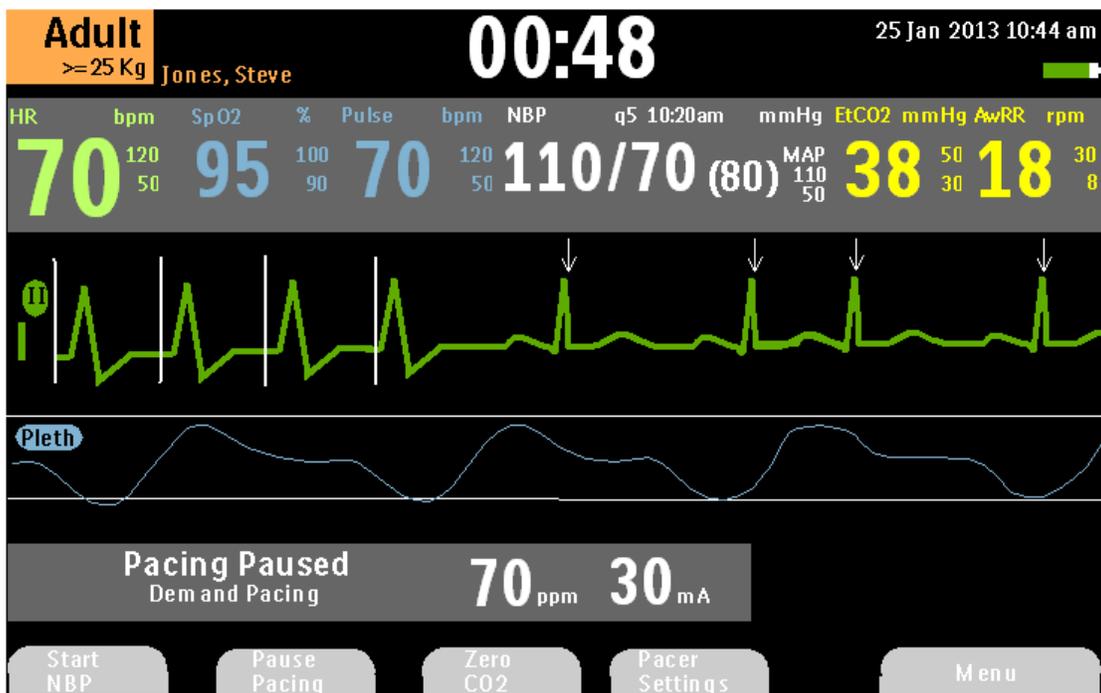# Non-Invasive Transcutaneous Pacing

Application Note

Introduction

This Application Note provides an overview of non-invasive transcutaneous pacing. It includes a brief history of pacing, describes the electrical activity of the heart, and explains how pacing works.

PHILIPS

# What is Non-Invasive Transcutaneous Pacing?

Non-invasive transcutaneous pacing is a technique of electrically stimulating the heart externally through a set of electrode pads. The stimulus is intended to cause cardiac depolarization and myocardial contraction. Pacing is one method of treating patients when their heart's own conduction system slows dangerously.

Electrical Activity of the Heart

The unique physiologic characteristics of cardiac muscle cells produce intrinsic pacing and efficient conduction, leading to optimal synchrony and contractility (see Figure 1).

Figure 1   **The Cardiac Cycle** [1]
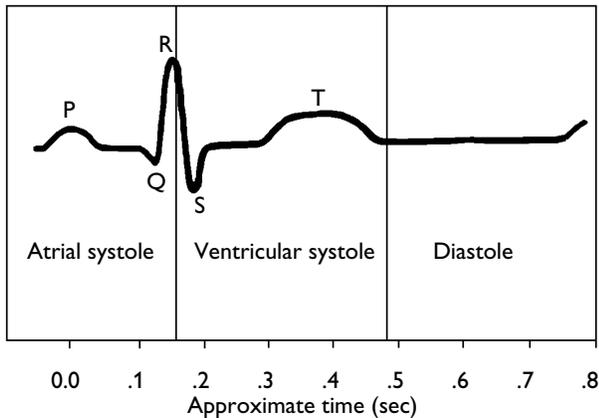
The myocardial cell membrane, like the membranes of other muscle cells in the body, has the ability to conduct a propagated action potential or depolarization wave. Likewise, depolarization of the myocardial cell membrane by a propagated action potential results in myocardial contraction. The propagation of an action potential can be initiated by intrinsic automaticity, direct electrical stimulation (i.e. transvenous or transthoracic pacing), or by a transmitted external stimulus.

The cardiac cell membrane does not require an external stimulus to reach threshold. It possesses a special ability that allows it to spontaneously depolarize at periodic intervals without the need for an external stimulus. This property is called automaticity.

In order for the heart to beat with a smooth and efficient pumping action, the various parts of the myocardium must contract in a well-defined sequence. The atria must contract before the ventricles, the ventricles must begin contracting near the apex, etc. An orderly sequence of contraction during systole is provided for by specialized conductive pathways in the myocardium.

These pathways consist of highly specialized myocardial cells which have the ability to conduct a depolarization wave at a much greater velocity than ordinary myocardial cells.

The normal sequence of myocardial depolarization and contraction, is described below:

1. A cluster of highly conductive cells called the sino-atrial (SA) node is located near the back wall of the right atrium (see Figure 2). It is at the SA node that the depolarization wave is normally initiated.

Figure 2   **Normal Myocardial Sequence**
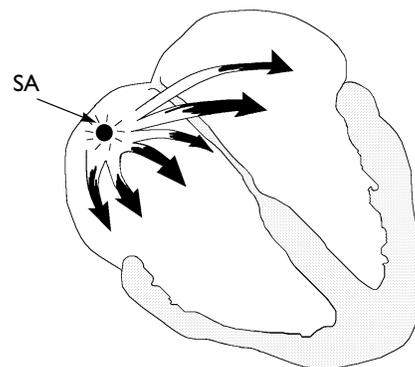
2. From the SA node, the depolarization wave passes from right to left over both atria, resulting in atrial systole. Within 70 ms, all portions of both atria have started to contract.

3. The atrio-ventricular (AV) node consists of a cluster of cells leading from the lower portion of the right atrium to the ventricular septum (see Figure 3). The AV node is highly specialized and conducts the depolarization wave very slowly. It delays its progress for approximately 70 ms, which allows atrial systole to reach completion before ventricular systole begins.

Figure 3   **AV Node**
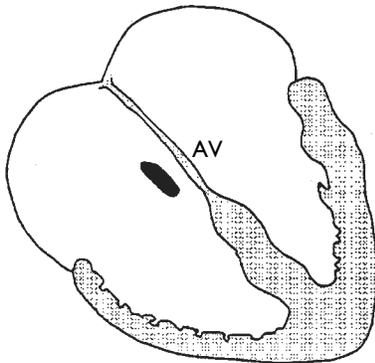
4. From the AV node, the depolarization wave moves to the Bundle of His and its bundle branches. These lie in the ventricular septum and conduct the depolarization wave to the apex of the heart.

5. From the bundle branches, the depolarization wave rapidly travels through the Purkinje network, a fine mesh of highly conductive fibers that cover the endocardial (inner) surfaces of both ventricles.

6. At this point, depolarization and contraction of the ventricular myocardium begin. From the Purkinje network, the depolarization wave moves through the ventricular myocardium from the endocardial (inner) to the epicardial (outer) surfaces. Because of its course of travel through the bundle branches and Purkinje network, myocardial contraction begins in the ventricular septum, then moves rapidly to the apex of the heart and finally proceeds towards the base.

7. As soon as the cells in the myocardium have depolarized, they begin entering the refractory state and repolarizing. They rest until the next stimulus, which under normal conditions, is supplied by the cells in the SA node.

Because our bodies require more oxygen during periods of physical and emotional stress, the SA node is liberally supplied with nerve endings which can stimulate its cells more rapidly when necessary. Hence, during periods of excitement, the SA node responds with a faster rate of depolarization and the heart rate increases to ensure the muscles have enough oxygen to cope with the sudden demand.

The properly functioning Bundle of His and Purkinje network ensure that the heart always contracts rhythmically, and therefore pumps efficiently. The fact that all conductive cells in the heart possess the capability of spontaneous depolarization provides a natural back-up pacemaking capability, in case the SA node should fail.

The progress of the depolarization wave causing the muscle contraction can be followed and recorded. The recorded ECG waveforms and their correlation to the activity of the heart are illustrated in Figures 1 through 7.
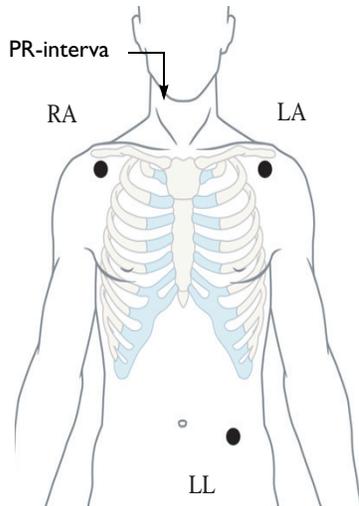
The depolarization of the atria is represented by the P-wave (Figure 4). During this time, the atria contract and force blood into the ventricles. At the point labeled "Q", the ventricles begin depolarizing and contracting.

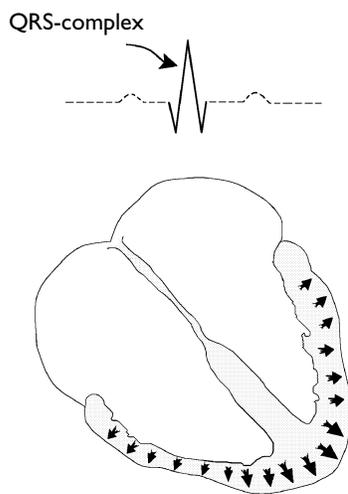Figure 4   **Atrial Depolarization**
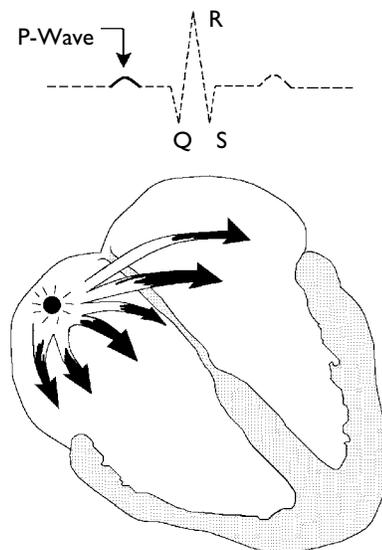
Notice the time delay between the P-wave and Q. This is the 70 ms delay introduced by the AV Node (Figure 5).

Figure 5   **AV Node Delay**

The time between "Q" and "S" (called the QRS complex) immediately precedes full contraction of the ventricles when blood is forced into the arterial system (Figure 6).

Figure 6   **Ventricular Depolarization**

The "T" wave represents the refractory period of the heart when the ventricles are repolarizing (Figure 7).

Figure 7   **Ventricular Repolarization**
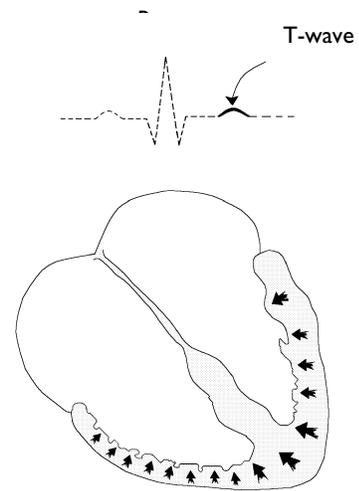
Application of Pacing

Disease in the conduction system or failure in the automaticity of the myocardial cells can lead to symptomatic bradycardia or other arrhythmias which may necessitate pacing intervention.

The following table explains the indications and symptoms of emergency pacing.

Table 1   **Indications of Emergency Pacing and Pacing Readiness [6]**

Immediate Emergent Pacing
Class I
Hemodynamically symptomatic, compromising bradycardias that are too slow and unresponsive to atropine.[a] Symptoms can include blood pressure less than 80 mmHg systolic, change in mental status, angina, pulmonary edema.
Class IIa
Bradycardia with escape rhythms unresponsive to pharmacologic therapy
Pacing for patients in cardiac arrest with profound bradycardia or PEA[b] due to drug overdose, acidosis, or electrolyte abnormalities
Standby pacing: prepare for pacing for specific AMI-associated[b] rhythms: • Symptomatic sinus node dysfunction • Mobitz type II second-degree heart block • Third degree heart block [c] • Newly acquired left, right, or alternating BBB[b] or bifascicular block
Class IIb
Override pacing of either supraventricular or ventricular tachycardia that is refractory to pharmacologic therapy or electrical cardioversion
Bradyasystolic cardiac arrest

a. Including complete heart block, symptomatic second-degree heart block, symptomatic sick sinus syndrome, drug-induced bradycardias (i.e., amiodarone, digoxin, β-blockers, calcium channel blockers, procainamide), permanent pacemaker failure, idioventricular bradycardias, symptomatic atrial fibrillation with slow ventricular response, refractory bradycardia during resuscitation of hypovolemic shock, and bradyarrhythmias with malignant ventricular escape mechanisms.

b. PEA indicates pulseless electrical activity; AMI, acute myocardail infarction; and BBB, bundle branch block.

c. Relatively asymptomatic second- or third-degree heart block can occur in patients with an inferior myocardial infarction. In such patients pacing should be based on symptoms of deteriorating bradycardia.

Fixed Rate or Demand Pacing—Which One To Use?

Many non-invasive transcutaneous pacemakers operate in two modes: fixed rate (also referred to as asynchronous or non-demand) and demand mode (also referred to as synchronous).

Fixed Rate Mode Pacing

In the fixed rate mode, the pace rate is set by the clinician regardless of the patient's intrinsic heart rate. This option is preferable when the ECG signal becomes extremely noisy due to motion artifact or when the pacemaker is sometimes unable to sense the intrinsic beat.

Another reason for using the fixed rate mode is to terminate tachyarrhythmias by overdriving the patient's intrinsic beat. This method has been successful in a limited number of patients[7,8,9]. The above methods are not widely used in a clinical setting. The danger with fixed rate mode pacing is the possibility of further exacerbating the tachyarrhythmia and triggering ventricular fibrillation[9]. Fixed rate mode does not sense the QRS complex for the R-wave and may pace on the T-wave, which could trigger ventricular fibrillation.

Demand Mode Pacing

In demand mode pacing, the pacer senses the patient's intrinsic heart rate and will pace if the intrinsic signal is slower than the rate programmed by the clinician.

For example, if the patient's heart rate becomes slower than the prescribed setting, the pacer will send an electrical stimulus. If the pacer senses that the heart rate is faster than the pacing rate, it inhibits an electrical signal. The advantages of demand mode pacing are: competition between the pacemaker stimuli and the intrinsic heart rate is minimized, decreasing the risk of R on T, and the number of pace pulses introduced are minimized reducing patient discomfort. For this reason, demand mode pacing is primarily used as the default setting.

During demand mode pacing, Philips' defibrillators detect R waves, or beats. Intrinsic beats are defined as those that are generated naturally by the patient. Paced (or captured) beats are defined as those that are a result of delivered pacing energy.

Philips' ALS defibrillators also define the paced refractory period, which is simply a period of time after the delivery of a pace pulse. The refractory period is approximately

340 ms for pacing rates less than or equal to 80 pulses per minute (PPM), and approximately 240 ms for pacing rates greater than or equal to 90 PPM.

With the pacer on, the defibrillator marks intrinsic beats on the R-wave with an arrow on the Efficia DFM100. The intrinsic beats are also marked in the print strip. When an intrinsic beat is detected, the time interval for the next pace pulse starts at the intrinsic beat. If no intrinsic beat is detected, the time interval for the next pace pulse starts at the last pace pulse.

Philips' defibrillators use the ST/AR Basic Arrhythmia Algorithm for arrhythmia analysis. This analysis provides information on your patient's condition, including heart rate and arrhythmia alarms. The lead appearing in Wave Sector 1 is used for arrhythmia analysis. Because the ST/AR Basic Arrhythmia Algorithm is the source needed to generate heart rate and heart rate alarms, the algorithm cannot be disabled. However, if desired, arrhythmia and heart rate alarms can be turned off. During arrhythmia analysis, the ECG monitoring parameter continuously:

- Optimize ECG signal quality to facilitate arrhythmia analysis. The ECG signal is continuously filtered to remove baseline wander, muscle artifact, and signal irregularities.

- Measure signal features such as R-wave height, width, and timing.

- Create beat templates and classify beats to aid in rhythm analysis and alarm detection.

- Examine the ECG signal for ventricular arrhythmias and asystole.

It is impossible to design a computerized arrhythmia algorithm that accurately identifies R-waves 100% of the time. Several conditions can cause difficulty for the algorithm to interpret R-waves, such as tall P-waves, tall T-waves, aberrantly conducted beats, and P-waves without a conducted R-wave that follows. In most instances, tall T-waves and P-waves can be addressed by selecting different leads to minimize the P-wave or T-wave amplitudes relative to the R-wave amplitude.

You may choose to display an annotated ECG with ST/AR arrhythmia beat labels in Wave Sector 2. The same ECG source appearing in Wave Sector 1 may be displayed with a six-second delay along with white arrhythmia beat labels. The R-waves marked in Wave Sector 1 are annotating the intrinsic beats that control the demand pacing function. The R-waves marked with beat labels in Wave Sector 2 control the analysis and arrhythmia alarms.

Clinicians must not rely solely on the defibrillator's classification of beats as intrinsic or paced to determine electrical capture. Consider the situation where the patient's intrinsic HR is 62, and the pacer is set at a rate of 60. Since the two rates are very close, pacer spikes and intrinsic beats may occur very close to each other for several seconds. In this circumstance, the defibrillator may think the beats are paced based on its simple timing algorithm, but in fact the beats are intrinsic and the timing coincidental. There may also be cardiac conditions which can cause a truly paced beat to fall outside of the refractory period.

Capture must be determined by a clinician who is trained to interpret the ECG being displayed by the defibrillator. The defibrillator's marking of beats can be used as a guide, but not as an absolute indication, of whether or not capture has been achieved.

How to Implement Non-Invasive Pacing

Pads Application and Placement

Before applying the pads, clean and dry skin sites with a towel. It may be necessary to shave or clip excessive hair in the area of the pads. Follow the manufacturer's suggested placement of electrode pads. Do not reverse the pads since reversing electrode pad placement increases the pacing threshold. Thus, more current will be needed to capture the heart resulting in greater patient discomfort[10]. The most common electrode placement is the anterior-anterior position (Figure 8).

Figure 8   **Anterior-Anterior Position**
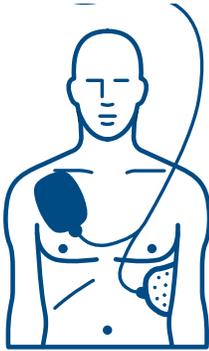
The anterior-posterior position is also an effective placement (Figure 9).

Figure 9   **Anterior-Posterior Position**
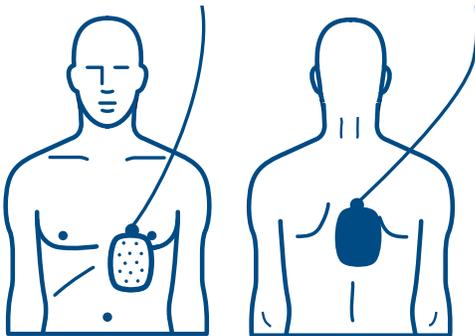
Monitoring Electrodes

When performing demand mode pacing, the patient must be monitored through either 3- or 5-lead monitoring electrodes. Philips' ALS defibrillator/ monitors use the heart rate from this monitoring source to determine if a paced pulse should be delivered. Monitoring electrodes are not required for fixed mode pacing.

Setting Pacing Rate and Pacing Output (mA)

Setting the Default Configuration

The Efficia DFM100 allows you to customize the defibrillator to best meet your needs with configurable default pacer rate and output settings. Configuration is performed through Configuration mode of the device and may be saved to a data card for replication on multiple devices. At any time, configuration settings may be viewed and exported to a data card, and the date and time may be changed. However, a password is required to change the configuration of the device. When modifying configuration settings, the device should be connected to external power and have a battery with at least 20% capacity installed.

Changing Rate and Output (mA) During an Event

The pacer rate can be adjusted during pacing from the default configuration by pressing the [**Pacer Rate**] soft key on the device and then the navigational keys to select the desired number of pace pulses per minute. The pacer output can also be adjusted during pacing from the default configuration by pressing the [**Pacer Output**] soft key and then the navigational keys to:

a.   Increase the output until electrical capture occurs. Electrical capture is indicated by the appearance of a QRS complex after each pacing marker.

b.   Decrease the output to the lowest level that still maintains capture.

Presence of a peripheral pulse must be verified to assess mechanical capture.

During pacing, verify that arrows appear above the R-wave on the ECG waveform. A single arrow should be associated with each R-wave. If the arrows do not appear or do not coincide with the R-wave, select another lead. Press the Lead Select button to select the best lead with an easily detectable R-wave. If you are using anterior-anterior pads placement while pacing and are experiencing difficulty with Lead II, select another lead. If a lead cannot be found with correct markers or arrows above the R-waves, then switch to fixed mode pacing.

Changing to Fixed Mode Pacing During an Event

With the Efficia DFM100, you can switch to fixed mode pacing during an event, as follows:

1.   Press the [**Pause Pacing**] soft key and acknowledge the message prompt.

2.   Press the Smart Select knob and select **Pacer Mode**.

3.   Use the Smart Select knob to highlight **Fixed** and press the Menu Select button.

4.   Press the [**Resume Pacing**] soft key to resume pacing in fixed mode.

What to Look for When Pacing

Cardiac Capture

Capture is defined as gaining control of the patient's dysfunctional heart by an electrical stimulus (that is, depolarization). Electrical capture can be determined by observing the monitor, which should show a clear indication of the ECG and the pulse marker. Skeletal muscle contraction is not an indication that capture has been established, nor is electrical capture alone an indication of effective cardiac perfusion. The patient may be suffering from pulseless electrical activity (PEA), previously referred to as electromechanical dissociation (EMD). When the patient is successfully paced:

- electrical and mechanical capture are achieved

- cardiac output may improve

- the patient's pulse rate is at least equal to the pacer rate

- blood pressure may improve

- skin color may improve

A pulse oximeter is a powerful tool when used in conjunction with the pacer/ECG monitor for confirming capture because both mechanical and electrical activities can be measured. ECRI provided a theoretical and an operational overview of non-invasive pacemakers and concluded that monitoring ECG alone is not enough to verify that the patient's heart is providing cardiac output.

The article states that:

- Clear recognition of capture continues to be a challenge with today's non-invasive pacemakers.

- Artifact from skeletal muscle contractions induced by pacing stimuli can mimic a captured waveform, making verification of capture with the ECG alone unreliable.

- Electrical capture does not always produce an effective mechanical (cardiac) contraction.

Therefore, a patient's response to pacing must be verified by signs of improved cardiac output, such as: a palpable pulse rate the same as the rate which pace pulses are being delivered, a rise in blood pressure, and/or improved skin color.

An integral or standalone pulse oximeter can be useful for confirming capture (by comparing the pulse rate measured by the pulse oximeter to the set pacing rate) and perfusion (by measuring blood oxygen saturation, SpO_2)[11]. Philips' defibrillator/monitors integrate the optional SpO_2 package into the defibrillator for speed, simplicity, and convenience. The SpO_2 percentage (%) and SpO_2 alarm violations will be recorded in the Event Summary.

Another important factor in effective pacing is the amount of current applied. Sufficient current must be applied to stimulate the heart into contractions. Unlike invasive pacemakers, non-invasive pacemakers must pass electrical current through the skin and thorax. This requires significantly higher current. The higher current loads can cause the chest muscles to contract and relax strenuously, which may be painful and distressing for the patient.

Using the lowest output setting necessary to achieve capture will minimize the discomfort. If the discomfort is intolerable, sedation and/or analgesia may be necessary.

Patient Discomfort

Pacing will likely cause some discomfort in conscious patients. Two types of discomfort often experienced are a burning sensation at the electrode site, and muscle contraction. Thus, setting the expectations with the patient and family is very important. This should alleviate any anxiety or tension and allow the patient to become more relaxed. Patients should always be comforted throughout the procedure, and sedation may be necessary. Make sure the pads are positioned according to the manufacturer's recommendation. Avoid placing the pads directly over lesions, injuries or large bone structures (sternum, spine, scapula). Otherwise, more pacer output will be needed to achieve capture.

Special Pacing Situations

Pediatric Pacing

If transcutaneous pacing is elected, the physiological differences between adults and pediatric patients must be considered:

Electrode Size per AAMI Specification

- *adult* — total active area of both pads combined should be at least 150 cm^2. Adult pads are recommended for use on children weighing 10 kg or more.
- *pediatric* — total active area of both pads combined should be at least 45 cm^2. Recommended for use on children weighing less than 10kg.

See ANSI/AAMI DF2-1996.

Skin Fragility

Electrodes should be checked frequently (every thirty minutes) to avoid burns and should not be left on for more than two hours.

Higher Heart Rate

- 100 BPM is considered tachycardia in adults. A heart rate less than 100 BPM is considered bradycardia in a neonate.
- Supraventricular tachycardia is faster in children than in adults (the heart rate is typically 300 BPM in infants younger than three months of age). Supraventricular tachycardia is more common than ventricular tachycardia in children.

- Atrial flutter is faster in children than in adults. Newborns may have rates of 600 BPM with distinct P-waves. The usual ventricular rate in a newborn with atrial flutter is 200 to 300 BPM[12].
- Ventricular tachycardia in infants under the age of two years averages 260 BPM, which is similar to the rates for supraventricular tachycardia[13].

Pain Threshold

The discomfort associated with pacing may necessitate sedation in younger conscious children.

Lead Placement

The use of Lead II is not recommended when using anterior-anterior lead placement.

NOTE: Unlike adult pacing, the use of pediatric transcutaneous pacing will likely be limited for cardiopulmonary arrest since most complications are precipitated by respiratory failure rather than by a primary cardiac problem[14].

Defibrillation During Pacing

In defibrillator/pacer combinations, Philips defibrillators will automatically terminate pacing once the defibrillator charge switch is enabled.

Troubleshooting Transcutaneous Pacing

1. Is there discomfort or pain during pacing?

   ▸ Explain and set expectations with the patient on the procedure. Comfort and provide encouragement to the patient.

   ▸ Make sure electrodes have been applied following manufacturer's guidelines. Electrodes should not be placed over bone (i.e. on the sternum or scapula).

   ▸ Consider analgesia or sedation.

2. Is there electrical capture?

   ▸ Pacer output may be too low. Increase pacer output.

   ▸ Observe the ECG monitor and not just the patient. Electrical capture occurs when there is a consistent ST segment and T-wave after each spike.

   ▸ Check electrode placement.

   ▸ Check contact between the electrode and the patient.

   ▸ Check to see if the pacer is turned on.

   ▸ Check viability of the patient.

3. Is there mechanical capture?

   ▸ Check pulse (palpate).

   ▸ Check pulse oximeter (SpO2).

   ▸ Check blood pressure.

   ▸ Check skin color.

   ▸ Evaluate mental status of patient.

4. Is the pacer not sensing heart rate in demand mode?

   ▸ Check size of R-wave. If it is too small, increase the ECG size.

   ▸ Check to see if the pacer is turned on.

   ▸ Check electrode/patient contact.

   ▸ Check electrode connections.

   ▸ ECG signal may be too noisy (see below).

5. Is there a noisy ECG signal?

   ▸ Was the patient's skin clean and dry and excessive hair removed prior to electrode attachment?

   ▸ Was the expiration date checked before applying the electrodes? Dry gel will have an adverse effect on the ECG signal.

   ▸ Check the electrode connections.

   ▸ Were the electrodes positioned according to the manufacturer's guidelines?

6. Is there redness or burns after electrode removal?

   ▸ Ring-shaped erythema can be expected after pacing. The appearance is similar to the erythema seen after receiving shocks from standard external paddle sets. The reaction may vary from patient to patient due to a number of variables: skin composition, medication at the time of pacing, age (very young and older patients may be more prone to erythema), dehydration, length of time the patient was paced, and improper pads application. Case reports of burns on neonates and children have been documented. In most cases, the children had numerous other complications which may have contributed to the burns. Neonates in particular may be more susceptible to thermal injury than adults due to thinner skin, less hair, weaker intercellular attachments, fewer eccrine and sebaceous gland secretions, and an increased susceptibility to external irritants. To minimize thermal injury, pacing duration should be kept to a minimum with frequent skin inspection (every 30 minutes)[15].

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