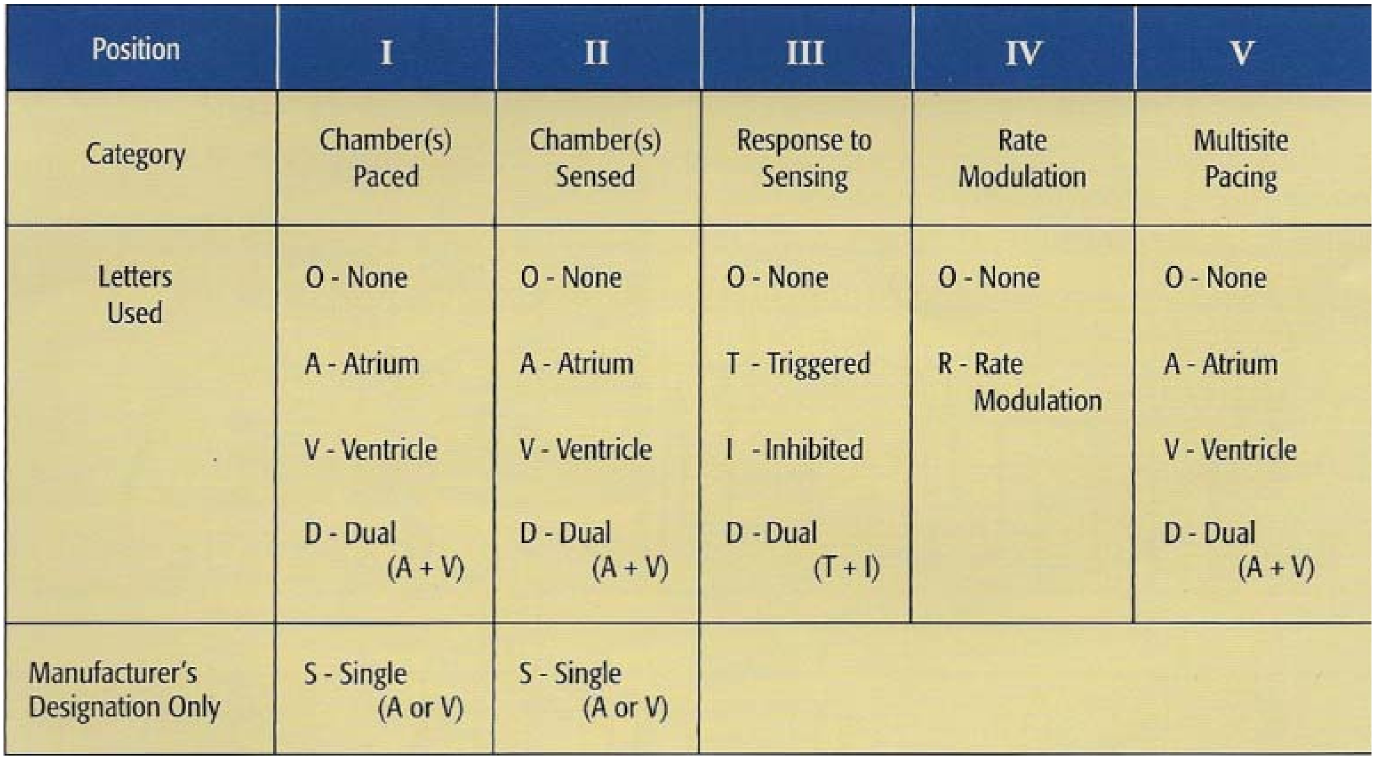
# Basic Concepts of Cardiac Pacing

## The NBG Code

In its simplest form, a pacemaker is a power source with a timing circuit. This unit is attached to a wire, called a lead, designed to carry electrical energy to the heart. This energy is used to pace the hear (cause a contraction) when necessary. Pacemakers today can do what we call "dual-chamber pacing;" that is cause a contraction in either the atrium, the ventricle, or both.

Heart Failure devices can even pace in both ventricles simultaneously.

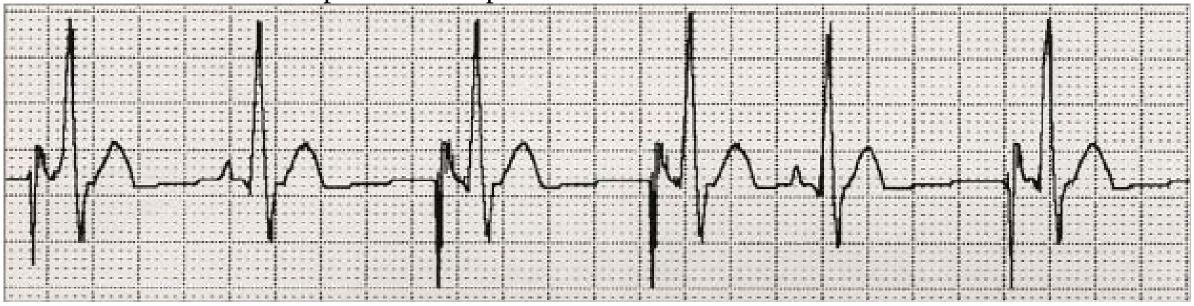
In order to track pacing, sensing and programmability, a three-letter code was first proposed in 1974. This code was later expanded to five letters, and was further revised in 2002. The responsibility for keeping this code up-to-date is given to a committee made of members of the North American Society for Pacing and Electrophysiology (NASPE, now known as Heart Rhythm Society (HRS), and the British Pacing and Electrophysiology Group (BPEG). It has become known as "The NASPE/BPEG Generic Code" or the "NBG Code." This code communicates the mode and functional characteristics of a pacemaker. The following chart illustrates the newest version of the NBG code, which was adopted in 2002.



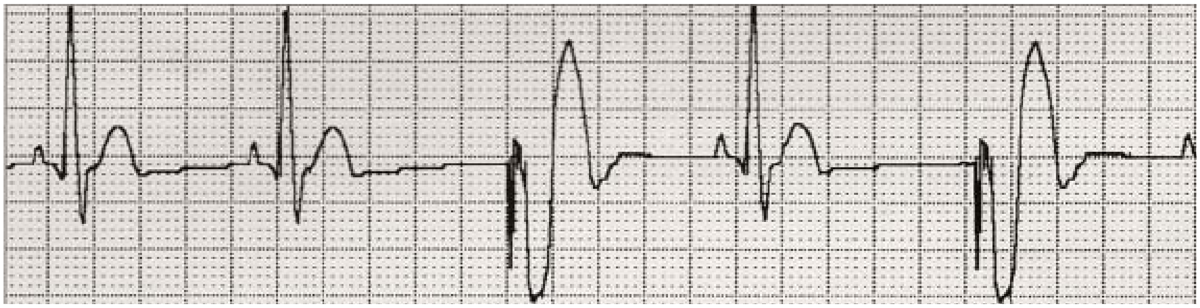
| Position | I | II | III | IV | V |
|---|---|---|---|---|---|
| Category | Chamber(s) Paced | Chamber(s) Sensed | Response to Sensing | Rate Modulation | Multisite Pacing |
| Letters Used | O - None<br>A - Atrium<br>V - Ventricle<br>D - Dual (A + V) | O - None<br>A - Atrium<br>V - Ventricle<br>D - Dual (A + V) | O - None<br>T - Triggered<br>I - Inhibited<br>D - Dual (T + I) | O - None<br>R - Rate Modulation | O - None<br>A - Atrium<br>V - Ventricle<br>D - Dual (A + V) |
| Manufacturer's Designation Only | S - Single (A or V) | S - Single (A or V) | | | |

## NBG Code

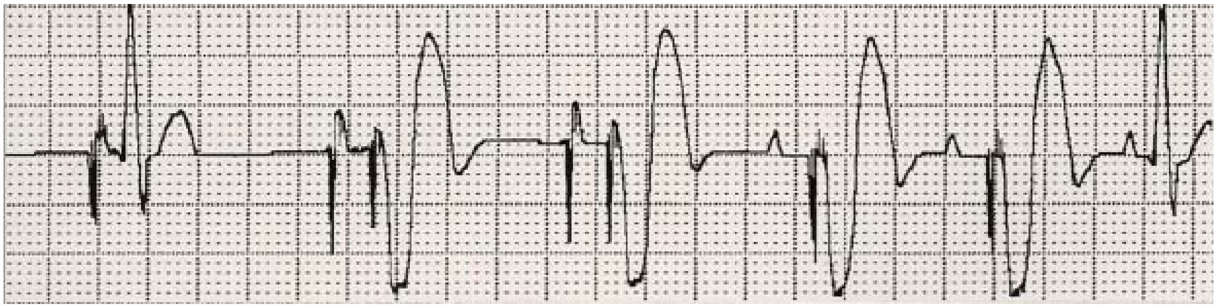
Pacemakers can be programmed to function in ways that ca be described by the NBG code. Here are some examples of common modes of pacemaker operation:



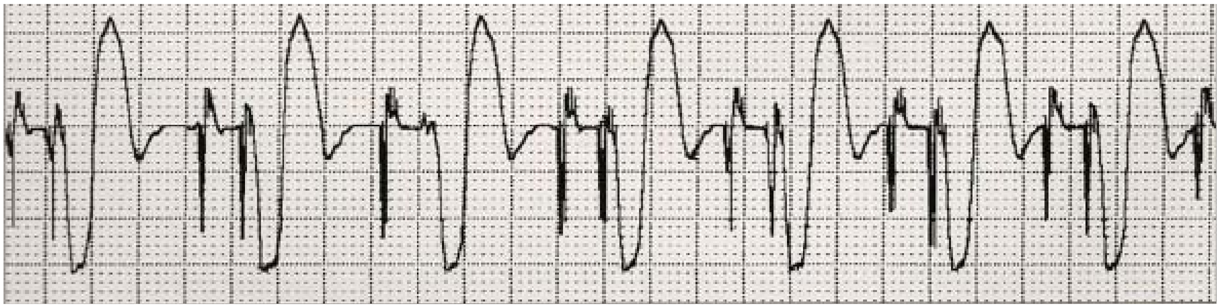
**AAI:** Pace in atrium, sense in atrium, inhibit in atrial activity



**VVI:** Pace in ventricle, sense in ventricle, inhibit in response to ventricular activity



**DDD:** Pace in atrium and ventricle, sense in atrium and ventricle, inhibit in response to atrial or ventricular activity, track atrial activity.



**DDDR:** Dual-chamber pacing with rate response

## Capture and Output

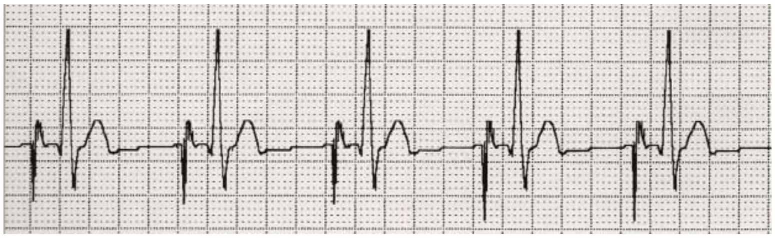
Capture of the heart happens when enough energy is sent down the lead to cause depolarization and contraction. This depolarization can be seen on the ECG. Capture in the atrium will result in a paced P wave on the ECG, and capture in the ventricle will be seen as a QRS complex that can resemble a PVC preceded by a pacing spike.

The goal of the pacemaker is to capture the heart every time it sends energy down the lead. The lead is tested at implant to assure that there is consistent capture with a low enough energy level to ensure that the pacemaker will function properly. Pacemaker **output** is the amount of energy sent out with each pulse. Energy can be described as “voltage over time,” or voltage at a certain pulse width. Voltage is the electrical pressure that causes electrical current to flow, and is measured in volts (V). The pulse width is the length of time that the current is flowing, and is measured in milliseconds (ms). An example is “2.5V at 0.5ms.”

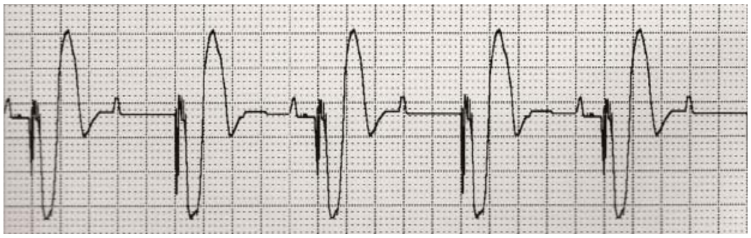
The minimum amount of energy required to capture the heart is known as the capture threshold. Thresholds can be measured by varying the voltage with a constant pulse width or by varying the pulse width while keeping the voltage constant.

Once the threshold is determined, the output is programmed to provide a safety margin of at least 2:1. For example, a patient with a chronic threshold of 1 volt would satisfy the 2:1 safety margin if his device were programmed to 2.0V. Most centers, through, will not program the device lower than 2.5V (This is for devices without AutoCapture pacing systems.)

If using pulse width to determine threshold, the safety margin ratio is 3:1. A patient with a pulse width threshold of 0.2ms should have the final pulse width programmed to 0.6ms to provide adequate safety. Pulse width programming is generally considered less favorable than voltage programming, if both options are available.



**Atrial Capture:** AAI Mode



**Ventricular Capture:** VVI Mode

## Sensitivity

Along with capturing the heart every time, the pacemaker needs to recognize intrinsic events (beats) to prevent it from interfering or competing with the patient's intrinsic rhythm: When the pacemaker senses an intrinsic beat, the pacing energy is withheld (the pacemaker "inhibits") and the clock is reset.

The pacemaker should sense, or see, these intrinsic beats every time they occur. The size of the intrinsic beat is measured at implant, and then again at follow-up. It is important to know how large the intrinsic signal is so that the pacemaker's **sensitivity** can be set appropriately. Sensitivity is the ability of the pacemaker to "see" intrinsic cardiac events such as native P and R waves. Think of the pacemaker's sensitivity as a wall that can be raised and owered.

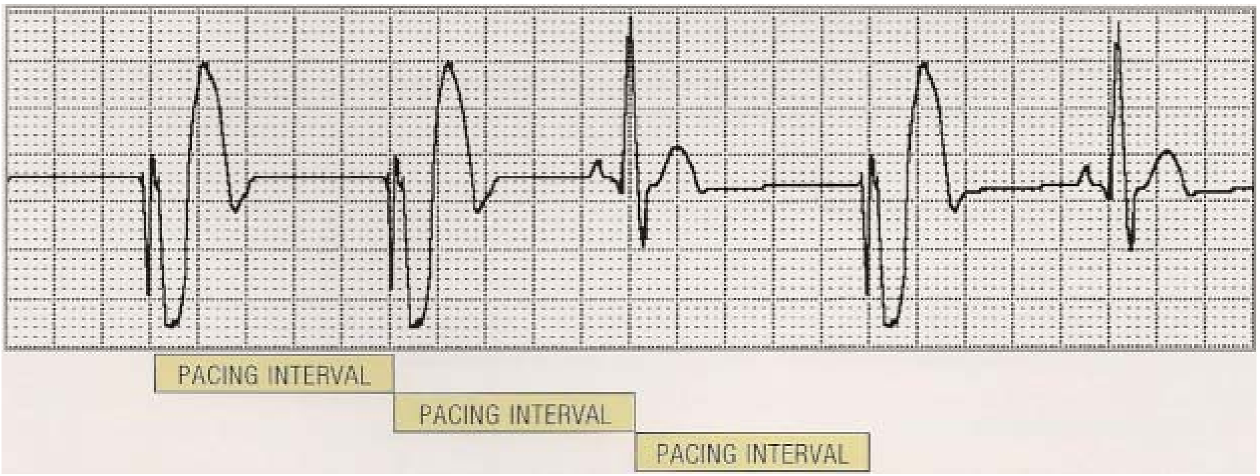
The goal is for the pacemaker's sensing circuit to be able to see the intrinsic signal over the wall.

If the wall is too high, the signal wont' be seen and the pacemaker won't inhibit appropriately, causing competition with the intrinsic rhythm. This is called "undersensing".

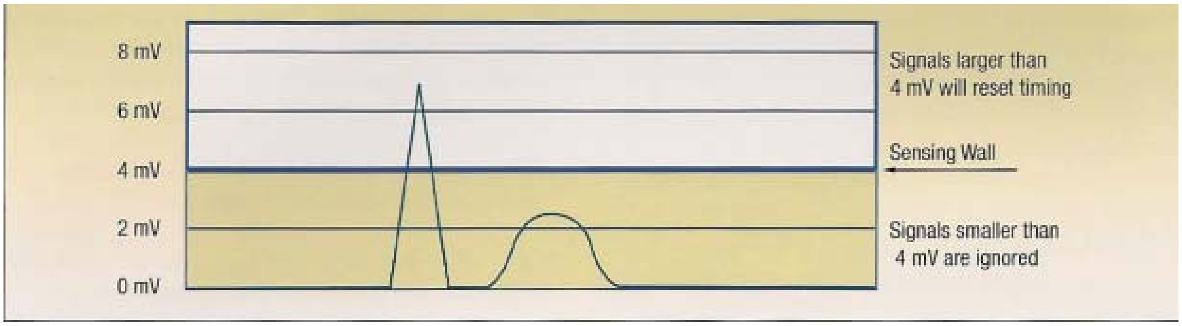
If the signal is small, the wall may need to be lowered so the signal can be seen.

If the wall is too low, however, there is a risk that the pacemaker may see a non-cardiac signal, misinterpreting it as a heartbeat and then inhibiting the pacemaker output. This is called "oversensing" and may result in a pause on the ECG.

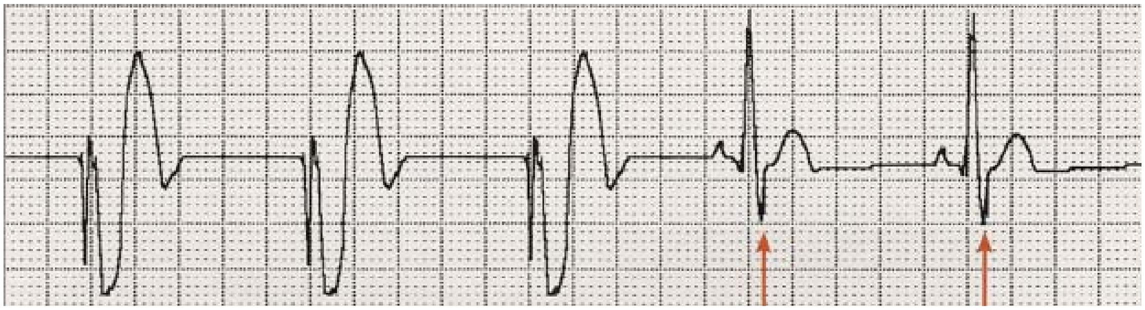
Intrinsic signal are measured in millivolts (mV),
So the pacemaker's sensitivity is set in millivolts as well. The typical nominal value for ventricular sensitivity is 2mV, and the typical value for atrial sensitivity is 0.75mV.



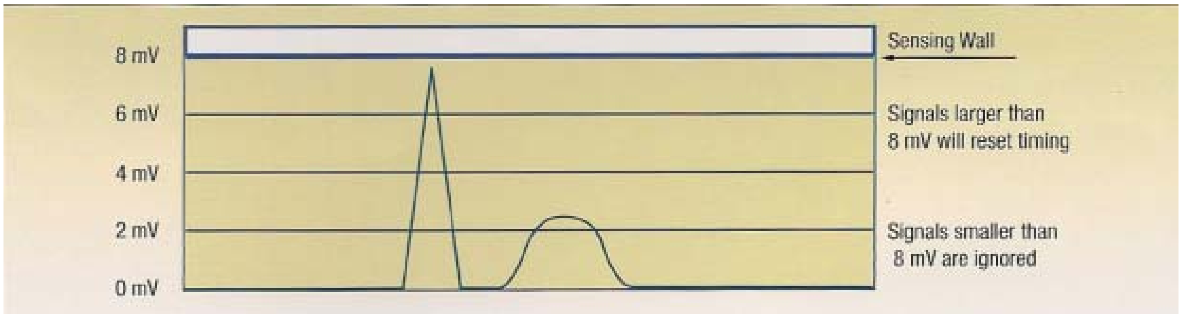
Pacing Interval - VVI Mode



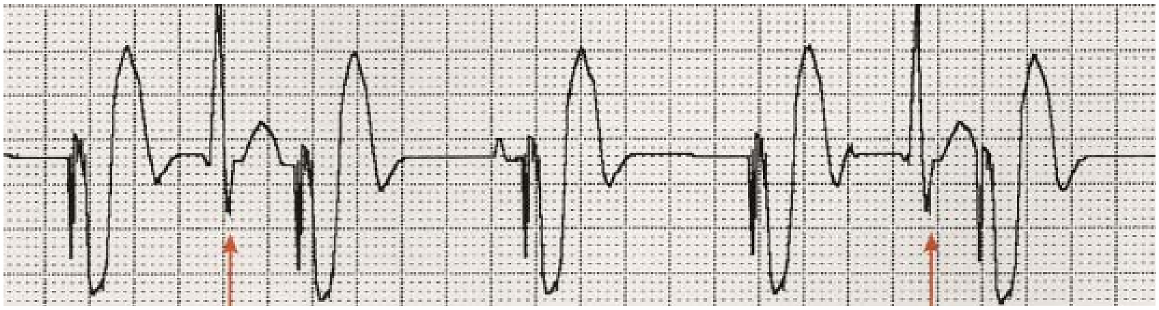
Pacemaker sensitivity programmed to 4mV.



Appropriate pacing and sensing in VVI Mode. Arrows illustrate inhibit pacing and reset timing.



Pacemaker sensitivity programmed to 8mV.



Undersensing in VVI mode. Intrinsic events (arrows) are not sensed by the pacemaker so the interval time is not reset.

# Basic Concepts of Defibrillation

The implantable cardioverter defibrillator (ICD) has had a major impact on the treatment and prevention of SCD for those at risk; ICDs were originally approved only if a patient had survived two SCD episodes when the FDA approved the device in 1985. The highly sophisticated ICD device will sense a tachyarrhythmia in the ventricular, determine if it is atrial or ventricular in origin and deliver therapy of pace termination or high voltage shock.

**History**

Dr. Michel Morowski lost his best friend to arrhythmic death in the 1960s and became obsessed with an idea his colleagues considered a technological impossibility: an implantable defibrillator. Over the years, Morowski and others worked tirelessly on the concept Morowski with tampering with film evidence of how the device worked in dogs. In 1980, the first device for human implant was successfully implanted in a 57-year-old patient. In 1982, cardioverter capabilities were added and the device was called the AICD, Automatic Implantable Cardiac Defibrillator.

The FDA approved the AICD in 1985 and programmability was added in 1988. Those early devices were large, had short service lives, and required an abdominal implant and epicardial leads, necessitating a thoracotomy for implant. A wave of technological innovations followed. As the AICD shortened its name to ICD the devices also got much smaller and smarter. Today, an ICD can be implanted pectorally, works with transvenous leads, and may offer device longevity of five years or more.

**Indications**

When the ICD was first approved, a patient had to have survived tow episodes of sudden cardiac death (SCD) to be indicated for device implantation. The purpose of the ICD was to rescue a patient from SCD. Large randomized clinical trials compare the ICD to antiarrhythmic drug therapy and showed that ICDs reduce mortality. More recent randomized clinical trials have demonstrated that ICDs reduce mortality in those at risk fro SCD (such as a patients with a low ejection fraction and previous myocardial infarction), even if they had no history of arrhythmia. A few highlights from some landmark ICD studies follow.

## MADIT Multicenter Automatic Defibrillator Implantation Trial

**Objective:**
Compare ICDs and conventional medical treatment in patients at high risk for SCD who could be induced to ventricular tachyarrhythmias in an EP Lab

**Inclusion:**
Prior MI (>1 month before enrollment), asymptomatic non-sustained VT, EF $\leq$ 35% and a positive EP study

**Exclusion:**
Standard ICD indication, revascularization within past 3 months or planned, NYHA Class IV

**Results:**
ICDs significantly reduced all-cause mortaility by 54% compared to state-of-the-art drug therapy in heart attack survivors with a low ejection fraction (high-risk patients)

***MADIT was the first major trial to show all-cause mortality benefit with ICDs in patients with a prior MI and LV dysfunction, but patients in MADIT had to be inducible in the EP lab to enroll in the study.***

## MADIT II Multicenter Automatic Defibrillator Implantation Trail II

**Objective:**
Compare ICDs and conventional medical treatment in patients at high risk for SCD who had not previously been stratified by an EP study

**Inclusion:**
Prior MI (>1 month before enrollment), EF $\leq$ 30%.

**Exclusion:**
Standard ICD indication, revascularization within past 3 months or planned, NYHA Class IV

**Results:**
ICDs significantly reduced all-cause mortality by 31% compared to state-of-the-art drug therapy in primary prevention patients (patients without history of life-threatening ventricular tachyarrhythmias or positive EP study)

## SCD-HeFT Sudden Cardiac Death in Heart Failure Trail

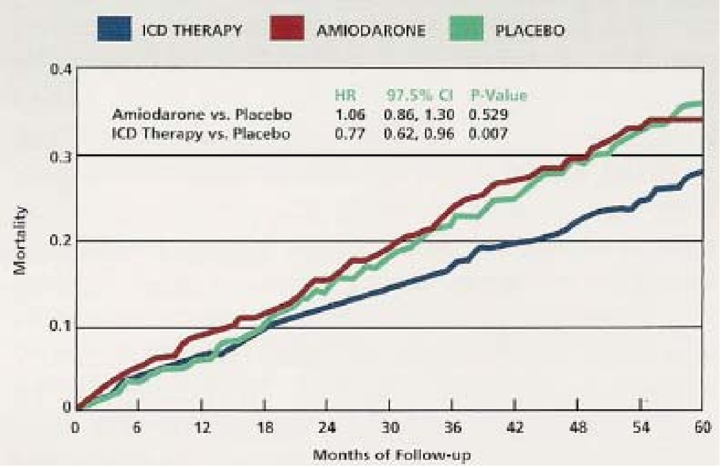
**Objective:**
To compare the use of ICDs versus amiodarone in terms of mortality rates for heart failure patients on state-of-the-are drug therapy

**Inclusion:**
NYHA Class II or III, EF < 35%, patients could be ischemic or nonischemic

**Exclusion:**
NYHA Class I or IV, history of cardiac arrest, ventricular tachyarrhythmia (VT or VF) or one episode of unexplained syncope in past five years

**Results:**
ICDs significantly reduced all-cause mortality by 23% compared to state-of-the-art drug therapy or amiodarone in these primary-prevention heart failure patients.

***SCD-HeFT showed Class II and III CHF patients with and without ischemia reduce their risk of all-cause mortality 23% using a single-chamber ICD system versus amiodarone or a placebo***

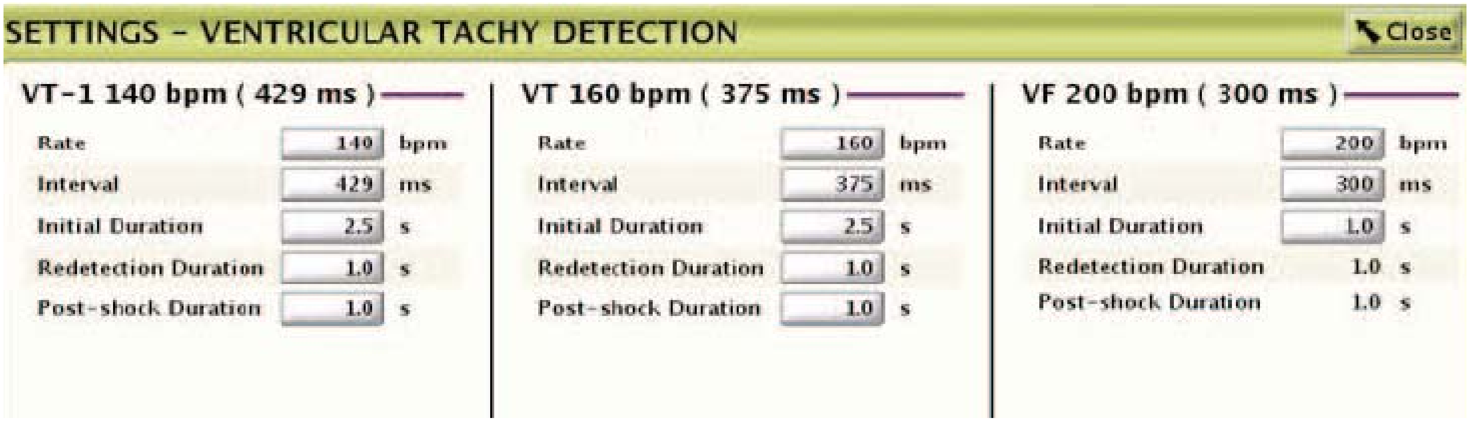


SCD-HeFT found that ICDs conferred a significant mortality benefit compared to placebo (conventional medical therapy) and amiodarone. It is interesting that both amiodarone and placebo had about the same mortality results.

## Arrhythmia Detection

Before a device can declare a tachyarrhythmia is present, the rhythm must meet a programmable rate cutoff value. Most devices offer multiple tachycardia zones for differentiating different types of arrhythmias. For example, a patient with a slow tachycardia, fast tachycardia and an unstable tachycardia of VF may be programmed in a three zone configuration with three rate cutoffs. Once the patient's rate reaches a specific zone, detection of the tachyarrhythmia begins with appropriate sensing.

In addition to meeting rate criteria, the device must count and recognize a certain number of events before the device can determine an arrhythmia is present and needs treatment. Some manufacturers require a pure minimum number binned while others use an x/y scheme. Regardless of the method, a single premature ventricular contraction will not begin the detection and binning phase of arrhythmia detection.



SETTINGS - VENTRICULAR TACHY DETECTION

**VT-1 140 bpm ( 429 ms )**

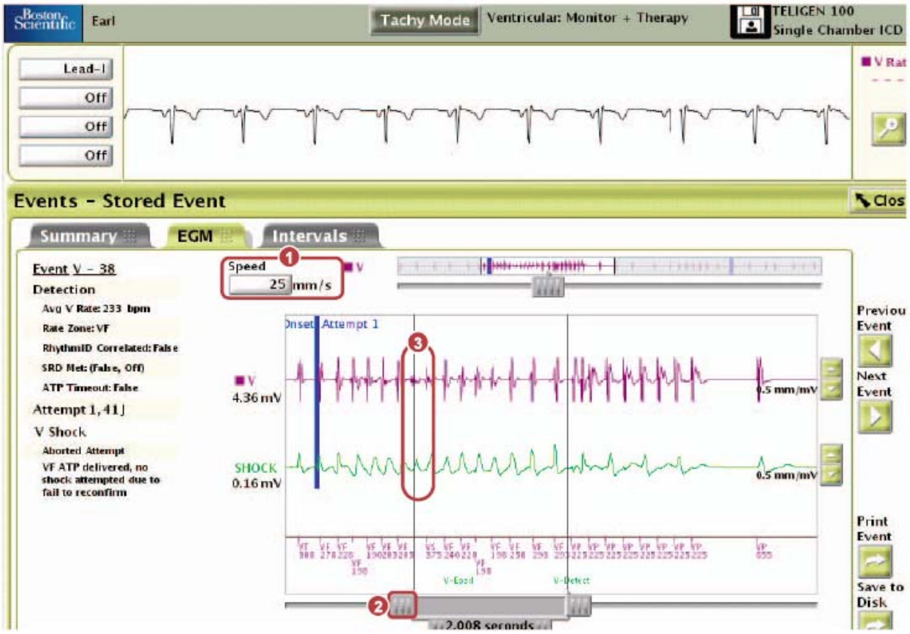
| | | |
|---|---|---|
| Rate | 140 | bpm |
| Interval | 429 | ms |
| Initial Duration | 2.5 | s |
| Redetection Duration | 1.0 | s |
| Post-shock Duration | 1.0 | s |

**VT 160 bpm ( 375 ms )**

| | | |
|---|---|---|
| Rate | 160 | bpm |
| Interval | 375 | ms |
| Initial Duration | 2.5 | s |
| Redetection Duration | 1.0 | s |
| Post-shock Duration | 1.0 | s |

**VF 200 bpm ( 300 ms )**

| | | |
|---|---|---|
| Rate | 200 | bpm |
| Interval | 300 | ms |
| Initial Duration | 1.0 | s |
| Redetection Duration | 1.0 | s |
| Post-shock Duration | 1.0 | s |



Events - Stored Event

Event V - 38

Detection

Avg V Rate: 233 bpm

Rate Zone: VF

RhythmID Correlated: False

SRD Met: (False, Off)

ATP Timeout: False

Attempt 1, 41 J

V Shock

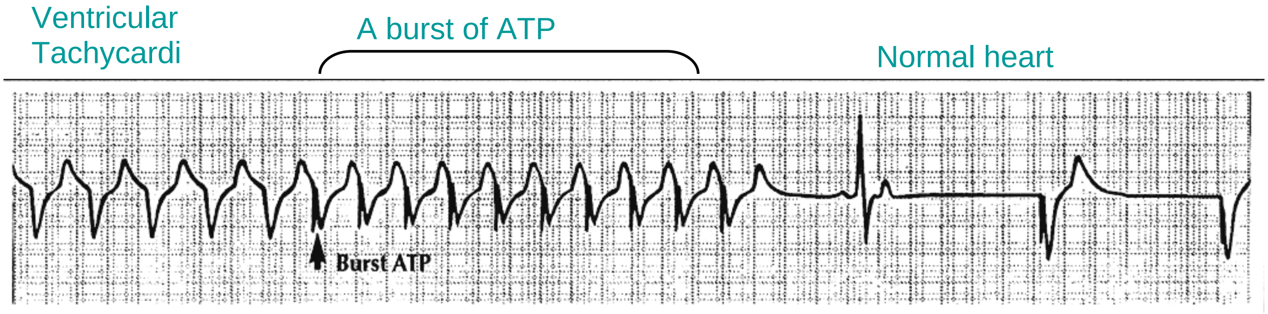
Aborted Attempt

VF ATP delivered, no shock attempted due to fail to reconfirm

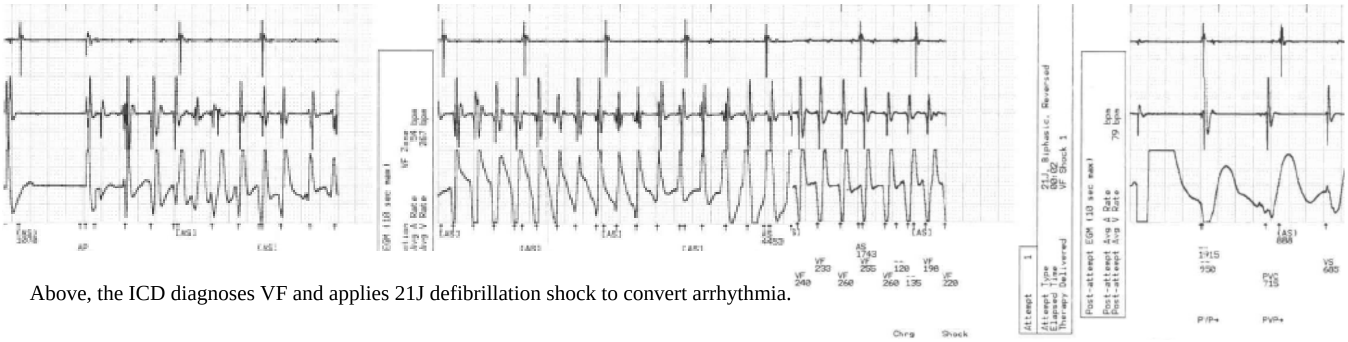
Speed 25 mm/s

## Therapy

ICD device therapy usually consists of two methods, antitachycardia pacing (ATP) or cardioversion-defibrillation, that is, the patient receives a high voltage shock. ATP consists of a series of pacing impulses at a rate faster than the arrhythmia in order to break the reentrant circuit or the electrical pathway of the arrhythmia. ATP is most successful on patients with monomorphic VT, which is a VT that originates from one focus at rates below 200bpm.. Although studies show some effectiveness on faster VTs, ATP is typically used on slower arrhythmias. However, if ATP is ineffective or the arrhythmia causes a patient to be hemodynamically unstable, a hight voltage shock may be required to terminate the arrhythmia.



Above, the ICD diagnoses VT (1st 5 beats) and applies ATP therapy which converts the VT to a paced rhythm.



Above, the ICD diagnoses VF and applies 21J defibrillation shock to convert arrhythmia.

# Notes / Questions