beginning of the T wave.\(^2\) It is this segment that should be isoelectric.\(^2\) Changes to ST segments may indicate myocardial damage becoming either depressed or elevated.\(^1\)

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**Golden Rule**

When ST elevation or depression has been detected in cardiac monitoring, a 12 Lead ECG must be performed to identify the specific changes and their possible cause. Other investigations may also be required.

Elevation or depression of the ST segment is considered significant when it is **greater than 1mm** (1 small square) and it occurs in **at least two leads** representative of the same area of the heart.

- ST segment depression of more than 0.5mm is suggestive of myocardial ischaemia.
- ST segment elevation of more than 1 mm is suggestive of myocardial injury.\(^6\)

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**Activity 5**

Answer the following questions to consolidate your learning.

1. The normal heart rate is initiated in the
   a) SA node
   b) AV node
   c) Bundle of his
   d) Purkinje fibres.

2. The structure that briefly delays the impulse (to let the ventricles fill) is the
   a) SA node
   b) AV node
   c) Bundle of his
   d) Purkinje fibres.
3. The QRS represents
   a) Atrial repolarisation
   b) Atrial depolarisation
   c) Ventricular depolarisation
   d) Ventricular repolarisation.

4. Which QRS duration is prolonged?
   a) 0.04 seconds
   b) 0.8 seconds
   c) 0.10 seconds
   d) 0.14 seconds.

5. The T wave represents
   a) Atrial repolarisation
   b) Atrial depolarisation
   c) Ventricular depolarisation
   d) Ventricular repolarisation.

**Activity 5**
CSAT: - Set up Telemetry

**2.5 Summary**

This section of the course has introduced the ways in which a rhythm may be monitored and the various components of the rhythm. In the following section you will apply this knowledge to interpreting a rhythm.

**3.0 Rhythm Interpretation**

An arrhythmia describes any cardiac rhythm other than sinus rhythm. The framework for rhythm analysis that is utilised should be reflective of the components of a normal cardiac rhythm (sinus rhythm) and enable identification of any abnormalities present.

An ECG is the graphic representation of the heart's electrical activity. An ECG does not relate the electrical activity to the clinical situation at hand. That is the role of the interpreter. Therefore, a systematic approach is required to interpretation is required.

**3.1 Steps to interpreting a rhythm**

The following steps should be used to interpret a rhythm:

a) assess the rate
b) assess rhythm regularity
c) identify and examine the P wave
d) assess intervals
e) evaluate overall appearance
f) interpretation
a) Assess the Rate

- Is the heart rate normal? (rate 60 - 100/minute)
- Is there a tachycardia? (rate > 100/minute)
- Is there a bradycardia? (rate < 60/minute)
- Is the atrial rate the same as the ventricular rate? [6]

• Method 1- **Number of RR intervals in 6 Second** [1, 3] - If every small square measures 0.04 seconds, then a large square (made up of 5 small squares) measures 0.2 seconds. 30 large squares measure 6 seconds (30 x 0.2 = 6 seconds). Mark 6 seconds. Count the number of R waves in 6 seconds and multiply by 10 to obtain the number in 60 seconds. In the above example there are 8 R waves. If there are 8 R waves in 6 seconds then there must be 80 R waves in 60 seconds.

• Method 2: **300 Rule** [3, 6] - For regular rhythms, the 300 rule may be used (as there are 300 large squares in 60 seconds). Count the number of large squares between 2 R waves and divide into 300. For example, method 2 example has 4 large squares between the R waves, hence 300 divided by 4 equals 75.
b) Assess Rhythm/ Regularity
Evaluate the regularity by measuring the distance between the R-R intervals across the entire 6 second strip. Is the rhythm:
- regular
- irregular
- irregularly irregular? [2]

c) Identify and Examine the P wave
- Is there a P wave?
- Is the P wave normal (upright)? It should be upright in Lead II.
- Does the P wave occur before, during or after the QRS complex?
  Conduction ratio of P waves to QRS complex 1:1
- If there is no P wave, is there any other atrial wave that may give rise to the conduction of the cardiac impulse? (e.g. flutter or fibrillatory waves.)
  P waves may be present or absent. [2, 3]

d) Assess Intervals (evaluate conduction)

**PR interval** (conduction) is measured from the beginning of the P wave to the beginning of the QRS.
- Is it normal? (i.e. 0.12 - 0.20 seconds)
- Is it constant?
- Does it vary? [1]

QRS complex (conduction) duration is measured from where the QRS complex leaves and returns to the baseline. [3]
- Is it normal? (< 0.12 seconds) < than 3 squares.
- Is it widened? (> 0.12 seconds) = or > than 3 squares
- Are all the complexes the same size and shape? [1]
**QT Interval** is measured from the beginning of the QRS complex to the end of the T wave.\(^1\)
- Is it normal? In general, a QT interval should be less than half the R-R interval.
- Is it abnormal? (> than half the R-R interval) or > than 0.45 seconds.\(^9\)

**e) Evaluate Overall Appearance of the rhythm**
- ST Segment is evaluated by identifying the isoelectric (baseline) line. Determine whether there is elevation or depression from the isoelectric line.\(^1\)
- T wave - Are the T waves inverted, flattened, upright or normal height?\(^1\)

**f) Interpretation**
Interpret the rhythm by identifying the site or origin (pacemaker site) of the rhythm (sinus), the mechanism (bradycardia), and the ventricular rate.\(^1\)

**Sinus Rhythm**
Sinus Rhythm reflects normal electrical conduction and heart rhythm. That is the rhythm starts in the SA node and the impulse is spread through the atria and to the AV node where there is slight delay before it travels through the bundle of His, right and left bundle branches and the Purkinje fibers into the ventricles.\(^5\)

**Diagnostic Criteria for Normal Sinus Rhythm**

<table>
<thead>
<tr>
<th>Rate</th>
<th>60-100 bpm</th>
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</thead>
<tbody>
<tr>
<td>Rhythm</td>
<td>Regular, P-P interval equal, R-R interval equal</td>
</tr>
<tr>
<td>P waves</td>
<td>Positive lead II, Present and before every QRS, 1Pwave: 1QRS complex conduction ratio</td>
</tr>
<tr>
<td>Intervals</td>
<td>PR: 0.12-0.2 sec, constant, QRS: &lt; 0.12 sec, QT: &lt; 0.46 sec</td>
</tr>
<tr>
<td>ST segment / T wave</td>
<td>Isoelectric, T wave upright</td>
</tr>
<tr>
<td>Site of Origin</td>
<td>Sinoatrial node.</td>
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</tbody>
</table>
Activity 5
Using the systematic approach provided, interpret the following rhythm

<table>
<thead>
<tr>
<th>Rate</th>
<th>Rhythm</th>
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<th>Intervals</th>
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<th>QRS:</th>
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<th>ST segment / T wave</th>
<th>ST:</th>
<th>T wave:</th>
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<th>Interpretation</th>
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Activity 6
Spend more time ensuring that you recognise sinus rhythm. Collect and attach two rhythm strips that reflects sinus and its variations

Rhythm Strip Placed Here

Rhythm Strip Placed Here
3.2 Summary

This unit has laid the foundations for rhythm interpretation by the application of a framework. To increase your familiarity with the framework, continue to practice interpreting rhythm strips. This knowledge will be further applied in the next section exploring particular arrhythmias that you may encounter in your clinical practice.
4.0 Arrhythmia Interpretation

Arrhythmias occur when there is an alteration to the usual initiation or conduction of the heart's electrical impulses. It is helpful at this point to identify some of the terminology you will use to describe arrhythmias. Their site of origin is not the only method of classifying abnormalities; it is also done by the mechanism that is occurring for example in an adult:

<table>
<thead>
<tr>
<th>Term</th>
<th>Definition</th>
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<tbody>
<tr>
<td>Tachycardia:</td>
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<tr>
<td>Bradycardia</td>
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<tr>
<td>Accelerated</td>
<td></td>
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<tr>
<td>Ectopic beats or rhythms</td>
<td></td>
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<tr>
<td>Escape beats or rhythms</td>
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<tr>
<td>Flutter</td>
<td></td>
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<tr>
<td>Fibrillation</td>
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<tr>
<td>AV Blocks</td>
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</table>

Caution
When identifying an arrhythmia always check the patient’s haemodynamic status and level of consciousness to ensure that the arrhythmia is not life threatening.

Arrhythmias include sinus node, atrial, junctional and ventricular. [2]
You may have rhythm workbooks or simulators in your clinical unit and or healthcare facility which will enhance your skills.

Required Reading
Access a current and relevant text and review the section on arrhythmias, in particular:
- Sinus node arrhythmias
- Atrial arrhythmias
- AV junctional arrhythmias

Identify from your reading the following:
- Mechanism of the arrhythmia
- Identifying features
- Causes
- Clinical significance
- Broad treatment options – treatment will be discussed in detail later in this course, examples of rhythms provided and their identify features.
4.1 Sinus Node Arrhythmias

These arrhythmias originate in the sinoatrial node. They are also known as supraventricular arrhythmias, along with atrial and junctional rhythm disturbances. The arrhythmias in this section include [1, 2]:

- Sinus bradycardia
- Sinus tachycardia
- Sinus arrhythmia
- Sinus arrest and or sinoatrial block.

4.1.1 Sinus bradycardia

Sinus bradycardia is an arrhythmia originating from the sinoatrial node (SA) and is characterised by a heart rate < 60 beats per minute. It can be a normal variant, especially in athletes and during sleep. [5,9] Causes of sinus bradycardia include:

- Excessive inhibitory vagal tone on the SA node caused by vomiting, valsalva maneuvers or neurocardiogenic syncope;
- In response to administration of several types of medications including: beta blockers, calcium channel blockers and anti-arrhythmias
- Disease of the SA node
- Acute inferior wall, right ventricular wall myocardial infarctions
- Hypothyroidism
- Hypothermia
- Hypoxia, sleep apnea
- Increased intracranial pressure
- Infection

Sinus bradycardia does not require treatment unless the patient is symptomatic. However if the arrhythmia is accompanied by hypotension, restlessness, dizziness, decreased level of consciousness, syncope, diaphoresis, chest pain, shortness of breath or other signs of hemodynamic compromise, prompt treatment is required. [5,9]

Activity 7

Using the systematic approach provided, interpret the following rhythm

Rhythm Strip: Lead 11


<table>
<thead>
<tr>
<th>Rate</th>
<th>Rhythm</th>
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<td></td>
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<tr>
<td>P waves</td>
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<tr>
<th>Assess Intervals</th>
<th>PR:</th>
<th>QRS:</th>
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<tr>
<td>ST segment / T wave</td>
<td>ST:</td>
<td>T wave:</td>
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<tr>
<th>Interpretation</th>
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Activity 8
Using physiological principals, explain why some bradycardias are not symptomatic.

Activity 9
Source the treatment procedure for symptomatic bradycardia in your hospital’s Adult Advanced Life Support procedure or alternatively, refer to the Australian Resuscitation Council’s guidelines. List two possible treatments.

4.1.2 Sinus tachycardia
Sinus tachycardia is sinus rhythm, characterised by a rate > 100 beats per minute. Sinus tachycardia is a normal physiological response to anything that stimulates the sympathetic nervous system. However sinus tachycardia that persists at rest usually indicates some underlying problem.

- Fever / Infection
- Blood loss
- Anxiety, pain,
- Heart failure
- Anaemia
- Pain
- Dehydration, hypovolemia
- Shock
- Hyperthyroidism
- Medications: adrenaline, atropine, dopamine and dobutamine. [5]

Treatment of sinus tachycardia is directed at correcting the underlying cause. Because this arrhythmia is a physiological response to a decrease in cardiac output, it should never be ignored, as it is one of the earliest indicators of the deteriorating patient. [8]
Activity 10
Using the systematic approach provided, interpret the following rhythm

Rhythm Strip: Lead 11


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<tr>
<th>Rate</th>
<th>Rhythm</th>
<th>P waves</th>
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Assess Intervals

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Interpretation

1. How may changing the sweep speed calibration on the monitor assist in identification of the above rhythm?

4.1.3 Sinus arrhythmia
Sinus arrhythmia occurs when the SA node discharges irregularly. It is a normal occurrence and observed in children and the elderly. It is commonly associated with the phases of respiration due to changes in the intrathoracic pressure: the heart rate increases with inspiration and decreases with expiration. Other than this aphasic increase and decrease in rate, sinus arrhythmia looks like normal sinus rhythm and does not require treatment. [5,8]

Activity 11
Using the systematic approach provided, interpret the following rhythms

Rhythm Strip: Lead 11


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<th>Rate</th>
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<th>P waves</th>
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Assess Intervals

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Interpretation
Activity 12
Using a good text book outline a clinical situations where sinus arrhythmia may be prominent.

4.1.4 Sinus Arrest / Pause
In sinus arrest/pause the pacemaker cells of the sinoatrial node (SA) have failed to initiate an electrical impulse for one or more beats. This delay results in the absence of a p wave at the time when it is expected to occur and unless there is an escape from a lower pacemaker site either from the Atrioventricular junction or the ventricles the QRS is also missing. Treatment of sinus arrest is aimed at the cause and increasing ventricular rate if the patient is symptomatic. When interpreting the rhythm we describe length of time arrest/pause as occurred in seconds.

Rhythm Strip: Lead 11

| Rate | Rhythm | P waves |
| Asses Intervals | PR: | QRS: | QT: |
| ST segment / T wave | ST: | T wave: |
| Interpretation |

Activity 13
Print a rhythm strip and interpret it using the framework provided in this section. Discuss your interpretation with your resource person.

Rhythm Strip Placed Here

Comments:
_____________________________________________________________________________________________
_____________________________________________________________________________________________
_____________________________________________________________________________________________
4.2 Atrial Arrhythmias

Atrial arrhythmias result form impulses originating form outside the sinoatrial node. In your reading you should have explored the following atrial arrhythmias:

- Premature Atrial Contractions (PACs) or atrial ectopics
- Atrial Flutter
- Atrial Fibrillation
- (Supraventricular Tachycardia (SVT)) you will find SVT in the junctional arrhythmias section in some textbooks.

Caution

If patients are unstable and have serious haemodynamic compromise the patient should undergo urgent cardioversion.

4.2.1 Premature Atrial Contractions (PACs)

This is a single ECG complex that occurs when an electrical impulse starts outside of the SA node from an irritated spot and fires an impulse before the next normal impulse of the sinus node. It can be caused by caffeine, alcohol, nicotine and anxiety or may be associated with respiratory failure, hypoxia or digoxin toxicity among others. They are common in normal hearts and if infrequent no treatment is required, the person reports a palpitation or skipping a beat. If they are frequent (more than 6 a minute) they may indicate an underlying issue which required treatment. The classic characteristic on a ECG is a premature ‘P’ wave with an abnormal configuration.

Diagnostic Criteria for Premature Atrial Ectopic

<table>
<thead>
<tr>
<th>Rate</th>
<th>Underlying rhythm</th>
</tr>
</thead>
<tbody>
<tr>
<td>Rhythm</td>
<td>Irregular: P - P interval varies for ectopic beat.</td>
</tr>
<tr>
<td>P waves</td>
<td>P AC varies in shape, size (morphology) compared with sinus beats</td>
</tr>
<tr>
<td>Assess Intervals</td>
<td>PR: Normal or short</td>
</tr>
<tr>
<td>ST segment / T wave</td>
<td>ST segment isoelectric, T wave - upright</td>
</tr>
<tr>
<td>Site of origin</td>
<td>Multiple sites in the atria.</td>
</tr>
</tbody>
</table>

4.2.2 Atrial Fibrillation (AF)

Irregular narrow complex tachycardia is commonly atrial fibrillation (AF). This is the most common of all cardiac arrhythmias arising from the upper chambers of the heart, occurring in 1-2% of the general population. The arrhythmia is caused by multiple ectopic atrial site or sites. Irritable sites from the atrial can fire at 400-600 times/minute, (incomplete depolarisation) this causes a rapid, disorganized and uncoordinated twitching of atrial musculature.

There is no P wave. Instead, the baseline looks erratic (wavy). These wavy deflections are called fibrillatory waves, “f” waves. In AF the atrioventricular (AV) node attempts to protect the ventricles from hundreds of impulses bombarding it per minute by blocking many of those impulses and only randomly conducting through to
the ventricles by the normal conduction pathway. The hallmark of this arrhythmia is that it produces an irregular heart rhythm. The clinical consequences of AF are ineffectual atrial contraction (loss of atrial kick), decrease stroke volume (atrial contraction contributes up to 30% of cardiac output) and the risk of systemic emboli, particularly stroke. Initial treatment is aimed at eliminating the cause, ventricular rate control, restoring and maintaining sinus rhythm if possible and preventing thromboembolism, in particular stroke, through anticoagulation medication.

---

Diagnostic Criteria for Atrial Fibrillation

Rate | Atrial rate 400 – 600 bpm, Ventricular rate: variable
Rhythm | Ventricular rhythm: irregularly irregular
P waves | Not present. Fibrillatory waves present varying in size and shape from course to fine
Assess Intervals | PR: not measurable, QRS: < 0.12 sec, QT: < 0.45 sec
ST segment / T wave | ST segment isoelectric, T wave - upright
Site of origin | Atria

---

Activity 14

Using the systematic approach provided, interpret the following rhythms

Rhythm Strip: Lead 11

Rate
Rhythm
P waves
Assess Intervals | PR: | QRS: | QT:
ST segment / T wave | ST: | T wave:
Interpretation
Activity 15
Source: Investigate the tool utilised in your unit for atrial fibrillation stroke risk i.e. CHADS2 Score or CHA2DS2-VASc. Discuss with your facilitator how the tool is used to determine anticoagulant therapy for patients in atrial fibrillation

Comments.

4.2.3 Atrial Flutter
Atrial Flutter is an ectopic atrial rhythm in which the irritable site fires regularly at a very rapid rate between 250-400 times per minute (typically 300). This extremely rapid stimulation produces waveforms that resemble the teeth of a saw, called “flutter” waves. A healthy AV node cannot usually conduct faster than approximately 180 impulses / minute. Thus with an atrial rate of 300 bpm every other impulse that arrives at the AV node while it is still refractory. The resulting ventricular response of 150 bpm is called 2:1 conduction. Therefore:

The ratio of atrial rate 300 impulses / minute and ventricular rate 150 bpm is 2:1
The ratio of atrial rate 300 impulses / minute and ventricular rate 100 bpm is 3:1
The ratio of atrial rate 300 impulses / minute and ventricular rate 75 bpm is 4:1
The ratio of atrial rate 300 impulses / minute and ventricular rate 50 bpm is 6:1 [5,9]

The AV conduction ratios are generally constant producing a regular ventricular rhythm. However if AV conduction varies, usually due to disease of the AV node, then the ventricular rate will be irregular. Rarely is there 1:1 conduction unless the AV node has been bypassed with an accessory pathway producing extremely rapid rates. [9]

Diagnostic Criteria for Atrial Flutter [9]

<table>
<thead>
<tr>
<th>Rate</th>
<th>Atrial rate 250 – 400 bpm, Ventricular rate: half the atrial rate or less</th>
</tr>
</thead>
<tbody>
<tr>
<td>Rhythm</td>
<td>Atrial regular, ventricular rate variable determined by AV conduction</td>
</tr>
<tr>
<td>P waves</td>
<td>Not present. Flutter waves (sawtooth) 'F' waves</td>
</tr>
<tr>
<td>Assess Intervals</td>
<td>PR: not measurable</td>
</tr>
<tr>
<td>ST segment / T wave</td>
<td>F wave buried in T wave</td>
</tr>
<tr>
<td>Site of origin</td>
<td>Atria</td>
</tr>
</tbody>
</table>

Activity 16
Using the systematic approach provided, interpret the following rhythm

Rhythm Strip: Lead 11

<table>
<thead>
<tr>
<th>Rate</th>
<th>Rhythm</th>
<th>P waves</th>
</tr>
</thead>
</table>

Assess Intervals

<table>
<thead>
<tr>
<th>PR:</th>
<th>QRS:</th>
<th>QT:</th>
</tr>
</thead>
</table>

<table>
<thead>
<tr>
<th>ST segment / T wave</th>
<th>ST:</th>
<th>T wave:</th>
</tr>
</thead>
</table>

Interpretation

4.2.4 Supraventricular Tachycardia

Supraventricular Tachycardia (SVT) is an umbrella term used to describe a group of arrhythmias that originate above ventricles (resulting in a narrow QRS complex), rate greater than 150 beats per minute, but exact mechanism cannot be determined from the ECG. The most common rhythms included in this term are atrial tachycardia, atrial flutter, atrial fibrillation and junctional tachycardia. Once the specific dysrhythmia has been identified the rhythm is referred to by the specific name. Paroxysmal SVT is an SVT that abruptly starts and self terminates. Drugs such as adenosine, β-blockers, or calcium channel blockers (verapamil and diltiazem), can slow the ventricular rate or terminate many SVTs

Diagnostic Criteria for Supraventricular Tachycardia


<table>
<thead>
<tr>
<th>Rate</th>
<th>150-250 bpm</th>
</tr>
</thead>
<tbody>
<tr>
<td>Rhythm</td>
<td>Regular</td>
</tr>
<tr>
<td>P waves</td>
<td>Buried in T wave</td>
</tr>
<tr>
<td>Assess Intervals</td>
<td>PR: not measurable</td>
</tr>
<tr>
<td>Site of origin</td>
<td>Above the ventricles</td>
</tr>
</tbody>
</table>
Activity 17
Using the systematic approach provided, interpret the following rhythm

Rhythm Strip: Lead 11

<table>
<thead>
<tr>
<th>Rate</th>
<th>Rhythm</th>
<th>P waves</th>
</tr>
</thead>
</table>

Assess Intervals

<table>
<thead>
<tr>
<th>PR:</th>
<th>QRS:</th>
<th>QT:</th>
</tr>
</thead>
</table>

ST segment / T wave

<table>
<thead>
<tr>
<th>ST:</th>
<th>T wave:</th>
</tr>
</thead>
</table>

Interpretation

4.3 AV Junctional Arrhythmias

Atrioventricular (AV) junctional impulses originate from cells located within the AV junctional area (terminal portion of the AV node and the proximal part of the bundle of His). As this impulse does not originate from the SA node there will not be a normal P wave, however the QRS complex will generally resemble that of sinus rhythm as the ventricles have been activated by the normal conduction pathway. The SA node is usually the heart's primary pacemaker however the AV junction may assume responsibility for pacing the heart if:

- The SA node fails to discharge
- The rate of discharge of the SA node is slower than that of the AV junction
- An impulse from the SA node is generated and conducted through the atria, but is not conducted to the ventricles (such as an 3rd degree AV block) \(^5\)

The junctional escape mechanisms are all protective to ensure cardiac output is maintained.

Junctional beats / rhythms may have three possible presentations on the ECG, depending on the location of the junctional pacemaker and the speed of conduction of the impulse into the atria and ventricles. \(^5\) The distinguishing ECG features are:

- P wave may be inverted preceding the QRS. This is due to retrograde delopolarisation of the atria.
- P wave is absent (usually buried in QRS) or
- The P wave appears after the QRS complex, it may be hidden in the ST segment or distort the shape of the T wave. \(^9\)

AV junctional rhythms are further classified by their rate:

- Junctional escape rhythm 40-60 beats per minute
- Accelerated Junctional 60-100 beats per minute
- Junctional Tachycardia > 100 bpm \(^5,9\)

Premature junctional ectopic beats (PJC) occurs when an irritable site within the AV junction fires before the next sinoatrial node beat is due to fire. These are easy to identify as they have the same ECG features as junctional rhythms. \(^8\)
Diagnostic Criteria for Premature Junctional Complex \[^9\]

<table>
<thead>
<tr>
<th>Rate</th>
<th>Underlying rhythm</th>
</tr>
</thead>
<tbody>
<tr>
<td>Rhythm</td>
<td>Irregular: P–P or R–R interval varies for ectopic beat.</td>
</tr>
<tr>
<td>P waves</td>
<td>Absent / inverted lead 11 or after QRS</td>
</tr>
<tr>
<td>Assess Intervals</td>
<td>PR: &lt; 0.12 sec</td>
</tr>
<tr>
<td>ST segment / T wave</td>
<td>ST segment isoelectric, T wave - upright</td>
</tr>
<tr>
<td>Site of origin</td>
<td>Ectopic pacemaker in AV junction.</td>
</tr>
</tbody>
</table>

Activity 18
Using the systematic approach provided, interpret the following rhythms

Rhythm Strip: Lead 11

<table>
<thead>
<tr>
<th>Rate</th>
<th>Rhythm</th>
<th>P waves</th>
<th>Assess Intervals</th>
<th>PR:</th>
<th>QRS:</th>
<th>QT:</th>
</tr>
</thead>
<tbody>
<tr>
<td>ST segment / T wave</td>
<td>ST:</td>
<td>T wave:</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Interpretation

Rhythm Strip: Lead 11

<table>
<thead>
<tr>
<th>Rate</th>
<th>Rhythm</th>
<th>P waves</th>
<th>Assess Intervals</th>
<th>PR:</th>
<th>QRS:</th>
<th>QT:</th>
</tr>
</thead>
<tbody>
<tr>
<td>ST segment / T wave</td>
<td>ST:</td>
<td>T wave:</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Interpretation
4.4 Ventricular arrhythmias

Ventricular arrhythmias result from an ectopic focus in any portion of the ventricular myocardium. They are represented on an ECG as having wider QRS complexes than normal and the P wave is absent as atrial depolarization does not occur. They are potentially dangerous as the ventricles are ultimately responsible for cardiac output. It is therefore imperative that the nurse is able to recognize ventricular arrhythmias to increase chances of successful resuscitation.

Required Reading

Refer to a current and relevant textbook and read the section on ventricular rhythms, concentrating on rhythm interpretation.

- Premature ventricular contractions (PVCs)
- Idioventricular rhythm or ventricular escape rhythm
- Ventricular tachycardia
- Torsades de Pointes (multi focal ventricular tachycardia)
- Ventricular fibrillation
- Asystole

In the following reading note the:
- Mechanism of the arrhythmia, identifying features, causes
- Clinical significance
- Broad treatment options – treatment will be discussed in detail later in this module
- Examples of rhythms provided and their identifying features.
4.4.1 Premature Ventricular Contraction

Premature Ventricular Contractions (PVC’s) is an extra ventricular complex consisting of an abnormally wide and bizarre QRS complex that has originates from ventricular tissue. [9] PVCs may occur from one or multiple focus sites and can occur singularly, in groups of 2 or more, or patterned. PVCs are not dangerous in people with normal hearts but are associated with higher mortality rates in patients with structural heart disease or acute myocardial infarct, particularly when left ventricular function is reduced. [8]

### Diagnostic Criteria for Premature Ventricular Complexes [5,9]

<table>
<thead>
<tr>
<th>Rate</th>
<th>Underlying rhythm</th>
</tr>
</thead>
<tbody>
<tr>
<td>Rhythm</td>
<td>Irregular: P–P or R-R interval varies for ectopic beat.</td>
</tr>
<tr>
<td>P waves</td>
<td>Absent, inverted lead 11 or after QRS</td>
</tr>
<tr>
<td>Assess Intervals</td>
<td>PR: not associated with PVC</td>
</tr>
<tr>
<td></td>
<td>PVC QRS: &gt;0.12 sec with bizarre configuration</td>
</tr>
<tr>
<td></td>
<td>QT: not usually measured in the PVC</td>
</tr>
<tr>
<td>T wave</td>
<td>T wave – occurs in the opposite direction to the QRS for PVC beat.</td>
</tr>
<tr>
<td>Site of origin</td>
<td>Ectopic pacemaker in ventricular tissue.</td>
</tr>
</tbody>
</table>

### Activity 19

Identify the meaning of the following terms that are commonly used in relation to ectopic beats.

<table>
<thead>
<tr>
<th>Term</th>
<th>Meaning</th>
</tr>
</thead>
<tbody>
<tr>
<td>Unifocal or uniform</td>
<td></td>
</tr>
<tr>
<td>Multifocal or multiformed</td>
<td></td>
</tr>
<tr>
<td>Couplets / Paired beats</td>
<td></td>
</tr>
<tr>
<td>Salvos</td>
<td></td>
</tr>
<tr>
<td>Ventricular Bigeminy</td>
<td></td>
</tr>
<tr>
<td>Ventricular Trigeminy</td>
<td></td>
</tr>
<tr>
<td>R on T PVCs</td>
<td></td>
</tr>
</tbody>
</table>
Activity 20
Answer the following short questions

1. Identify 5 common causes for premature ventricular complexes occurring

2. Why are the QRS complexes widened when they originate in the ventricles?

3. Why are the ST segment and T waves altered? What is different about their appearance?

4.4.2 Ventricular Rhythms

Idioventricular Rhythm (Ventricular Escape Rhythm), Accelerated Idioventricular Rhythms and Ventricular Tachycardia are all arrhythmias originating from an ectopic pacemaker in the bundle branches, purkinje network or the ventricular myocardium. [9]

The distinguishing ECG features are:

- Wide > 0.12 sec bizarre QRS complexes followed by a T wave deflected in the opposite direction to the QRS complex
- QRS shape and size is the same for each complex, (monomorphic) indicating the rhythm is arising from one focus.
- Rhythm is usually regular with equal R-R intervals.
- P waves are generally absent, however if present are not related to the QRS complex (AV dissociation). [8,9]

These rhythms are classified by their rate:

- Idioventricular Rhythm less than 40 beats per minute.
- Accelerated Idioventricular Rhythm 40-100 beats per minute.
- Ventricular Tachycardia 100 – 250 beats per minute. (Usually >150 and <200 beats per minute)[9]

4.4.3 Idioventricular Rhythm (Ventricular Escape Rhythm)

This arrhythmia acts as a protective mechanism. In the hierarchy of cardiac pacemakers, the ventricular escape pacemaker is the lowest and slowest. If it fails to fire when needed the result is asystole. [5]
4.4.4 Accelerated Idioventricular Rhythm
The mechanism for Accelerated Idioventricular Rhythm is usually enhanced automaticity and is commonly seen clinically post thrombolytic therapy, when reperfusion of the damaged myocardium occurs, post inferior myocardial infarction and patients with digitalis toxicity. [9]

Diagnostic Criteria for AIVR [9]

<table>
<thead>
<tr>
<th>Rate</th>
<th>40-100 bpm</th>
</tr>
</thead>
<tbody>
<tr>
<td>Rhythm</td>
<td>Regular</td>
</tr>
<tr>
<td>P waves</td>
<td>Present or absent, no relationship to Ventrices</td>
</tr>
<tr>
<td>Assess Intervals</td>
<td>PR: none QRS: &gt; 0.12 sec abnormal QT: none</td>
</tr>
<tr>
<td>Site of origin</td>
<td>Ventricles</td>
</tr>
</tbody>
</table>

4.4.5 Ventricular tachycardia (Monomorphic VT)
Ventricular Tachycardia develops when three or more PVCs occur in row at a rate greater than 100 bpm and is often as a result from either enhanced automaticity or re-entry mechanism. VT may be haemodynamically stable or pulseless depending on the ventricular rate and filling time, however if left untreated may deteriorate into ventricular fibrillation (VF). VT is said to be sustained if it lasts greater than 30 seconds and non-sustained if it self terminates within a 30 second period. [5]
Diagnostic Criteria for VT

<table>
<thead>
<tr>
<th>Rate</th>
<th>100-250 bpm</th>
</tr>
</thead>
<tbody>
<tr>
<td>Rhythm</td>
<td>Regular</td>
</tr>
<tr>
<td>P waves</td>
<td>Present or absent, no relationship to Ventrictles</td>
</tr>
<tr>
<td>Assess Intervals</td>
<td>PR: none</td>
</tr>
<tr>
<td>Site of origin</td>
<td>Ventrictles</td>
</tr>
</tbody>
</table>

4.4.6 Torsades de Pointes (Polymorphic Ventricular Tachycardia)

Torsades de Pointes (TSP) means "twisting on points", a type of polymorphic VT. TSP is characterised by QRS complexes that gradually change back and forth form one shape, size and direction to another and appears to twist around the isoelectric line. Torsades de Pointes may be sustained or unsustained and is usually arises in patients who have a prolonged QT interval. This can occur as an inherited phenomenon in some families, or from medications including anti-arrhythmia drugs or less commonly as a manifestation of myocardial ischemia

Diagnostic Criteria for Torsades de Pointes

<table>
<thead>
<tr>
<th>Rate</th>
<th>100-250 bpm</th>
</tr>
</thead>
<tbody>
<tr>
<td>Rhythm</td>
<td>Regular</td>
</tr>
<tr>
<td>P waves</td>
<td>Present or absent, no relationship to Ventrictles</td>
</tr>
<tr>
<td>Assess Intervals</td>
<td>PR: none</td>
</tr>
<tr>
<td>Site of origin</td>
<td>Ventrictles</td>
</tr>
</tbody>
</table>

4.4.7 Ventricular Fibrillation

Ventricular Fibrillation (VF) is one of the most common cardiac arrest arrhythmias and is fatal without immediate treatment. It is characterised on ECG by chaotic asynchronous electrical activity originating from numerous ectopic sites in the Purkinje network or the ventricular myocytes. There is no organised depolarisation of the ventricles, resulting in uncoordinated quivering of the ventricular musculature. There is no effective myocardial contraction and no pulse. VF can be described as being course (> 3mm in amplitude) indicating a more recent onset of the arrhythmia or fine (<3mm in amplitude).
Diagnostic Criteria for Ventricular Fibrillation\(^9\)

<table>
<thead>
<tr>
<th>Rate</th>
<th>None</th>
</tr>
</thead>
<tbody>
<tr>
<td>Rhythm</td>
<td>None</td>
</tr>
<tr>
<td>P waves</td>
<td>absent</td>
</tr>
<tr>
<td>PR</td>
<td>None</td>
</tr>
<tr>
<td>QRS</td>
<td>“f” waves, variable size, shape, chaotic</td>
</tr>
<tr>
<td>QT</td>
<td>None</td>
</tr>
<tr>
<td>Site of origin</td>
<td>Ventricle</td>
</tr>
</tbody>
</table>

**Caution**

When a monitor shows VF check the patient immediately to establish whether this VF requiring immediate defibrillation, or whether the appearance is due to artifact for example the patient brushing his or her teeth. \(^{13}\)

### 4.4.5 Asystole

Asystole is the absence of ventricular rhythm; there is no QRS complex, no pulse and no cardiac output. Some atrial electrical activity may be evident and this rhythm is called “P wave” asystole or ventricular standstill.

Diagnostic Criteria for Asystole\(^9\)

<table>
<thead>
<tr>
<th>Rate</th>
<th>None</th>
</tr>
</thead>
<tbody>
<tr>
<td>Rhythm</td>
<td>None</td>
</tr>
<tr>
<td>P waves</td>
<td>Present or absent</td>
</tr>
<tr>
<td>PR</td>
<td>None</td>
</tr>
<tr>
<td>QRS</td>
<td>None</td>
</tr>
<tr>
<td>QT</td>
<td>None</td>
</tr>
</tbody>
</table>

4.5 Heart Blocks – Atrioventricular Blocks (AV Blocks)

Heart blocks arise from an interruption in the conduction of impulses from the atria to the ventricles and can be total, partial or it may delay conduction. [1]

Required Reading
Access a current and relevant textbook and read the section on atrioventricular blocks, concentrating on rhythm interpretation.

- First degree AV block
- Second degree AV block
  - Mobitz type I or Wenckebach
  - Mobitz type II
- Third degree or complete heart block.

In the following reading note the:
- mechanism of the arrhythmia, identifying features and causes
- clinical significance
- broad treatment options – treatment will be discussed in detail later in this module.

4.5.1 First Degree AV Block

First degree AV block is defined as a prolonged AV conduction time of the electrical impulse from the atria to the ventricles. [5]

Diagnostic Criteria for First degree AV block [9]

<table>
<thead>
<tr>
<th>Rate</th>
<th>60-100 bpm</th>
</tr>
</thead>
<tbody>
<tr>
<td>Rhythm</td>
<td>Regular: P-P interval equal, R-R interval equal</td>
</tr>
<tr>
<td>P waves</td>
<td>Positive lead II, Present and before every QRS. 1Pwave: 1QRS complex conduction ratio</td>
</tr>
<tr>
<td>Intervals PR</td>
<td>&gt;0.20 but constant</td>
</tr>
<tr>
<td>Site of Origin</td>
<td>Sinoatrial node.</td>
</tr>
</tbody>
</table>
Activity 21
Using the systematic approach provided, interpret the following rhythm

**Rhythm Strip: Lead 11**

<table>
<thead>
<tr>
<th>Rate</th>
<th>Rhythm</th>
<th>P waves</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Assess Intervals</th>
<th>PR:</th>
<th>QRS:</th>
<th>QT:</th>
</tr>
</thead>
<tbody>
<tr>
<td>ST segment / T wave</td>
<td>ST:</td>
<td>T wave:</td>
<td></td>
</tr>
</tbody>
</table>

| Interpretation | |
|----------------||

4.5.2 Second degree AV Block
Second degree AV block occurs when one atrial impulse at a time fails to be conducted to the ventricles. Second degree AV block is divided into two categories: type 1 and type 2.

4.5.1-1 Second degree AV Block Type 1 (Wenchebach or Mobitz 1)
Second degree AV Block Type 1 often referred to as Wenchebach or Mobitz 1, is characterised by a progressive lengthening in conduction times of consecutive atrial impulses into the ventricles (gradual lengthening of the PR interval) until one impulse fails to conduct, culminating in a dropped beat (missing QRS) after which the cycle repeats itself. [9]

**Diagnostic Criteria for Second degree AV Block Type 1** [9]

<table>
<thead>
<tr>
<th>Rate</th>
<th>Can occur at sinus or atrial rate</th>
</tr>
</thead>
<tbody>
<tr>
<td>Rhythm</td>
<td>Irregular: P-P interval equal, R-R interval unequal</td>
</tr>
<tr>
<td>P waves</td>
<td>Positive lead II, usually followed by QRS, periodically a P wave is not followed by a QRS complex</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Intervals</th>
<th>PR: Progressive lengthening, culminating in a dropped beat. Then PR interval returns to original length and the cycle repeats again.</th>
<th>QRS: &lt; 0.10 sec</th>
<th>QT: &lt; 0.46 sec</th>
</tr>
</thead>
<tbody>
<tr>
<td>Site of Origin</td>
<td>Sinoatrial node or atrial pacemaker</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
Activity 22
Using the systematic approach provided, interpret the following rhythm

Rhythm Strip: Lead 11

<table>
<thead>
<tr>
<th>Rate</th>
<th>Rhythm</th>
<th>P waves</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Assess Intervals</th>
<th>PR:</th>
<th>QRS:</th>
<th>QT:</th>
</tr>
</thead>
<tbody>
<tr>
<td>ST segment / T wave</td>
<td>ST:</td>
<td>T wave:</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Interpretation</th>
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</table>

**4.5.1-2 Second degree AV Block Type 11 (Mobitz 11)**

Second degree AV Block Type 11 often called Mobitz 11, is characterised by a sudden failure of conduction of an atrial impulse to the ventricles. This is characterised on ECG as a P wave not followed by a QRS complex. The conduction delay in second degree AV block type 11 occurs below the AV node, either in the bundle of His or at the level of the bundle branches. This is considered to be a more serious block than type 1 and often progresses to third degree AV block. [5,8]

**Diagnostic Criteria for Second degree AV Block Type 11 (Mobitz 11)** [9]

<table>
<thead>
<tr>
<th>Rate</th>
<th>Can occur at any basic rate</th>
</tr>
</thead>
<tbody>
<tr>
<td>Rhythm</td>
<td>Irregular: P-P interval equal. R-R interval unequal</td>
</tr>
<tr>
<td>P waves</td>
<td>Positive lead II, usually followed by QRS, periodically a P wave is not followed by a QRS complex</td>
</tr>
<tr>
<td>Intervals</td>
<td>PR: Constant 0.12-0.2 sec, or prolonged. Random or patterned dropped QRS complex.</td>
</tr>
<tr>
<td>QT:</td>
<td>&lt; 0.46 sec</td>
</tr>
<tr>
<td>Site of Origin</td>
<td>Sinoatrial node or atrial pacemaker</td>
</tr>
</tbody>
</table>

Activity 23
Using the systematic approach provided, interpret the following rhythm

Rhythm Strip: Lead 11

<table>
<thead>
<tr>
<th>Rate</th>
<th>Rhythm</th>
<th>P waves</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
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</tbody>
</table>

Assess Intervals

<table>
<thead>
<tr>
<th>PR:</th>
<th>QRS:</th>
<th>QT:</th>
</tr>
</thead>
</table>

ST segment / T wave

<table>
<thead>
<tr>
<th>ST:</th>
<th>T wave:</th>
</tr>
</thead>
</table>

Interpretation

4.5.2 Third degree AV Block Type 11

Third degree heart block is characterised by the complete failure of conduction of all atrial impulses to the ventricles. In third degree AV block the impulses generated by the sinoatrial node are blocked before reaching the ventricles, so no P waves are conducted. The atria and ventricles are dissociated from each other and therefore beat independently.\(^5,8\)

A secondary pacemaker either from the junctional or ventricle areas stimulates the ventricles. If the ventricular escape is from the high junctional area the QRS will be narrow and usually produces a ventricular rate greater than 40 bpm. However if the ventricular escape is from the low purkinje system the QRS will be wide and usually conducted at a slower rate of less than 40 bpm.\(^5,8\)

Diagnostic Criteria for Third degree AV Block \(^9\)

<table>
<thead>
<tr>
<th>Rate</th>
<th>Can occur at any basic rate</th>
</tr>
</thead>
<tbody>
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<td>Rhythm</td>
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<tr>
<td>Intervals</td>
<td>PR: 0.12-0.2 sec, or prolonged but constant until dropped QRS complex</td>
</tr>
<tr>
<td>Site of Origin</td>
<td>Sinoatrial node or atrial pacemaker</td>
</tr>
</tbody>
</table>
Activity 24
Using the systematic approach provided, interpret the following rhythm

Rhythm Strip: Lead 11

<table>
<thead>
<tr>
<th>Rate</th>
<th>Rhythm</th>
<th>P waves</th>
</tr>
</thead>
</table>

<table>
<thead>
<tr>
<th>Assess Intervals</th>
<th>PR:</th>
<th>QRS:</th>
<th>QT:</th>
</tr>
</thead>
<tbody>
<tr>
<td>ST segment / T wave</td>
<td>ST:</td>
<td>T wave:</td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Interpretation</th>
</tr>
</thead>
</table>

4.6 Summary
This section discussed the mechanisms for identifying arrhythmias and the process of identifying the many different arrhythmias that occur. The following may facilitate remembering the criteria for arrhythmia interpretation:

- Learn the features of sinus rhythm and what all the waveforms, intervals and segments represent.
- Learn the identifying features of groups of rhythms by their origin, for example ventricular rhythms are wide and bizarre, as conduction does not follow the normal pathway, no regular P wave.
- Identify the mechanism present, for example tachycardia, flutter.

For further practice at interpretation of rhythms, internet sites with rhythm strips can be accessed for practice and associated with tests to assess your skills. Development of skills in rhythm interpretation will take practice, practice and then some more practice!

Further Reading
For further practice at interpretation of rhythms, internet sites with rhythm strips can be accessed for practice and associated with tests to assess your skills. Development of skills in rhythm interpretation will take practice, practice and then some more practice!

You may find the following website useful
http://www.skillstat.com/tools/ecg-simulator/-home
5.0 Management of Arrhythmias

The overall aim of arrhythmia management is to restore sinus rhythm and prevent recurrences. If this is not possible then the aim is to ‘normalise’ the ventricular rate and increase cardiac output. Any decrease in cardiac output which can result from arrhythmias results in decreased perfusion to the body including the gut and muscle tissue, leading to wider adverse effects. Remember that if you do not correct the underlying cause then the arrhythmia will return. The patient’s haemodynamic status will dictate urgency of management.

Knowledge of the underlying physiology of cardiac cells in relation to conduction and contraction is vital to understanding the actions of anti arrhythmic drugs. Mechanical, chemical or electrical stimulus may lead to cell depolarisation that is excitation.

Required Reading

Access a current and relevant textbook and review the section on management of tachycardias. Access UptoDate via QHEPS and search ‘acute management of tachyarrhythmias’ and review the current literature that is available.


5.1 General Considerations in arrhythmia management

Arrhythmia management depends on whether the arrhythmia is acute or chronic, the underlying cause, and the hemodynamic effects. When a new arrhythmia is present or suspected, assessment of the patient using the ABCDE approach should be utilised including:

- Establish cardiac monitoring if not already present and assess the nature of the arrhythmia
- Monitor vital signs
- Establish oxygen therapy (if appropriate)
- Obtain a 12 lead ECG
- Assess the patient’s condition for the presence or absence of adverse features. (See below)
- Obtain intravenous access. As much of the management of arrhythmias requires the administration of intravenous drugs, a reliable intravenous (IV) cannula will be needed for drug administration,
- Assessing for factors contributing to the arrhythmia.
- Administering medications as prescribed and observing the beneficial/adverse effects

The Australian Resuscitation Council (ARC) refers to the presence or absence of adverse signs and symptoms as dictating the urgency and choice of treatment for most arrhythmias. They have identified the following list of adverse features indicating that the patient is unstable and at risk of deterioration, wholly or partly because of the arrhythmia.
• **Shock** - Hypotension (systolic blood pressure < 90 mmHg), pallor, sweating, cold extremities, confusion or a decreased level of consciousness.

• **Syncope** – transient loss of consciousness because of a reduction in blood flow to the brain.

• **Heart failure** – pulmonary oedema and/or raised jugular venous pressure

• **Myocardial ischaemia** – typical ischemic chest pain (diabetics may not have typical pain) and/or evidence of myocardial ischemia on a 12 lead ECG.

• **Extreme tachycardia** defined as a heart rate > 150 bpm causing a reduction in cardiac output and coronary perfusion filling time potentially inducing myocardial ischemia.

• **Extreme bradycardia** - defined as a heart rate < 40 bpm. Often tolerated poorly, particularly in patients who have severe heart disease and are unable to compensate for the bradycardia by increasing their stroke volume.\(^{[16]}\)

### 5.1.1 Treatment options

Treatment options may be divided into four broad categories, dependent upon the clinical status of the patient.\(^{[13]}\)

1. **Electrical** – cardioversion for tachyarrhythmias and cardiac pacing for bradyarrhythmias

2. **Simple clinical intervention** – includes vagal manoeuvres

3. **Pharmacological**

4. **No treatment**

In general the accepted practice is to use electrical therapy to treat arrhythmias in the unstable patient or the patient showing adverse features, and reserve pharmacologic agents for the stable patients.\(^{[8]}\) However it is important to recognise that all anti-arrhythmic treatments have the potential to make a rhythm worse, causing clinical deterioration.\(^{[13]}\)

### 5.2 Electrical Therapies

#### 5.2.1 Defibrillation and Cardioversion

If the patient is in a shockable rhythm, the single most important determinant of outcome in patients with ventricular fibrillation (VF) or pulseless ventricular tachycardia (VT) is the time to defibrillation.\(^{[9]}\)

• **Defibrillators** can deliver monophasic or biphasic waveforms. With biphasic waveform, current is delivered in one direction, stops and is then reversed to travel in the opposite direction; biphasic defibrillation terminates arrhythmias more consistently and at lower energies.\(^{[3]}\)

• **Cardioversion** is a synchronised defibrillation which delivers the current on the QRS complex.\(^{[3]}\) It is used to terminate such rhythms as ventricular tachycardia, atrial fibrillation and atrial flutter when there is cardiac output. It is usually an elective procedure and requires light anaesthesia for the patient. For this procedure the monitoring leads from the defibrillator must be attached to the patient (even if using the hands free pads) and the ‘Synchronise’ button must be activated.
5.2.2 Artificial Cardiac Pacing

Cardiac pacing can be used to increase the patient’s heart rate and therefore their cardiac output, or can be used to overdrive pace and take control of the heart when there is tachyarrhythmia. There are four forms of pacing. Three (3) are temporary cardiac pacing – transcutaneous, transvenous and epicardial and the fourth is permanent pacing. Permanent pacing is discussed in the Cardiac Investigations and interventions course.

- **Transcutaneous Pacing** – In an emergency situation, transcutaneous also known as external pacing may be used. This is a non-invasive method of cardiac pacing with an electrode placed on the patient’s anterior chest and a second applied to their back and a pulse generator then emits pacing impulses that travel through the skin to the heart muscle. This is a temporary measure to stabilise the patient’s cardiac output until a transvenous wire can be inserted.

- **Transvenous** – a transvenous pacing wire is inserted through a vein making it possible to insert in a nonsurgical environment. The lead wires are advanced through a catheter to the right atrium or ventricle and connected to a pulse generator. This is a temporary measure until a permanent pacemaker can be inserted.

- **Epicardial Pacing** - epicardial pacing wire(s) are loosely sewn to the surface of the atrium and or ventricle and are removed longer required.

5.3 Simple Clinical Intervention

5.3.1 Vagal Stimulation

This may be used to terminate supraventricular tachyarrhythmias or to slow the ventricular response to enable correct rhythm interpretation. The Valsalva manoeuvre is one such technique.

Carotid sinus massage is another technique which may slow tachyarrhythmias to enable diagnosis of the arrhythmia or occasionally revert the rhythm, for example in AV nodal reentrant tachycardia.

**Caution**

The registered nurse does not perform carotid sinus massage; this procedure is only undertaken by the medical officer following comprehensive assessment of the patient.

Ensure that full resuscitation equipment is available if this manoeuvre is performed. Some patients have carotid sinus sensitivity and could go into asystole requiring full resuscitation.\[5\]
5.4 Pharmacological Therapy

5.4.1 Electrolytes Imbalances and the ECG
Sodium, potassium and calcium ions are vital to cellular depolarization and repolarisation. Magnesium is integral to the absorption of calcium and the maintenance of potassium stores. Electrolytes have a major effect on action potential, by maintaining a stable resting membrane. Therefore evaluation of electrolyte levels is of prime importance when treating cardiac arrhythmias. The rhythm is much more likely to respond to either pharmacological or electrical management if the electrolytes are within normal limits.

Required Reading
Access a current and relevant textbook and read the section on ECG changes associated with electrolytes disturbances; noting the changes to the waveforms caused by each of the electrolyte disturbances.

- Potassium
- Magnesium
- Sodium
- Calcium

As identified in the reading electrolyte disturbances can affect all parts of the cardiac cycle. If there are changes to the waveforms or time intervals on a patient’s ECG or rhythm strip, always remember to check electrolyte levels.

Activity 26
Answer the following questions about electrolytes in relation to cardiology.

1. What are the normal serum blood levels of the following electrolytes? What is the ideal level that these electrolytes are maintained for patients in your unit with an underlying cardiac problem? What is the rational for maintaining these levels?

<table>
<thead>
<tr>
<th>Electrolyte</th>
<th>Normal range mmol/L</th>
<th>Ideal range for cardiac patients</th>
<th>Rationale</th>
</tr>
</thead>
<tbody>
<tr>
<td>Potassium</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Magnesium</td>
<td></td>
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<td></td>
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<tr>
<td>Sodium</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Calcium</td>
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2. Identify three clinical condition/s or medications that may predisposes patients to the development of the following electrolyte imbalances:

(a) Hyperkalaemia / Hypermagnesia

(b) Hypokalaemia / Hypomagnesia.

(c) Hypocalcemia
3. Differentiate between hypokalaemia and hyperkalaemia in terms of the effects on the ECG.\(^5\)

<table>
<thead>
<tr>
<th>Hypokalaemia</th>
<th>Hyperkalaemia</th>
</tr>
</thead>
<tbody>
<tr>
<td>P wave: tall and peaked</td>
<td>P wave:</td>
</tr>
<tr>
<td>QRS complex: widen</td>
<td>QRS complex:</td>
</tr>
<tr>
<td>ST segment:</td>
<td>ST Segment:</td>
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<tr>
<td>T wave:</td>
<td>T wave:</td>
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<tr>
<td>U wave:</td>
<td>U wave:</td>
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</table>

**Reminder**
Electrolytes assist muscular function therefore they are important for the heart to function.

### 5.4.2 Management of Bradyarrhythmias

Management of bradycardia is dependant on how slow the rhythm becomes and the patient’s clinical response to the arrhythmia. Cardiac output decreases sustainability at low ranges of heart rate\(^9\) and can therefore result in increased myocardial ischaemia and haemodynamic compromise.

The following arrhythmias could potentially cause symptomatic bradycardia:
- Sinus bradycardia
- Sinus arrest
- Junctional escape rhythm – occasionally
- Ventricular escape rhythm
- Second degree heart block – occasionally
- Third degree heart block.

**Activity 27**

Outline the common symptoms for symptomatic bradycardia.

**Answer**

**Golden Rule**
Remember that not all patients become compromised with these arrhythmias; check the pulse and blood pressure.
Required Reading


Activity 28  Clinical scenario
Answer the following questions related to the scenario.

Mr. Smith is a 70 yr old gentleman admitted to your unit following a fall at home for a repair of his fractured right neck of femur. He has a history of esophageal reflux and glaucoma. When you answer Mr. Smith’s call bell, he is complaining of dizziness and is short of breath. His blood pressure is 70/40 mmHg.

1. Outline your response to Mr. Smith’s deterioration

2. Mr. Smith’s rhythm strip is shown below. Analyse the rhythm:

Rhythm Strip: Lead 11

<table>
<thead>
<tr>
<th>Rate</th>
<th>Rhythm</th>
<th>P waves</th>
<th>PR:</th>
<th>QRS:</th>
<th>QT:</th>
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<table>
<thead>
<tr>
<th>ST segment / T wave</th>
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</tr>
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<tbody>
<tr>
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</tbody>
</table>

3. What is the type of medication, method of administration and standard dose used to initially manage this arrhythmia?

4. What are the major side effects of the drug?

5. Mr. Rodgers has failed to respond to pharmacotherapy, what other therapy is available on your unit to assist Mr. Rodgers.

6. What are the other types of medications that could be used to manage this arrhythmia if external pacing is not available?
5.4.3 Management of Tachyarrhythmias

Tachyarrhythmias are common occurrences in acutely ill patients and may arise from the upper chambers of the heart (atria) or the lower chambers of the heart (ventricular). In the setting of cardiac arrest most arrhythmias are ventricular in origin. The common symptoms of tachycardia include syncope, shortness of breath, dizziness, chest pain or palpitations.\(^\text{[13]}\)

As the rate increases so does myocardial demands for oxygen.\(^\text{[1]}\) Myocardial ischaemia can result. As the heart rate increases, the diastolic filling time decreases possibly resulting in reduced cardiac output and subsequent symptoms of syncope and low blood pressure.\(^\text{[2]}\)

There are number of tachyarrhythmias including:

- Sinus tachycardia
- Supraventricular tachycardia (includes atrial and junctional tachycardia)
- Atrial fibrillation
- Atrial flutter
- Ventricular tachycardia (sustained)
- Torsades de Pointes.

Management of these arrhythmias varies depending on the site of origin, mechanism of arrhythmia and the patient’s clinical response to the arrhythmia. If the patient is severely compromised with any of the above tachyarrhythmias (except sinus tachycardia) synchronised cardioversion should be considered to facilitate rapid reversion.\(^\text{[9]}\) Treatment of the tachyarrhythmia is directed at the cause.\(^\text{[6]}\)

It has to be remembered that many antiarrhythmic drugs have been shown to have proarrhythmic effects, especially in patients with impaired left ventricular function.\(^\text{[8]}\) Before administering an antiarrhythmic drug, the medical officer and nurse have to balance the risks and benefits of the therapy. To reduce the risk of toxicity the metabolism and excretion of the drug should be considered in relation to the individual patient’s condition and coexisting conditions. A pulseless patient with a tachyarrhythmia requires cardiac arrest management according to the cardiac arrest algorithm.\(^\text{[13]}\)

5.4.4 Atrial Fibrillation (AF)

The debate in the management of AF is whether the aim should be for rhythm control or rate control. There have been a number of studies that have showed rate control was at least as good as rhythm control and should be considered a prime strategy as it may decrease complications and hospitalisations.\(^\text{[10]}\)

Reminder

The danger to patients of remaining in atrial flutter or fibrillation is the formation of a clot in the atria with the potential for embolisation.

Required Reading

Access UptoDate via QHEPS and search the topic ‘atrial fibrillation management’ and or ‘atrial flutter and fibrillation management’ and review the current literature.
5.4.3 Supraventricular Tachycardia (SVT)

Supraventricular Tachycardia (SVT) commonly includes atrial and AV junctional tachycardias and the term is used when it is not possible to differentiate between them. Strictly speaking atrial flutter and atrial fibrillation are also SVTs but their pharmacological management can be different so it will be discussed separately in this module, they are also not commonly referred to as a SVT, but by their specific name. Drugs such as adenosine, β-blockers, or calcium channel blockers (verapamil and diltiazem), can slow the ventricular rate or terminate many SVTs. [6]

Activity 29 Clinical scenario

Answer the following questions related to the scenario.

Mr. Charles is an 82 yr old gentleman admitted yesterday to your unit with a diagnosis of right upper lobe pneumonia. He is currently on oral antibiotic therapy and having 2 lpm of O₂ therapy via nasal prongs and is resting in bed. He has a history of hypertension and sleep apnea. You have just commenced the shift and are introducing yourself to Mr. Charles and observe he is short of breath and his pulse is fast and irregular.

1. Outline five nursing actions in response to this finding

2. You obtain a rhythm strip and it looks like this: analyse the following rhythm:

Rhythm Strip: Lead 11

<table>
<thead>
<tr>
<th>Rate</th>
<th>Rhythm</th>
<th>P waves</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
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<table>
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ST segment / T wave

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</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Interpretation .
Broad Complex Tachycardia's

5.4.5 Ventricular Tachycardia, Torsades de Pointes, Ventricular Fibrillation

Treatment depends on whether the patient has a detectable or undetectable pulse.\(^1\) Treatment for those with a detectable pulse depends on whether their condition is stable or unstable. Some patients may be suitable for cardioversion.

### Required Reading

- Access a current and relevant critical care textbook and review the section on ventricular fibrillation.
- Access MIMS Online via QHEPS or the Australian Medicines Handbook Online and review the section on amiodarone.

### Caution

Patients with wide QRS complex tachycardia should be treated as ventricular tachycardia until a definitive cause is established.\(^1\)

### Activity 30 Clinical scenario

Answer the following questions related to the scenario.

You are a Registered Nurse working in a telemetry unit; the crisis alarm has been activated on the central monitor. You note the following rhythm:

![Rhythm Diagram]

You immediately go to the patient's beside and he is sitting up in bed, talking to visitor and **conscious**.

1. Identify the rhythm

2. Outline your nursing management.
5.5 Life Threatening Situations and rhythms

Asystole, ventricular fibrillation, pulseless ventricular tachycardia, ventricular standstill and pulseless electrical activity (PEA) are medical emergencies. There is no cardiac output and cardiopulmonary resuscitation (CPR) must be initiated immediately if the patient is to survive. Early advanced life support measures are also crucial. Third degree heart block may also be a medical emergency depending on the ventricular rate. In this case, CPR (if indicated) and pacing is the mode of treatment for these patients.

Management of VT and VF has been discussed earlier in this unit; it is imperative for the medical/surgical nurse to have the knowledge to enable rapid recognition and management of these rhythms. Training in basic life support and AED use enables implementation of defibrillation immediately, improving patient outcomes.

For the non shockable emergency rhythms, that is asystole, ventricular standstill and PEA Electromechanical Dissociation (EMD) the priorities of management are:

- Commence BLS
- Call an emergency code as per your facility guidelines.

Required Reading
Access and review the policies, procedures and or work unit guidelines for adult cardiac arrest.


Activity 31
Assess four patients in the unit and for each identify one potential risk factor for development of life threatening arrhythmias.
Activity 31
Analyse the strategies undertaken to prevent or reduce the risk of these patients developing a life threatening arrhythmia.

5.6 Summary
This concludes this section of the course, discussing both the management of arrhythmias which you may encounter in your clinical unit and or healthcare facility. Accurate identification of arrhythmias and appropriate early management improves the patient outcome and potentially decreases the length of stay in hospital. The major effect of arrhythmias is decreased cardiac output, which can lead to other complications. To decrease recurrences of the arrhythmia always look for the cause of the arrhythmia and treat same.

Conclusion
This concludes the Rhythm Monitoring, Interpretation and Management course. This course has provided you with the knowledge and skills to understand concepts of cardiac monitoring, rhythm interpretation, arrhythmia recognition and management. Whilst rhythm interpretation is an important skill it is wise to remember that you must always assess your patient’s haemodynamic status when assessing arrhythmias, as this will dictate the urgency and type of intervention required.

It is not possible in this course to provide enough examples of arrhythmias for you to become highly skilled in rhythm interpretation. You need to do further practice either using the examples in a comprehensive arrhythmia textbook and or a rhythm simulator.
References

2. Farrell, M. and J. Dempsey, eds. Smeltzer and Bare's textbook of medical surgical nursing. 2nd ed. 2010, Lippincott, Williams and Wilkins: Broadway, N.S.W.