Self-Directed Learning Package

Temporary External Pacing

September 2011

Participants Name:

Health
Hunter New England
Local Health District

HUNTER NEW ENGLAND

LOCAL HEALTH DISTRICT

Module prepared by: N. Churms, CNC, BH.

Module reviewed by: Rebecca Butler, CNC, BH. and
HNEH ALS Committee

Acknowledgements and thanks to: Phil Way

For review and update: 2013

The following package has been written using the references cited in the reference list. Much of the information presented in this module outline is considered generic information and is widely reflected in nursing and pharmacology literature; as such it is not feasible to reference all sources of information. Only citations that are directly attributed to a single source/s are referenced in the text presented herein

USING THIS SELF DIRECTED LEARNING PACKAGE

Through out this self-directed learning package there are readings and activities that you need to complete.

This SDLP uses the following icons:

R	**READING** This icon alerts you to undertake reading related to the topic this may include Safe Work Practices, Journal Articles or Books

A	**LEARNING ACTIVITY** This icon denotes a learning activity or competency assessment that you will need to complete

OBJECTIVES

This package aims to introduce the subject of temporary external pacing to the nurse undertaking advanced life support training.

At the completion of this package, the Nurse will:

- Understand the anatomy and physiology of the heart, specifically the conduction system
- Identify and understand why temporary cardiac pacing is necessary;
- Be able to explain the different types of temporary pacing;
- Be able to explain how to prepare for, perform, monitor and evaluate effective transcutaneous cardiac pacing;
- Identify patient care needs and possible complications in relation to temporary cardiac pacing.

Cardiac Anatomy and Physiology

The cardiovascular system consists of the heart, blood vessels, and lymphatics. This network brings life-sustaining oxygen and nutrients to the body's cells, removes metabolic waste products, and carries hormones from one part of the body to another.

The heart is the centre of the cardiovascular system and it is a biochemically driven pump. It is located in the mediastinum beneath the sternum and is about the size of a closed fist.

Surrounded by a sac called the pericardium, the heart has a wall made up of three layers: the epicardium, myocardium, and endocardium.
Within the heart lie four chambers (2 atria and 2 ventricles) and four valves (2 atrioventricular (AV) and 2 semilunar valves).

For further in-depth information on cardiac A&P refer to Moser & Riegel

Copyright © 2007 Cardiac Nursing 1st edition – available via CIAP:

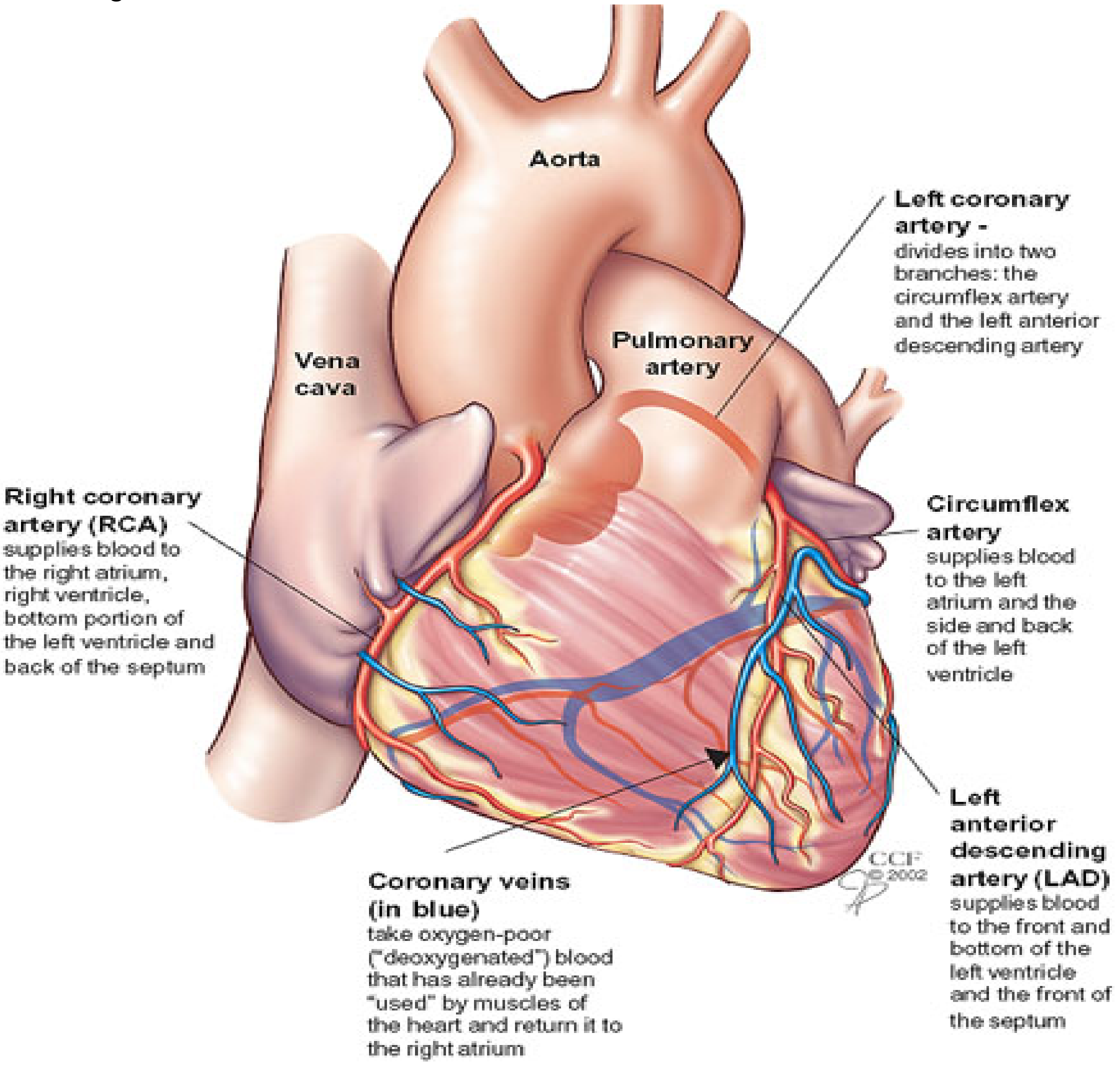
Coronary Circulation: *The Coronary Arteries*.

The heart needs an adequate blood supply to survive.

There are two coronary arteries that arise in the aorta at the sinus of valsalva:

- Right Coronary Artery (RCA).
- Left Coronary Artery, also known as the left main; has two important branches;
    - Left Anterior Descending, LAD.
    - Left Circumflex Artery, LCX.

The arteries transverse the epicardial layer of the heart with numerous branches leaving to distribute blood throughout the myocardium as shown in the diagram below:



The Conduction System

The hearts conduction system causes it to contract, moving blood throughout the body. For this to work properly, the heart requires nerve control, electrical stimulation as well as a mechanical response.

Nervous Control
Both sympathetic and para-sympathetic nerves supply the heart.

♥ **Sympathetic Nerves** - distributed to all parts of the heart.

Stimulation causes:

- Increased rate of sinus nodal discharge.
- Increases rate of conduction.
- Increases excitability.
- Increases force of contraction.

- **Para-sympathetic Nerves** - vagus nerve.

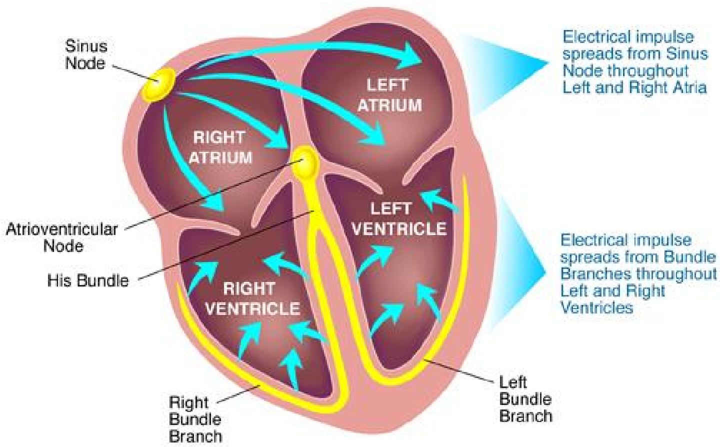
Stimulation causes:

- Acetylcholine release.
- Decreases rate of sinus node discharge.
- Decreases excitability of AV fibres leading to slowing of transmission of impulses through the AV junction.
- Bradycardia and complete heart block can eventuate.

Electrical Stimulation

The cardiac conduction system is a made up of specialised conduction tissue that has inherent properties of;

- Automaticity - ability to generate an electrical impulse automatically.
- Rhythmicity - depolarize the impulse in a regular fashion
- Conductivity – ability to pass the impulse across cell membranes.
- Contractibility – the ability to shorten the fibres in the heart when receiving the impulse.



The conduction system is made up of the following structures that are arranged in order of their dominance. That is the system is designed so that if one part breaks another can take over. The SA node is the overall controller of the system, but if for some reason it fails the next structure down the list, the AV node, will take over;

- Sinoatrial (SA) node.
- Atrioventricular (AV) node.
- Bundle of His.
- Right and left bundle branches.
- Purkinje system.

Sinoatrial (SA) node

- Situated at the junction of superior vena cava and the right atrium.
- Contains approx. 1.5 cm of conducting tissue.
- Fibres of SA node are continuous with atrial fibres so that any action potential developed in the node spreads immediately to the atria.
- Strongest and fastest of all pacemakers.
- Discharges 60-100 times per minute.

Atrioventricular (AV) node

- ♥ Situated in the right posterior portion of the inter-atrial septum near the base of the tricuspid valve and adjacent to the opening of the coronary sinus.
- ♥ Is continuous with the bundle of HIS.
- ♥ Is specially designed to slow conduction
- ♥ Delays conduction of impulses by 0.08-0.12 seconds to allow for ventricular filling.
- ♥ Can discharge at 40-60 times per minute.
- ♥ Allows only one-way conduction so only forward conduction from atria to ventricles can occur.

Bundle of His

- ♥ Extremely rapid conduction of impulses.
- ♥ Composed of a thick bundle of fibres that runs down the right side of the intraventricular septum and then divides into the right and left bundle branches at the muscular portion of the septum.

Right and left bundle branches

- ♥ Right consists of one branch.
- ♥ Left consists of one long thin branch anteriorly and one short thick branch posteriorly.
- ♥ Continuous with purkinje system.

Purkinje system

- ♥ Extremely rapid conduction of impulses.
- ♥ Transports impulse from the atrioventricular node into the ventricles.
- ♥ Can discharge at 20-40 times a minute.

The Cardiac Cycle

Is the time period from the beginning of one heartbeat to the beginning of the next. It involves both mechanical and electrical events. An electrical event (depolarisation) leads to a mechanical event (systole) while electrical recovery (repolarisation) is accompanied by mechanical relaxation (diastole).

<u>**Events:**</u>

1) **Atrial contraction;**

- Known as the atrial kick, atrial systole (coinciding with late ventricular diastole) supplies the ventricles with 30% of the blood for each heart beat.
- Atrial contraction is complete before the ventricle begins to contract
- Is preceded on ECG by P-wave that signals atrial depolarisation

2) **Isovolumic Ventricular Contraction;**

- In response to ventricular depolarisation, tension in the ventricles increases. This raise in pressure within the ventricles leads to closure of the mitral and tricuspid valves.
- The pulmonic and aortic valves stay closed during the entire phase.
- Occurs immediately after QRS complex on ECG.
- In this phase the heart consumes up to 75% of its oxygen

3) **Rapid Ventricular Ejection;**

- When ventricular pressure exceeds aortic and pulmonary arterial pressure, the aortic and pulmonic valves open and the ventricles eject blood
- Two thirds or more of the ventricular volume is ejected.
- The left atrium is relaxed at this time and gradually filling with blood returning from the pulmonary circulation.
- Ventricular repolarisation as indicated by the T-wave begins.

4) **Isovolumic Ventricular Relaxation;**

- When ventricular pressure falls below the pressure in the aorta and pulmonary artery, the aortic and pulmonic valves close.
- All valves are closed during this phase.
- Atrial diastole occurs as blood fills the atria

5) **Rapid Ventricular Filling;**

- Atrial pressure exceeds ventricular pressure, which causes the mitral and tricuspid valves to open.
- Blood then flows passively into the ventricles.
- About 70% of ventricular filling takes place during this phase

The cardiac cycle produces cardiac output, which is the amount of blood the heart pumps in one minute. It’s measured by multiplying heart rate by the stroke volume.

The term stroke volume refers to the amount of blood ejected with each ventricular contraction.

Three factors affect stroke volume – *preload, afterload*, and *myocardial contractibility* – a balance of these three factors produces optimal cardiac output.

For further in-depth information refer to Moser & Riegel Copyright © 2007

Cardiac Nursing 1st edition – available via CIAP:

Temporary External Pacing

The most common reasons for using temporary pacing are to maintain an optimal cardiac output when the patient has developed a symptomatic bradycardia or is asystolic.

The cause for this is frequently due to impairment within the conduction system in the heart. The problem may be located within the atrioventricular (AV) node or below, within the bundle branches coursing through the ventricles. Patients can experience bradycardia as a result of several types of AV blocks. This, in turn, frequently causes decreased cardiac output. Permanent pacemakers can be implanted into these individuals to increase and maintain adequate heart rate – thus temporary pacing provides a bridge until this can be undertaken.

A further reason patient's may require temporary pacing is because they are experiencing symptomatic tachycardias (rates greater than 180) which may produce inadequate ventricular filling with inadequate perfusion. Some very chaotic rhythms, like ventricular fibrillation, require defibrillation. Some fast rhythms (tachycardia) are amenable to overdrive pacing by a pacemaker. These may be atrial tachydysrhythmias (atrial flutter, atrial fibrillation or atrial tachycardia) or ventricular tachycardia. This rapid rate may impair the ability of the heart to eject an adequate stroke volume. A temporary non-invasive pacemaker can be used to pace the atria at a rapid rate. If one of the pacer stimuli falls at the right time, it gains the responsibility for pacing the heart. This is known as overdrive pacing, and it either stops the tachydysrhythmia or converts the patient to a sinus rhythm.

Temporary pacing can be performed by the following routes:

- Epicardial
- Transvenous
- Transoesphageal
- Transcutaneous (the focus of this package)

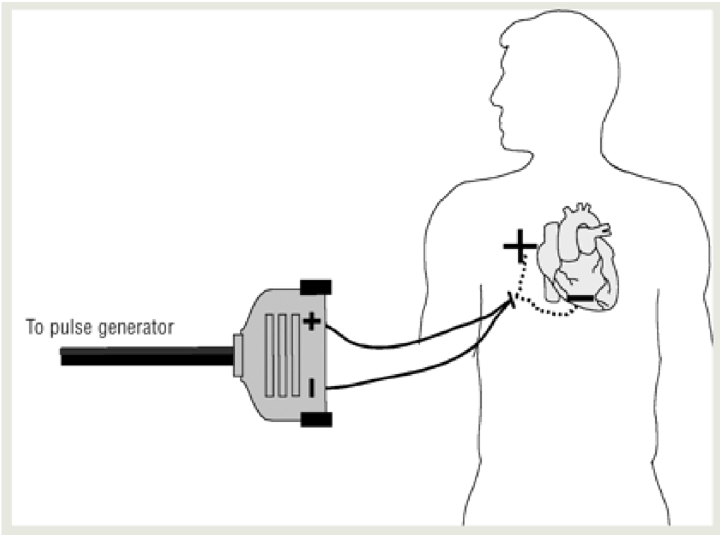
In order to achieve temporary pacing the electrode or lead wire is attached to an external pulse generator capable of providing sufficient energy to pace the heart at the desired rate.

The flow of electrical current is the basis for both normal and artificial depolarisation of the heart. In cardiac pacing there are two important points to remember:

- Electricity always flows from a negative pole to a positive pole. You will notice that the pacemakers are marked in this way.

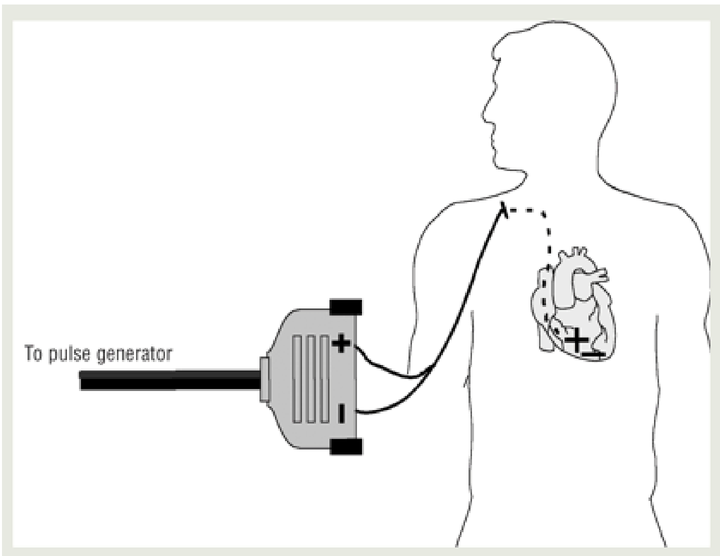
- There must be conductive tissue such as muscle between the two poles to allow for completion of an electrical circuit. If the circuit cannot be completed then depolarisation will not occur. Interruptions to the circuit can come about if a wire comes loose or if the tissue between the poles becomes non-viable due to ischaemia or infarction.

Epicardial pacing is most commonly seen in the patient under going cardiac surgery and provides heart rate support during the postoperative period. The tips of the lead wires are loosely attached to the epicardium and are threaded through the chest wall so they can be attached to an external generator.



Epicardial pacing

Transvenous pacing involves the insertion of a pacing wire through a central vein – most commonly the internal jugular, subclavian, antecubital or femoral vein – under fluoroscopy or with a flotation-pacing catheter with guidance from an ECG. The wire enters the Epicardial surface of the right atrium or ventricle.



Transvenous pacing

Transoesphageal pacing is rarely used and involves the patient swallowing a pill electrode or an electrode is positioned into the oesophagus adjacent to the atrium.

Transcutaneous cardiac pacing is also known as TCP, non-invasive cardiac pacing, external cardiac pacing, precardial cardiac pacing, temporary pacing and transthoracic pacing.
Transcutaneous cardiac pacing can be quickly established, initiated by appropriately trained staff, be effective in up to 70% of patients and has its best use in an acute emergency – when pacing of short duration is required.

This is the oldest form of temporary pacing and was first introduced by Dr Mark C. Lidwill in the 1920s and was further developed by Dr Paul Zoll in 1952.

Transcutaneous pacing is indicated for:

- Significant bradycardias unresponsive to Atropine or when Atropine is not immediately available
- Asystolic cardiac arrest - <10minutes in duration
- Witnessed asystolic arrest
- Provide over-ride pacing in patients with SVT and VT's that are resistant to pharmacologic therapy or electrical cardioversion.

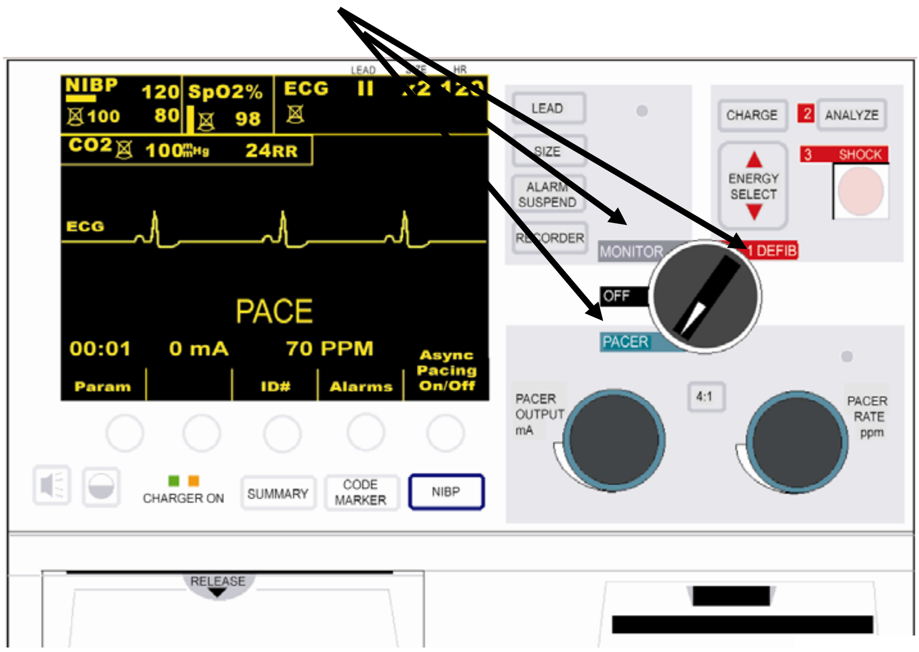
Contraindications

- Misinterpretation of fine ventricular fibrillation
- Severe hypothermia
- Asystolic cardiac arrest - >10minutes in duration

Equipment

- Transcutaneous pacemaker with monitor
- Pacing electrodes and cable
- Electrocardiogram electrodes and cable
- ALS equipment
- Sedatives or analgesics as indicated

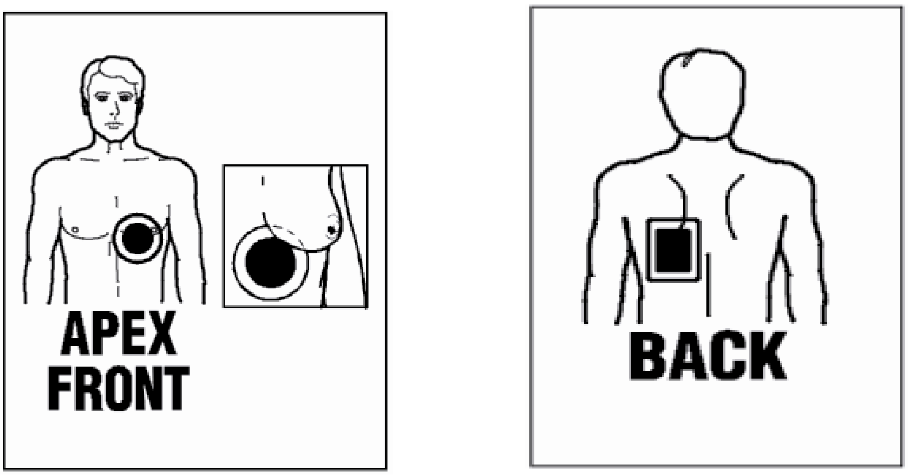
Transcutaneous pacemaker with monitor
Most defibrillators have the ability to act as cardiac pacemakers and monitors – and are easily interchangeable



Pacing electrodes and cable

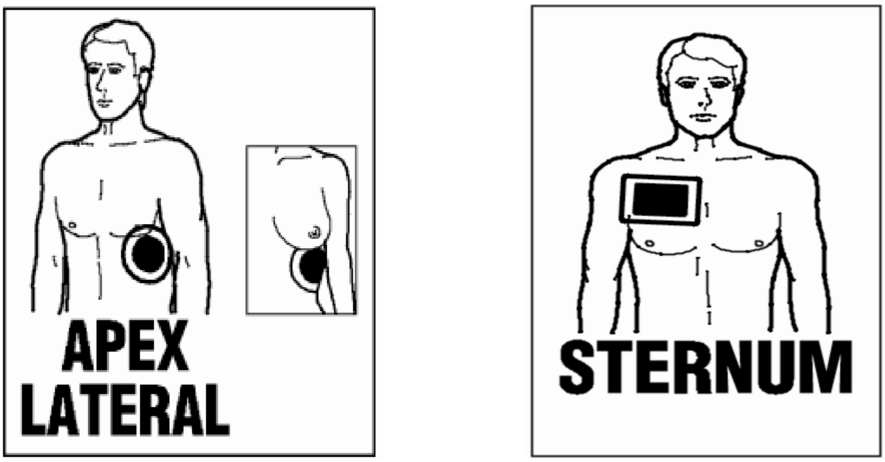
The transcutaneous cardiac pacing system uses two large low-impedance surface electrodes placed on the chest – anteriorly over the cardiac apex at V_3 and posteriorly level with the inferior aspect of the left scapula – figure 1.

Figure 1



NB: If the anterior-posterior electrode position is contraindicated, (e.g. due to trauma) the anterior-lateral position may be used as shown in figure 2.

Figure 2



Ensure the surface electrodes are compatible with the pacemaker being used and are suitable for pacing

NB: not all pads are suitable for pacing – check the manufactures guidelines

Electrodes must never be cut / trimmed. The manufacturer's guidelines regarding the length of time the pacing electrodes may be used for continuous pacing must be checked prior to use.

Always check the 'use by date' of the pads prior to applying.

Once the chest has been exposed the skin should be inspected to ensure it is dry, free of any transdermal patches, ECG electrodes, excessive hair (this should be clipped), etc. The sealed electrode packet should be opened and the pads peeled back – then rolled onto the skin and firmly pressed down all over to secure the seal to the skin – figure 3. The cable must then be plugged into the external pacemaker – figure 4.

Figure 3

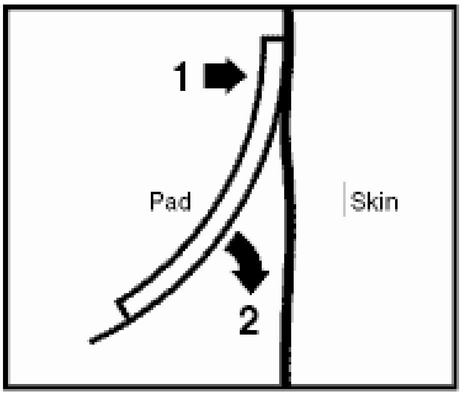
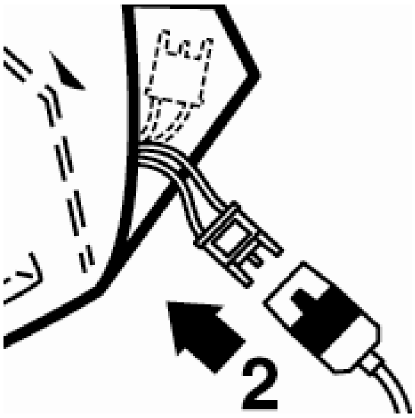
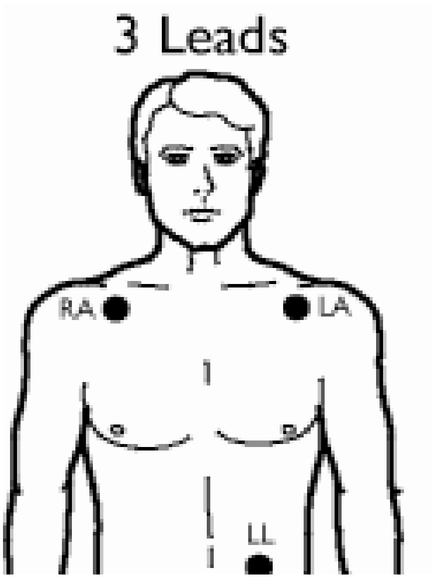


Figure 4



Electrocardiogram electrodes and cable
The 3-lead ECG electrodes must also be placed on the chest – but must not impede the pacing pads – figure 5.

Figure 5



ALS equipment
The resuscitation trolley / equipment should be readily checked and be accessible prior to commencement of TCP. An ALS trained Practitioner should attend the patient throughout.
Standard vital sign monitoring equipment should be used throughout the procedure.

Patient preparation

- As part of the patients preparation a simple explanation of the procedure should be provided and consent sought where necessary e.g. the conscious patient.
- The patient should be made aware of the possible sensations, discomfort or pain they may experience during the procedure.
- Sedative or analgesic options should be discussed, prescribed and administered prior to commencing the procedure.
- The effectiveness of any pre-medication should be monitored during and after the procedure.
- Baseline vital signs should be recorded including:
  - Continuous cardiac monitoring
  - Blood pressure
  - Pulse rate
  - Respiratory rate
  - SPO2
  - Pain score
  - GCS

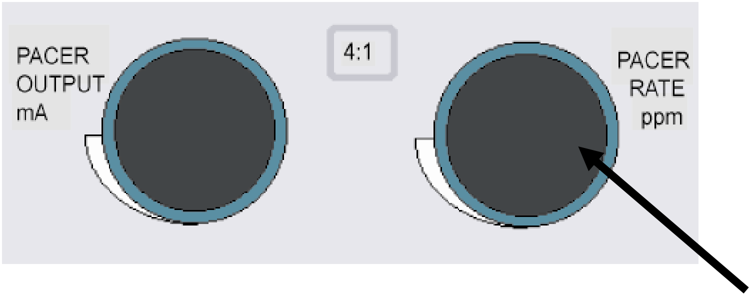
Procedural steps

Once the patient has been prepared as outlined above a baseline 12-lead ECG and rhythm strip must be obtained in order to verify the rhythm present is paceable.

The term *Stimulation Threshold* is used to describe the least amount of energy required to initiate a cardiac depolarisation and subsequent cardiac contraction. During pacing this stimulation and subsequent cardiac contraction is known as *capture*.

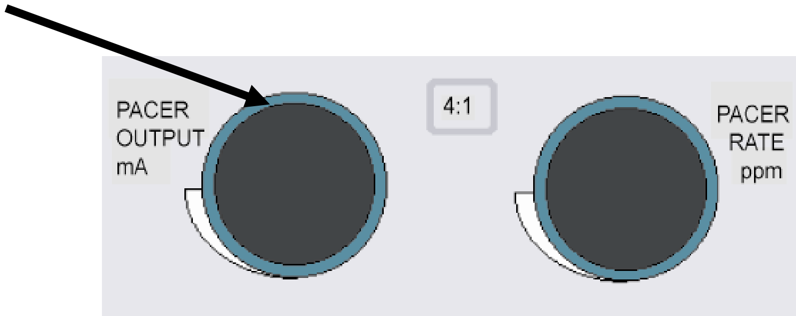
The external pacemaker should be turned ON and the PACER option selected:

Pacer markers (ppm) indicate the rate set to attempt to achieve capture. Select the appropriate rate for external pacing – this is usually 10-20ppm higher than the patient's intrinsic rate. If no intrinsic rate exists use 100ppm. This type of pacing is known as *Demand Pacing* and is the most frequent form of ventricular pacing.



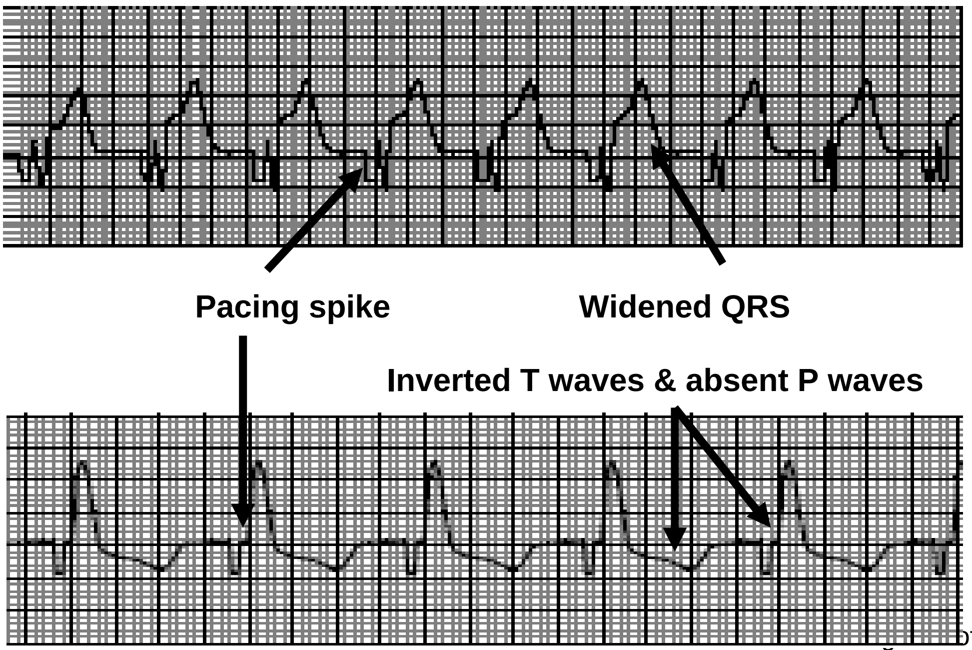
To increase or decrease pacer markers (PPM) turn the Pacer Rate dial – this will alter the rate in 2ppm increments.

In order to achieve capture the pacer output needs to be increased from its default setting of 0mA (Milliamps (mA) are the type of current which are utilised in this mode).



To increase the pacer output turn the relevant dial – this will alter the rate in 2mA increments. The pacer output should be increased until the pacer spike is seen on the monitor and electrical capture is detected.
Electrical capture is determined by the presence of a pacing spike followed by a widened QRS complex, the loss of any under lying intrinsic rhythm and the appearance of an extended and sometimes enlarged T-wave. It can also be verified by palpation of the peripheral pulse (**NB**: avoid using the carotid pulse as electrical stimulation may cause muscle contractions that maybe confused with carotid pulsations).

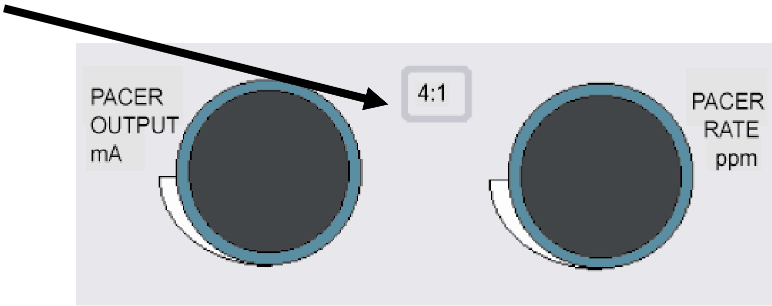
Effective pacing



Once capture has been confirmed, dial the pacer output up by 2mA or 10% higher from capture threshold as a safety margin.

NB: When a patient is in asystole the pacer output should be commenced at full output and if capture occurs should be slowly decreased until capture is lost (threshold) – then add 2mA or 10% higher than the threshold as a safety margin.

The 4:1 button may be pushed at anytime to pause the viewing of the mA on the screen for 4 beats. This allows for reassessment of the underlying rhythm



Stand-by Pacing is achieved by setting the PPM and Pacer output at a back-up rate less than a patient's intrinsic heart rate. The PPM will initially be set above the patient's heart rate and pacer output (mA) is increased to achieve 100% capture. The PPM is then decreased to desired rate below the patient's intrinsic heart rate. Should the HR drop, the stand-by pacer will initiate impulses and begin to pace.

Further nursing care

- Throughout the pacing procedure and for a minimum of 1hour post procedure the patient's vital signs must be monitored to assess the effectiveness of the pacing. This should include:
  - Continuous cardiac monitoring
  - Blood pressure
  - Pulse rate
  - Respiratory rate
  - SPO2
  - Pain score
  - GCS

- Any deterioration in the patient's condition should be reported and acted upon immediately.

- Continue to assess the patients comfort and pain levels and administer appropriate medications as indicated.

- Maintain electrical safety throughout the procedure.

NB: Shock hazard does exist if the patient is touched during the procedure and the pacing energy can be felt.

- Thorough documentation must be maintained throughout and after the procedure including indications, interventions and outcomes for the patient – including a post procedure ECG.

Possible complications

- Skin and muscular pain due to electrical stimulation
- Failure to recognise that the pacemaker is not capturing
- Failure to recognise the presence of underlying treatable VF
- Tissue damage including burns
- Pacing threshold changes => capture failure

A

Questions:

1. Briefly outline which sections of the heart are supplied by the following vessels:

- Right Coronary Artery (RCA)

- Left Anterior Descending (LAD)

- Left Circumflex Artery (LCX)

- Coronary veins

2. Briefly outline the functions of the conduction system structures:

- Sinoatrial (SA) node

- Atrioventricular (AV) node

- Bundle of His

- Right and left bundle branches

- Purkinje system

3. Briefly outline the events of the cardiac cycle:

- Atrial contraction;

- Isovolumic Ventricular Contraction;

- Rapid Ventricular Ejection;

- Isovolumic Ventricular Relaxation;

- Rapid Ventricular Filling;

4. What are the most common reasons for using temporary pacing?

5. Via which routes can temporary pacing be used?

1.
2.
3.
4.

6. List the indications for Transcutaneous pacing:

1.
2.
3.
4.

7. Describe where pacing electrodes/pads should be placed:

8. Describe how the electrodes should be placed onto the skin and what preparation and precautions should taken:

9. List the steps of patient preparation:

10. Which vital signs should be recorded prior to, during, and post procedure?

11. If a patient intrinsic rate is 20bpm what should the pacing rate be set to?

12. If a patient has no intrinsic rate what should the pacing rate be set to?

13. How is electrical capture determined?

14. What is the purpose of the 4:1 button?

15. What possible complications of this procedure could occur?

References:

Aehlert, B. 2002 ECGs made easy. 2nd edn. Mosby, Sydney. pp117-130.

Beverage, D., Haworth, K., Labus, D., Mayer.B.H. and Munson, C. (eds) 2005. ECG Interpretation Made Incredibly Easy. 3rd ed. Lippincott Williams and Wilkins, Sydney.

Biloota, K., Cohn, S., Harrington, S., Terry, D.P., and Wingood, P. (eds) 2005 Cardiovascular Care Made Incredibly Easy. Lippincott Williams and Wilkins. Sydney.

Craig, K. 2006 How to provide transcutaneous pacing. *Cardiac Insider Spring 2006*, pp22-23.

Curtis, K., Ramsden, C. and Friendship, J. 2007 Emergency and trauma nursing. Mosby Elsevier, Sydney. pp181-197.

Herbst, M. C. and Strang, V. 2004 Temporary pacing, cardioversion and defibrillation. In Davies, L. (ed) *Cardiovascular nursing secrets*. Mosby Elsevier, USA.

McMahon, M.D., 2009 Transcutaneous cardiac pacing. In Proehl, J.A. (ed) *Emergency nursing procedures.* Saunders Elsevier, USA.

Overbay, D. and Criddle, L. 2004 Mastering temporary invasive cardiac pacing. American Association of Critical-Care Nurses. <Accessed July 2011 http://ccn.aacnjournals.org/content/24/3/25.full > 24: pp25-32

Way, P. and Winskill, R. 2007 Basic Rhythm Interpretation Learning Package. HNEHS. Australia.

Way, P. and Winskill, R. 2007 Cardiac Physiology Learning Package. HNEHS. Australia.

Zoll M-Series Biphasic 2007 Non-invasive temporary pacing – section 8.