AV Node Reentry Tachycardia

In the past, questions concerning AVNRT have figured prominently in the NASPExAM. Although one can never be certain, I would expect a few questions centered around this disorder.

AVNRT accounts for about 60% of arrhythmias presenting as Supraventricular or Paroxysmal Atrial Tachycardia (PAT). It affects all age groups and sexes equally. There is no evidence that patients presenting with AVNRT have a higher percentage of heart disease than the normal population.

In AVNRT, the AV node can be thought of as divided into two conduction pathways, a fast pathway and a slow pathway. The fast pathway has a longer refractory period than the slow pathway. Tachycardias generally result when a premature impulse is blocked in the fast pathway but continues to travel down the slow pathway. By the time the impulse again reaches the fast pathway in a retrograde fashion, the fast pathway has repolarized allowing the impulse to circuitously reenter the slow pathway. AVNRT is born. Because of the way the tachycardia begins, one will see a prolonged PR interval in the beat that starts the reentry. This is caused by "jumping" from the fast to the slow pathway.

In AVNRT

1. The reentrant circuit is located within the AV Node
2. In most cases, both the atria and the ventricles are stimulated by impulses exiting from the circuit during each lap.
3. Neither the atria nor the ventricles are necessary for the maintenance of that reentrant circuit.
4. It is possible to have block in the His bundle, preventing the ventricles from being stimulated, without affecting the reentrant circuit itself.
5. It is possible to have retrograde block, preventing the atrial from being stimulated, but without affecting the arrhythmia.


Definition/Key Clinical Features

1. AVNRT is the most common form of supraventricular tachycardia; it results from conduction through a reentrant circuit comprising fast and slow atrioventricular nodal pathways
2. Heart rate, 150–250 beats/min
3. Neck pounding
4. Palpitations, light-headedness, near-syncope
5. Narrow QRS complexes on ECG
6. The P wave is either buried within the QRS complex or inscribed just after the QRS complex
7. The P wave inscribed by retroconduction over the AV node is negative in the inferior leads and positive in lead V1; PSVT may manifest as small negative deflections in the inferior leads and a small positive deflection in V1 (pseudo r’ pattern)
8. Abrupt onset and termination of episodes
9. More common in women than in men
10. Frequently presents after 20 yr of age

In AVNRT the anatomic substrate or abnormality is the presence of dual AV node pathways (designated α and β or slow and fast, respectively) each with slightly differing conduction and refractory periods. An extrasystole exposes the differing properties of the two pathways and often initiates tachycardia. During AVNRT, the atria are depolarized retrogradely at a time simultaneous with anterograde ventricular depolarization so that the retrograde P waves are buried in the QRS complex. Sometimes, they are just visible as part of the terminal QRS complex (the r’). Blocking AV node conduction by changing autonomic tone or using pharmacologic agents will terminate the tachycardia.

1Not really. Motivation, intellect and a willingness to spend only 1 minute answering each question are also required.
Rheobase and Chronaxie Time

Rheobase is the lowest point on a strength duration curve at an infinitely long pulse duration. For cardiac pacing, rheobase is usually reached between 1 and 2 milliseconds; at shorter durations, threshold rises.

Chronaxie Time is the pulse width at twice the rheobase value. The Chronaxie Time approximates the most efficient stimulation pulse duration.

Strength Duration Curve - the quantity of charge, current, voltage, or energy required to stimulate the heart at a series of pulse durations.

Within this section of the exam, you may be asked to determine certain electrical parameters using mathematical formulas.

To calculate "charge" in microcoulombs: multiply mean current times pulse duration (time). This is shown by the formula C (charge) = I x T (I is the symbol for current).

To calculate "energy" in microjoules: multiply mean current by mean voltage by pulse duration. The formula may be thus: E = I x V x T.

Try to remember these points:

1. The strength duration curve (SDC) is the quantity of charge, current, voltage, or energy required to stimulate the heart at a series of pulse durations.
2. These values vary significantly as a function of pulse duration.
3. Only charge is approximately linear.
4. In order to set a voltage or pulse duration for parameter output programming, the position that a specific voltage or pulse duration occupies on the curve must be known.

From "A Practice of Cardiac Pacing, Third Edition" by Furman, et. al. Published by Futura.

The following steps are followed in order to determine rheobase and chronaxie

Step 1 – determine the rheobase, which is the minimum Stimulus Strength that will produce a response. This is the voltage to which the Strength-Duration curve asymptotes. In the example above, this value is 0.35 V.
Step 2 – calculate 2\textsuperscript{r}heobase ( = 0.7 V in the above example).
Step 3 – determine chronaxie, which is the Stimulus Duration that yields a response when the Stimulus Strength is set to exactly 2\textsuperscript{r}heobase. In the example above, the chronaxie is 0.22 ms.

Here are a couple mnemonic hints to help you remember which term is which:

1. The root word “rheo” means current and “base” means foundation: thus the rheobase is the foundation, or minimum, current (stimulus strength) that will produce a response.
2. The root word “chron” means time and “axie” means axis: chronaxie, then, is measured along the time axis and, thus, is a Duration that gives a response when the nerve is stimulated at twice the rheobase strength.

\textsuperscript{1}Not really. Motivation, intellect and a willingness to spend only 1 minute answering each question are also required.
1. Functional Refractory Period (FRP)- the coupling interval which first results in a measurable degree of delay in impulse conduction.

2. Effective Refractory Period (ERP)- the longest coupling interval to be associated with block.

3. Resting (transmembrane) potential: the voltage difference between the inside and outside of the cell fiber.

4. Action Potential - the cellular characteristics of depolarization and repolarization. The action potential consists of five phases.

   Phase 0: The depolarization phase. During this phase, the rapid sodium channels are stimulated to open, causing the resting transmembrane potential to spike from about -90 mv to about 0 mv.

   Phase 1: Early repolarization.

   Phase 2: The "Plateau Phase." This phase, mediated by the slow calcium channels, essentially disrupts and delays the repolarization started in phase 1 and prolongs the refractory period.

   Phase 3: The end of repolarization. Note: the period beginning at the end of phase 0 through the end of phase 3 is the refractory period of cardiac tissue.

   Phase 4: The resting phase. It is during this phase that, in some cardiac cells, ions leak back and forth between membranes and cause a gradual increase in the transmembrane (resting) potential. When the potential (voltage) reaches the threshold voltage, the cell depolarizes. This spontaneous depolarization is called automaticity.

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It is not unusual to be presented with a pacemaker rhythm strip without any accompanying information. How does one begin to analyze the rhythm?

First of all, make sure you have a good familiarity with the NBG Code (there will be questions). Next take a systematic approach to interpreting the presenting rhythm.

1. Is pacing present? Yes/No.
   
   Yes: is the pacing appropriate for the rhythm? For instance, are pacer spikes present along with R waves? If so, are the intervals correct? Is the pacing rate the expected rate?

   Are P waves present? Yes/No.

   If so does the pacer seem to "track" the P waves in a 1:1 fashion? Yes/No.

   Yes: is this a DDD(R) pacer or a VDD(R)? On the ExAM, you will be given enough information to answer this question. Observe the surface ECG and note the absence of more than ventricular spikes.

   Do the V spikes appear to be tracking P waves? When the P waves are not present is there only a V spike? If both of these questions are answered YES...select VDD(R) as the answer. In short, think about all the possible modes of pacing and mentally select the appropriate mode BEFORE you look at the possible answers.

2. Is an intrinsic rhythm present? Yes/No.

   Yes: is the pacer appropriately inhibited? Yes, but the P-R interval is 250 ms and the device is programmed to an AV of 150.

   Is the intrinsic rate fast or slow?

   Fast. Possibly a DDD(R) pacemaker with P waves falling in the atrial refractory period and conducted R waves occurring before the end of the atrial pacing escape interval.

It is impossible to predict exactly what types of rhythms will be presented on the exam, however, a good working knowledge of pacemaker timing will go a long way in determining the correct answer.

*Remember:* in dual chamber pacemakers, MOST problems occur on the atrial channel, look there first. Also keep in mind, single chamber, ventricular pacing is usually only indicated in the presence of CHRONIC atrial fibrillation.

**The infamous ANP question:** A few years ago a question appeared on the NASPExAM related to Atrial Naturetic Peptide (these days more often referred to as BNP (Brain Naturetic Peptide), a substance produced by the atrium when the muscle is subjected to higher than normal pressure (such as closing against a closed tricuspid valve). Any question suggesting increased levels of ANP (or BNP) may also be pointing to VVI(R) pacing or loss of atrial capture (or synchrony) as the culprit.
AVNRT: a type of SVT confined within the AV junction requiring a fast pathway plus a slow pathway with unidirectional block in one of the pathways. P waves are absent from the surface ECG due to the junctional rhythm.

Rule of thumb: Ablate the SLOW pathway. Ablation of the fast pathway significantly increases the risk of complete heart block.

Sites used for ablation of the slow pathway range from the midseptal region between the compact AV node and the coronary sinus os to the posteroseptal region around the os. A successful ablation is indicated by:

1: an accelerated junctional rhythm with 1:1 VA conduction during the burn.
2: an increase in refractoriness of the anterograde AV node
3: elimination or alteration in dual AV nodal physiology

The retrograde AV nodal conduction is usually unchanged after slow pathway ablation.

Complications of FAST pathway ablations include:

1: High-grade (2nd or third-degree) heart block
2: Marked first-degree heart block
3: Pseudo-pacemaker syndrome caused by prolonged AV conduction times resulting in atrial contraction during AV valve closure.
4: Persistence of atypical AV nodal reentry employing slow pathways as both the anterograde and retrograde limbs of the tachycardia.

Clinical Presentation of AVNRT

1: Palpitations
2: Dizziness
3: Syncope (occasionally)
4: Rapid regular pounding in the neck is common in AVNRT but not in other SVTs.
5: Polyuria (due to increased levels of Atrial Natriuretic peptide (ANP))

Typical ECG presentation

1: Regular, narrow QRS complex tachycardia
2: No visible P wave (or P wave located in the terminal portion of the QRS)

Helpful ECG patterns:

1: r' pattern in lead V1
2: pseudo S waves in leads II, III, or aVF
3: no discernible P wave separate from the QRS complex

Other diagnostics:

1: Typical AVNRT is rarely induced with ventricular pacing in the EP lab, while this is the rule for Atypical AVNRT.
2: Administration of adenosine during the tachycardia helps to differentiate an atrial tachycardia from AVNRT. If AV block is produced while the atrial rhythm continues unchanged, this is highly suggestive of an atrial origin of the tachycardia.
3: Earliest activation at the low interatrial septum during tachycardia is consistent with AVNRT.

Much of the information presented in this edition is taken from the Electrophysiology Self-Assessment Program (EPSAP) published by the American College of Cardiology and NASPE.

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Possible Sources of Interference or Damage

Devices with NO effect

1. Microwave Oven
2. CT Scan/Ultrasound
3. X-Rays – diagnostic

Devices that cause transient or 1 beat Inhibition

1. Electronic Article Surveillance (EAS)
2. Cellular Telephones
3. Arc Welding (asynchronous pacing may also occur)
4. Airport Metal Detectors (asynchronous pacing may also occur)
5. TENS (nerve stimulator) - (asynchronous pacing or total inhibition may also occur)
6. Electric Appliances:
   - Electric blanket
   - Electric shaver
   - TV
   - Can opener
   - CB Radio
   - HAM Radio
   - Power tools
   - Metal detector
   (Note: all the electric appliances RARELY cause transient or 1 beat interference)

Devices that may damage the pacemaker

1. MRI: (April 19, 2005 — A growing body of evidence suggests that magnetic resonance imaging (MRI) may be safe for selected patients with implantable cardioverter defibrillators (ICDs) and pacemakers; however, more research is needed before this issue can be definitively resolved, according to a “mini symposium” on the subject that appears in the journal Pacing and Clinical Electrophysiology (PACE).
2. Defibrillator
3. Cardioversion
4. Cautery/RF Ablation
5. Radiation Therapy

Epsap II: Electrophysiology Self-Assessment Program
Authors: Gerald V. Naccarelli
Release: 2000-06
Publisher: American College of Cardiology
Format: Unknown Binding
ISBN: 158397007X EAN: 9781583970072
List Price: $645.00

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Electromagnetic Interference and the Pacemaker Patient

While clinically significant problems with electromagnetic interference (EMI) are rare, a pacemaker’s response to EMI becomes more diverse as technology advances. Pacemaker manufacturers continue to develop interference protection circuitry to keep up with these vast sources of EMI.

The pacemaker’s response to EMI is dependent on the characteristics of the EMI, proximity to the interference, available shielding, and the sensing characteristics and polarity of the pacemaker. The pacemaker circuitry is designed to attenuate any interference outside the normal intracardiac range (10 Hz – 100 Hz). This is achieved by using bandpass filters.

EMI sources can be broadly classified as galvanic, electromagnetic or magnetic.

- Galvanic interference requires direct contact with electrical current. This is most often seen in defibrillation/cardioversion, cautery, TENS units and diathermy.

- Electromagnetic or electrically coupled interference does not require direct body contact. This interference is most often seen with arc welders, ham radios, electrical appliances, metal detectors, therapeutic ultrasound and high voltage power lines.

- Magnetic interference occurs when a patient comes in close proximity with an intense magnetic field. This is often seen in nuclear magnetic resonance imaging (NMR/MRI) and steel mill induction furnaces.

EMI with signal modulation can mimic normal intracardiac signals. When detected, the response to EMI may present itself as a single beat inhibition, total inhibition, noise reversion/asynchronous pacing, rate increase, erratic pacing, or no output. These responses are usually temporary, but can be permanent if the pulse generator circuitry is damaged.

A pacemaker’s response to EMI is highly dependent on the specific EMI source, the pacemaker’s mode, and sensing polarity. Included is a list that details the interaction of commonly encountered pacemaker EMI sources. Accompanying this list is a summary table of these sources and reported associated pacemaker responses.
EMI Sources

Ablation (RF): Loss of capture - exit block is frequently seen during RF ablations. Arrhythmia induction, undersensing, inhibition, rate increase and noise reversion pacing are also possible. Circuit damage is less likely than DC ablation.

Acupuncture: Low frequency electroacupuncture may cause inhibition and noise reversion at high frequencies.

Airport detector/Metal detectors: Single beat inhibition is rare and seen only on unipolar devices.

Anti-theft devices/Electronic Article Surveillance (EAS): Possible inhibition or rate increase reported primarily on unipolar devices especially if patient leans or lingers near EAS. An increased incidence of cross-talk is seen on unipolar DDD pacers.

Arc welders: Single beat inhibition is commonly seen on unipolar devices each time the arc is struck. High magnetic fields from the cables may cause reed switch closure resulting in asynchronous pacing.

Bone Stimulator: Possible inhibition on unipolar devices.

Cardioversion: Cardioversion, performed at high energies similar to that of defibrillation or performed directly over the pulse generator, may damage circuitry resulting in no output, erratic pacing, or rate increases. Energy conducted through the lead may cause arrhythmias and myocardial burning.

Cautery: Cautery used near the pacing system may result in inhibition, asynchronous pacing and/or circuit damage. Energy conducted through the lead may cause arrhythmias and myocardial burning. Impedance-based rate responsive pulse generators may exhibit erratic pacing or rate increases.

CB radio: Single beat inhibition may be seen with microphone keying on unipolar devices.

Cellular Phone: Total inhibition or asynchronous pacing is possible with some digital cell phones if placed within 6 inches of the pacemaker. Current SJM pacemakers (Identity, Integrity, Affinity, Trilogy, Synchrony, Paragon, Solus) are cellular tested.

CT Scan: No documented reports of interference to date from CT scanners or full body scans.

Defibrillation: Defibrillation performed at high energies, or defibrillation directly over the pulse generator, may damage circuitry resulting in no output, erratic pacing, or rate increases. Energy conducted through the lead may cause arrhythmias and myocardial burning.

Dental scaler: Older ferromagnetic ultrasonic scalers may cause single beat inhibition on unipolar pacemakers. Piezo-electric scalers have no effect. Activity rate responsive devices may exhibit increased pacing rates.

Diathermy: Used in the near vicinity of the pacing system, diathermy may result in inhibition, asynchronous pacing, and/or circuit damage. Energy conducted through the lead may cause arrhythmias and myocardial burning.

Electric blanket/ Heating pad: Single beat inhibition is rare and seen only on unipolar devices.

Electric shaver: Single beat inhibition is rare and seen only on unipolar devices.

Electric switch: Single beat inhibition may be seen on unipolar devices.

Electric tools: Single beat inhibition is rare and may be seen on unipolar devices during use of power tools like drills and saws.

Electric toothbrush: No effect from standard or ultrasonic models.

Electro-convulsive shock therapy (ECT/EST): Inhibition and/or noise reversion is possible, especially with unipolar pulse generators. Activity sensor rate responsive pulse generators may track the seizure activity.
Electrotome (dental device): Single beat inhibition is rare and seen primarily on unipolar devices.

Ham radio: Single beat inhibition may be seen on unipolar devices during microphone keying.

Lithotripsy - ESWL: No effect on VVI and VOO pulse generators. DDD pulse generators may track to maximum rate or totally inhibit ventricular output due to ESWL triggering off the atrial output. Activity sensor rate responsive pulse generators may also track to maximum rate or permanently damaged (piezo crystal shatters near focal point).

Magnet therapy: Asynchronous pacing possible if magnetic pads/objects are used within 18 inches of pacemaker. Prolonged asynchronous pacing from magnetic mattress pads is not recommended. Magnetic pads used below the waist will not interfere with pacemaker operation.

Microwave ovens: In 1976 the FDA stated there is no longer substantial risk of pulse generator interference from microwave ovens which are now built with leakage protection. Pulse generators are now manufactured to prevent interference from microwaves.

MRI (Magnetic Resonance Imaging): Frequent effect from MRI is asynchronous pacing. Reed switch magnetization, rate increases in DDD, single beat inhibitions, component damage, lead dislodgment, rapid pacing (300 PPM), and generator movement within the pocket are also possible but not common.

PET Scan: Possible CMOS damage. See Radiation.

Power lines, high voltage: 400 kvolt high voltage power lines may cause asynchronous pacing, especially if patient is near a large metal object (e.g. car).

Pulp tester: Single beat inhibition is rare but may be seen on unipolar devices.

Radar: Single beat inhibition is rare but may be seen on unipolar devices.

Radiation, Diagnostic: No effect, even with cumulative doses.

Radiation, Therapeutic: Damage to the CMOS circuitry can occur as low as 2000 rads in some pacemakers. Devices now manufactured by SJM are tested to 3000 rads. Effect is cumulative in dose and affects both bipolar and unipolar pulse generators. Failure modes include circuit damage, run-away pacer, erratic pacing, sensing anomalies, and no output.

Radio transmitter, AM: If signal modulation occurs, inhibition may be seen on unipolar pulse generators, relative to power, frequency, modulation, and proximity. Noise reversion pacing is possible.

Radio transmitter, FM: If signal modulation occurs, inhibition may be seen on unipolar pacemakers relative to power, frequency modulation, and proximity. Noise reversion pacing is possible.

Respiratory/ECG monitors: Impedance based ECG/respiratory monitors may cause upper rate pacing in impedance based pacemakers especially with monitors emitting a current signal parallel to the pacer system.

Shaw scalpel: This non-electric cautery is thermally coupled and will not cause any interference.

TENS (Transcutaneous Electrical Nerve Stimulator): Normally used high frequencies (>30 Hz) may cause noise reversion on unipolar pulse generators. Low frequencies (<10 Hz) may cause inhibition on unipolar pulse generators. Burst mode on newer TENS units is contraindicated due to probable device inhibition.

TV transmitter: Although rare, inhibition and noise reversion of unipolar devices has been documented.

Ultrasound, Diagnostic: No effect.
**Ultrasound, Therapeutic:** Single beat inhibition is rare and may be seen on unipolar devices. Therapy should not be given directly over the pulse generator. Activity sensor rate responsive pulse generators may exhibit piezo crystal shatter.

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### EMI Summary Table

of Commonly Encountered Sources and Responses

<table>
<thead>
<tr>
<th>Source</th>
<th>Pacer Damage</th>
<th>Total Inhibition</th>
<th>1 Beat Inhibition</th>
<th>Asynch/Noise</th>
<th>Rate Increase</th>
<th>Unipolar Bipolar</th>
</tr>
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<tbody>
<tr>
<td>Ablation (RF)</td>
<td>Y*</td>
<td>Y</td>
<td>Y</td>
<td>Y*</td>
<td>U &amp; B</td>
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<tr>
<td>Acupuncture</td>
<td>N</td>
<td>Y</td>
<td>Y</td>
<td>N</td>
<td>U &amp; B</td>
<td></td>
</tr>
<tr>
<td>Airport detector</td>
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<td>N</td>
<td>Y</td>
<td>N</td>
<td>U</td>
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<td>Anti-theft device (EAS)</td>
<td>N</td>
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<td>Y</td>
<td>Y*</td>
<td>U&amp;B</td>
<td></td>
</tr>
<tr>
<td>Arc welder</td>
<td>N</td>
<td>Y</td>
<td>Y</td>
<td>N</td>
<td>U &amp; B</td>
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</tr>
<tr>
<td>Bone stimulator</td>
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<td>Y</td>
<td>Y</td>
<td>N</td>
<td>U</td>
<td></td>
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<td>Y</td>
<td>Y</td>
<td>N</td>
<td>U &amp; B</td>
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<td>Cardioversion</td>
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<td>N</td>
<td>Y</td>
<td>N</td>
<td>U &amp; B</td>
<td></td>
</tr>
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<td>Cautery/coagulation</td>
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<td>Y</td>
<td>Y</td>
<td>Y*</td>
<td>U &amp; B</td>
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<tr>
<td>CB radio</td>
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<td>Y</td>
<td>N</td>
<td>U</td>
<td></td>
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<td>Cellular phone</td>
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<td>Y</td>
<td>Y*</td>
<td>N</td>
<td>U &amp; B</td>
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<tr>
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<tr>
<td>Defibrillation</td>
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<td>N</td>
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<td>N</td>
<td>U &amp; B</td>
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<td>Dental scaler</td>
<td>N</td>
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<td>Y*</td>
<td>Y*</td>
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<td></td>
</tr>
<tr>
<td>Diathermy</td>
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<td>Y</td>
<td>U &amp; B</td>
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<td>ECT/EST</td>
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<td>N</td>
<td>Y</td>
<td>Y*</td>
<td>U</td>
<td></td>
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<td>Electric blanket/heating pad</td>
<td>N</td>
<td>N</td>
<td>Y*</td>
<td>N</td>
<td>U</td>
<td></td>
</tr>
<tr>
<td>Electric shaver</td>
<td>N</td>
<td>N</td>
<td>Y*</td>
<td>N</td>
<td>U</td>
<td></td>
</tr>
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<td>Electric switch</td>
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<td>N</td>
<td>Y</td>
<td>N</td>
<td>U</td>
<td></td>
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<tr>
<td>Electric tools</td>
<td>N</td>
<td>N</td>
<td>Y*</td>
<td>N</td>
<td>U</td>
<td></td>
</tr>
<tr>
<td>Electric toothbrush</td>
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<td>N</td>
<td>N</td>
<td>-</td>
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<td>Electrolysis</td>
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<td>Ham radio</td>
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<td>N</td>
<td>Y</td>
<td>N</td>
<td>U</td>
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<td>Lithotripsy</td>
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<td>Y</td>
<td>N</td>
<td>U &amp; B</td>
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<tr>
<td>Magnet therapy</td>
<td>N</td>
<td>N</td>
<td>Y</td>
<td>Y</td>
<td>U &amp; B</td>
<td></td>
</tr>
<tr>
<td>Microwave</td>
<td>N</td>
<td>N</td>
<td>N</td>
<td>N</td>
<td>-</td>
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<tr>
<td>MRI</td>
<td>Y</td>
<td>N</td>
<td>Y</td>
<td>Y*</td>
<td>U &amp; B</td>
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<td>PET scanner</td>
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<td>N</td>
<td>N</td>
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<td>Powerline, high voltage</td>
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<td>N</td>
<td>N</td>
<td>Y</td>
<td>N</td>
<td>U &amp; B</td>
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<tr>
<td>Pulp tester</td>
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<td>Y*</td>
<td>Y*</td>
<td>Y</td>
<td>N</td>
<td>U</td>
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<td>Radar</td>
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<td>N</td>
<td>N</td>
<td>U</td>
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<td>Radiation - Diagnostic</td>
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<td>N</td>
<td>N</td>
<td>N</td>
<td>-</td>
<td></td>
</tr>
<tr>
<td>Radiation - Therapeutic</td>
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<td>N</td>
<td>N</td>
<td>Y</td>
<td>U &amp; B</td>
<td></td>
</tr>
<tr>
<td>Radio transmitter AM</td>
<td>N</td>
<td>N</td>
<td>Y*</td>
<td>N</td>
<td>U</td>
<td></td>
</tr>
<tr>
<td>Radio transmitter FM</td>
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<td>N</td>
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<td>Y*</td>
<td>N</td>
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<td>U &amp; B</td>
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</table>
**EMI Bibliography**


FDA: Important information on anti-theft and metal detector systems and pacemaker, ICDs, and spinal cord stimulators. FDA Safety Note, Sept. 1998.


ExAM Tidbits in easy to digest, bite sized morsels

Invariably, the NASPExAM AP has a few questions that require a good working knowledge of Ohm's Law. To fully understand the concepts required for the ExAM, pay careful attention the following info taken from "A Practice of Cardiac Pacing – Third Edition" by Furman, et al.

Furman departs from the formula labels commonly taught by the device companies. In most classes teaching Ohm's Law, the formula is described thus

\[ V = IR \]

Where \( V \) is voltage (voltage is defined as electromotive force, or the force that drives electron flow)
Where \( I \) is current (or the flow of electrons)
Where \( R \) is the resistance to current flow measured in ohms

Furman's formula labeling is this: \( E = IR \) (where \( E \) represents Electromotive Force rather than calling it \( V \) for voltage). This can become confusing since Energy (measured in joules) is sometimes labeled \( E \). Make sure you know what you are reading. Furman's formula for determining energy is: \( \text{ENERGY} = EI t \) or voltage x current x time (pulse width). See what I mean? You have to keep your eyes open.

A favorite type of question concerns resistance in series and in parallel. Series means that the beginning of one resistance is connected to the end of another. Resistance in series is easy. It is simply the sum of the resistances: \( R_1 + R_2 = \text{Total resistance} \). As with all simple concepts, there is a backside to this one. What happens if the question asks you to determine the percentage of voltage drop across a resistance. The example given in Furman is fairly simple, but I'll use it.

Let's say one wire shows 10 ohms and the other 40 ohms. The total resistance is 50 ohms. The voltage drop across the 10 ohm wire will be 20% of the total and the voltage drop across the 40 ohm wire will be 80%. The sum of percentage must be 100%, therefore you must determine the ratio of \( R_1 \) to \( R_2 \) to determine the percentage drop across each point.

NOTE: a lead fracture is an example of a series resistance.

Resistances are said to be in Parallel when all the resistances are connected to the SAME point. An example of this would be a lead insulation defect. In parallel resistances the product of the resistances is divided by their sum. The formula is

\[ \frac{R_1 \times R_2}{R_1 + R_2} \]

Remember: Lead fractures cause an INCREASE in impedance. Lead insulation defects cause a DECREASE in impedance.

Furman's 3rd edition calls the normal lead impedance range 400-600 ohms. This could be argumentative since modern leads actually have a NORMAL range from less than 400 (in some cases mid 300 ohm) to over 1000 ohms. I'm really not sure what you will be given as choices on the exam.

Furman also discusses constant current vs constant voltage pulse generators. These days, ALL permanent pacemakers are constant voltage devices. SOME temporary pacemakers are constant voltage, most are constant current.

The term LOAD refers to impedance (or resistance) applied to a circuit. A system with a SMALL Load (low impedance) applied to the circuit is said to be a constant current device. A system with a LARGE Load is said to be a constant voltage device.

Had enough? Boy, I have. More on electricity next time (maybe).

Remember, your input is always appreciated and requested. Please feel free to get into the game and add your own spin to these newsletters.

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On nearly every AP ExAM there is at least one question centering around indications for permanent pacing. The following statement oversimplifies a bit, but may be useful for some of these questions. A permanent pacemaker is indicated when:

1. The patient is symptomatic
2. The heart rate is less than 40 beats per minute
3. Asystole of greater than 3 seconds is documented NOTE: the patient may be ASYMPTOMATIC with either 2 or 3, above.

A .pdf file containing the complete text of the guidelines is available at:
http://www.americanheart.org/downloadable/heart/1032981283481CleanPacemakerFinalFT.pdf

A few tidbits---


Believe it or not, you may have to actually calculate a "slew rate" from a given set of parameters. The slew rate is actually the "peak slope" of an electrogram signal. The formula for determining the slew rate is dV/dt or Voltage (in millivolts) divided by time (in milliseconds). The values that are considered "normal" are >.3 v/s in the atrial chamber and >.5 v/s in the ventricular chamber. These numbers are a little slippery and differences in opinion are almost certainly going to surface. Stay tuned. The slew rate is most important in the presence of low amplitude intrinsic signals and has almost NO effect on sensing when signal amplitudes are high (>2 mv p waves or >4 mv r waves).

Steroid-eluting electrodes use the corticosteroid dexamethasone sodium phosphate usually impregnated in a silicone core. Keywords here: corticosteroid and dexamethasone.

A couple of things to remember about steroid-eluting electrodes:

1. The acute threshold phase is relatively flat compared to non-steroid electrodes.
2. The initial capture threshold is similar to non-steroid leads.

Characteristics of Pacemaker Lead Insulation

Silicone Rubber

5. Tears easily if suture tied too tightly 6. Large diameter (compared to polyurethane)

Polyurethane: REMEMBER THIS: 80A = BAD and 55D = GOOD

Pros:
1. Relatively nonthrombogenic/fibrotic
2. Thin walls (compared to silicone)
3. High tear strength
4. Resists cutting
5. Low friction coefficient

Cons:
1. 80A = Bad
2. Cannot be repaired
3. Relatively stiff
4. Hard to make (manufacturing process must be meticulous)

NOTE: some of these Pros and Cons may be controversial among device manufacturers. The list as presented here was taken from "Cardiac Pacing" edited by Kenneth Ellenbogen. The specific section is "Basic Aspects of Cardiac Pacing" by G. Neal Kay, MD; pp 32-119.

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ExAM Tidbits in easy to digest, bite sized morsels

Volume 1: Number 9

**Pacemaker Syndrome/Upper Rate Behavior**

**Pacemaker Syndrome** – any combination of the variety of symptoms and signs occurring with ventricular pacing that are relieved by restoration of AV synchrony. ["Hemodynamics of Cardiac Pacing" by Dwight W. Reynolds, M.D., from Cardiac Pacing, Edited by Kenneth Ellenbogen, 1995]

**Causes:**
1. Loss of AV synchrony
2. Sustained retrograde conduction
3. A single ventricular rate when rate modulation is required for exercise

Approximately 25% of patients paced only from the ventricle may have some level of severity related to pacemaker syndrome.

**Diagnosis**
1. Observe fluctuation in the peripheral blood pressure
2. Cannon "A" wave in the neck
3. History alone

**Management** = restore AV synchrony

Counterintuitive management: lower the pacing rate to minimize ventricular only pacing. This may cause the blood pressure to fall, but the symptoms of pacemaker syndrome may disappear. DO NOT raise the pacing rate. Raising the rate may entrain P waves in a retrograde fashion. This will make the pacemaker syndrome worse.


**Upper Rate Behavior**

On both NASPExAMs that I was subjected to (AP & EP), there were questions on "Fallback," "Rate Smoothing," and Sensor Upper Rate behavior. With that in mind, here are a few thoughts.

**Fallback.**
1. Decouples atrial and ventricular events at the upper rate limit
2. The ventricular inhibited pacing rate then gradually decrements to a programmed lower or "fallback" rate over a programmed duration
3. When the fallback rate is reached, atrial synchrony is resumed

**Rate Smoothing.**
1. Eliminates large cycle to cycle variations by preventing the paced rate from changing more than a certain percentage (3%, 6%, 12%, etc.) from one paced V-V interval to the next
2. Eliminates large fluctuations in rate during fixed-ratio or pseudo-Wenckebach block

Rate smoothing is found in devices manufactured by Guidant.

**Sensor Upper Rate Behavior.**
1. The ventricle is paced in an AV sequential fashion in response to sensor input
2. If the sinus (P) rate is faster than the sensor indicated rate, P synchronous pacing occurs
3. If the sensor indicated rate is faster, AV pacing at the sensor indicated rate occurs
4. A "mixed" scenario can occur when the device is sensor driven AV pacing for a few cycles and a sinus rate sudden emerges faster than the sensor indicated rate. The sensor driven atrial output will be inhibited, a PR interval started, and a ventricular output will occur at the end of the sensor AV interval. That is, the ventricular rate will be equal to the sensor indicated rate, but the PV interval may be longer (by a few milliseconds) than expected.

**Bonus Bite:** 3 good case studies by Mark Sweesy can be found at the following link:

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Neurocardiogenic Syncope

An understanding of neurocardiogenic syncope may be helpful for a couple of the questions in the EP exam. I hope the following information is helpful. It pertains primarily to one manufacturers device, but the principles are generic. Pay closer attention to the causes of neurocardiogenic syncope, rather than this manufacturers' solution.

The Medtronic Rate Drop Response (RDR) is designed to detect rate drops associated with neurocardiogenic syncope (a.k.a. Vasovagal syncope (VVS)) and provide high rate pacing to eliminate or reduce the symptoms associated with the condition. Here is how the algorithm works:

1. Preceding an episode of VVS there is usually an increase in the heart rate: (HR). To identify this rate rise the pacemaker is programmed to a "Top" rate.

2. The rise in HR is immediately followed by a fall in the heart rate. To identify this drop in rate, the pacemaker is programmed to identify a "Bottom" rate.

3. The HR drop must be identified, therefore a number of "Width" beats must be programmed. The HR must fall to the "Bottom" rate in fewer than the programmed "Width" beats.

4. To confirm the rate drop, a small number of "Confirmation" beats must be programmed. The HR must remain below the "Bottom" rate for this number of "Confirmation" beats in order for the algorithm to activate.

(Benditt, et al: Clinical experience with Thera DR Rate-Drop Response pacing algorithm in carotid sinus syndrome and vasovagal syncope. pp. 832 - 839)

In order to properly program the RDR, the patient should undergo "at least" one tilt test cycle. Tilt testing is a requirement for making sure that the pacemaker is appropriately programmed. In order for the algorithm to be effective the following sequence appears to be essential:

1. An initial rise in the HR to a value at or above the "Top Rate" must occur.
2. A rapid fall in HR must then follow.
3. A fall in BP (resulting in symptoms) must occur next.
4. The above (1-3) should be present at all syncopal episodes.
5. Syncope should not occur until the HR has dropped by 20 to 30 bpm.
6. Rapid AAI or DDI pacing should then prevent the syncope, or at least reduce the symptoms.

Neurocardiogenic Syncope has been subdivided by Dr. Richard Sutton (1993) into three types.

Type 3: (pure vasovagal response). This type is unlikely to benefit from RDR since syncope occurs before significant bradycardia.

Type 2b: (cardioinhibitory). This type is the most likely to respond to pacing therapy.
   a. IDR almost certainly
   b. DDI with hysteresis, possibly, since the BP falls after significant bradycardia

Type 2a: (cardioinhibitory). In this subclassification, the HR falls below 40 bpm but the blood pressure falls before the heart rate.
   a. The patient may receive some small benefit for RDR
   b. Full resolution of symptoms is highly unlikely

Type 1: (mixed response). The bradycardia associated with this type usually occurs after the drop in B/P but the HR stays above 40 bpm,
   a. The patient may receive some small benefit from RDR
   b. Most likely will require some form of pharmaceutical therapy c. Reduction of symptoms rather than-resolution of symptoms is the goal.

Type 1a: (mixed response). The B/P falls simultaneously with or slightly after the HR. Profound symptoms occur, even though the HR may remain above 40bpm. In this type, the HR fall is often preceded by an increased "oscillation" in rate or more pronounced respiratory variation in the HR. Patients may benefit from RDR.

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So, who are the ideal patients for RDR therapy?

1. Must have an initial heart rate rise with tilting (or before syncopal episodes)
2. The heart rate must begin to fall before the blood pressure during tilt testing
3. The response must be reproducible and runs true on each occasion of tilt testing or spontaneous syncope
4. Rapid pacing (AAI/DDI) abolishes or markedly ameliorates the symptoms.

(Gammage MD; A useful screen for Rate-Drop Response. pp. 329–831)

Keep in mind that neurocardiogenic syncope is not as simple as it may, at first seem. It is a complex condition precipitated by a number of factors. These factors are generally accepted to include inappropriate slowing of the HR due to:

1. sudden augmentation of efferent vagal activity
2. arteriolar dilatation by sudden reduction of sympathetic activity

(Van Lieshout, el al: Neural Circulatory Control in vasovagal syncope. pp: 753 - 763)

These factors are regulated by the arterial baroreflexes and the cardiac reflexes. Baroreceptors are located in the Aortic Arch and the Carotid Sinus. Baroreceptor discharge causes excitation of resting parasympathetic output to the SA node and resting inhibition of sympathetic output to the heart and peripheral circulation.

Because of the complex nature of the VVS, simply increasing the heart rate may have little or no effect on symptoms. Injection of atropine to increase HR has, historically, shown no reduction in symptoms.

Quan KJ, Carlson MD, Thames MD: Mechanisms of heart rate and arterial blood pressure control: Implications for the pathophysiology of neurocardiogenic syncope. pp: 764 -774)


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The following may be more than you ever wanted to know about VDD(R) pacing, but some of this may be helpful come early-May.

**VDD or VDDR?**

To begin, since a VDD pacer only paces in the ventricle, it can only operate in the VDD mode when sensed atrial events are present. No P, no P tracking. In the absence of atrial sensed events the VDD pacer appears to be operating in the VVI mode (since the device is technically able to sense P waves it is actually operating as a VDD device). What about rate responsive operation? VDDR is really performing as though it were VVIR when the sensor indicated rate is greater than the sensed sinus rate. One may think of VDD(R) pacing as VVI pacing with a PV delay.

**SOMETHING TO THINK ABOUT:** In VDDR pacing there are four rates to always keep in mind. There is, of course, the base rate. This is the rate when there is no underlying sensed atrial or ventricular event to get in the way of the pure VVI (appearing) pacing. Next there are the maximum tracking rate and the upper sensor driven rates that operate along the same principles as their counterparts in DDDR pacing. The fourth rate is the minimum pacing rate. When a P wave occurs at the end of the escape interval, a PV delay is activated. This causes the minimum rate of a VDD device to be equal to the basic interval plus the PV delay interval. A pacer programmed to a 1000 ms interval (rate 60) with a PV of 200 ms will have a minimum pacing interval of 1200 ms (rate 50). Simply divide the interval into 60,000 any time there is a question about the minimal rate.

**LEADS, ELECTRODE SPACING, ETC.**

Single lead VDD pacing utilizes an electrode design wherein the atrial portion of the electrode is 'floating' in the atrial cavity detecting atrial depolarization through the bloodstream. At least five lead designs have been tried with varying results. These five designs are: unipolar, bipolar (wide ring spacing), bipolar (narrow ring spacing), orthogonal, and diagonal half ring (AKA, diagonal atrial bipolar or DAB).

Unipolar

The obvious downside to the unipolar design is the propensity towards oversensing extraneous signals such as myopotentials. In one study, using the CPI Ultra II, 98 patients were evaluated with the unipolar single lead system. Although some problems were noted, including myopotential inhibition and atrial undersensing, the authors did not feel the problems to be of any hemodynamic consequence. Ninety-three percent of the patients implanted in this study (1985-89) remain in the VDD mode.

Bipolar (wide spacing)

Although this configuration appears to eliminate the likelihood for myopotential oversensing it is also the configuration with the lowest and widest signal deflections. The ring to ring distance for this widely spaced electrode is approximately 3 cm. Bipolar (narrow spacing) Interestingly, this configuration produces the highest and fastest signal deflections. The optimal electrode spacing appears to be between 0.5 and 1 cm.

Orthogonal

Orthogonal electrodes are half rings located directly opposite each other on the lead body and each plate is, likewise, electrically opposite. The orthogonalized lead produces a resultant electrogram amplitude that is twice the differential field strength. This configuration significantly attenuates the far field R wave signal, totally eliminates myopotentials and EMI noise. Diagonal Bipolar Half Ring (DAB)

This system is similar to the orthogonal except that the half ring electrodes are spaced between 0.5 and 1 cm apart. This is the system used by Cardiac Control Systems and Intermedics in their single lead VDD systems. Signal amplitude with this type of lead is slightly less than that from the bipolar, narrow spaced leads. Ventricular Tip to Atrial Sensing Electrode Distances For optimal placement the dipole should be placed "as close as possible to the mid-to-high atrial wall. Active ventricular fixation may simplify this maneuver." This means that, in normal adults, the tip to dipole distance should be about 13 cm. Leads from various manufactures place the dipole from 9.5 to 17.5 cm from the ventricular tip. **INDICATIONS VDD pacing is indicated in patients with complete heart block without evidence of chronotropic incompetence, sinus node disease, retrograde conduction, or atrial arrhythmias.**

**REFERENCES**

Clinical Description:

1. Right bundle branch block
2. ST segment elevation in V1 to V3
   a. The electrocardiogram shows ST segment elevation in the precordial leads V1 to V3
   b. Morphology of the QRS complex resembling a right bundle block known as J point elevation
3. Sudden death
4. Structurally normal heart

Presentation:

1. Syncope and sudden death (aborted or not) caused by fast, polymorphic ventricular tachycardias or ventricular fibrillation.
2. These arrhythmias appear with no warning.
3. There is no prolongation of the QT interval during sinus rhythm.
4. Only in very few cases there is alternation of long-short sequences before the polymorphic ventricular tachycardia, a finding which is very common in other arrhythmias like "torsade de pointes" in the long QT syndrome.
5. There is no preceding acceleration in the heart rate as is the case of catecholamine-dependent polymorphic ventricular tachycardia.

The Asian Connection:

1. There is an abnormally high incidence of sudden death in young men from Southeast Asia.
2. In the Northeastern Thailand, this form of death is known as Lai Tai (death during sleep).
   a. Unexpected sudden death is the most common cause of natural death in young Thai people.
   b. Many of these patients suffer the Brugada syndrome.
3. In Philippines the phenomenon is known as Bangungut (scream followed by sudden death during sleep)
4. In Japan as Pokkuri (unexpected sudden death at night).
5. The incidence of this form of sudden death has been estimated between 26 and 38 cases per 100,000 inhabitants per year.
6. In Laos it may cause 1 sudden death per 1,000 inhabitants per year.
7. The higher prevalence of this syndrome in some areas can be explained by its genetic transmission.

Etiology:

1. This syndrome is genetically determined.
2. Approximately 60% of patients with (aborted) sudden death with the typical electrocardiogram have a family history of sudden death, or have family members with the same electrocardiographic abnormalities.
3. There are also sporadic cases who are probably the patients with a de novo mutation in the family.
4. The pattern of transmission is autosomic dominant.
5. There is a predominance of affected males.
6. In Thailand the disease almost exclusively affects males.
7. Several mutations linked to this syndrome affecting the gene SCN5A which encodes for the cardiac sodium channel have been described.
8. Some of the families studied do not present these mutations in this gene, indicating that other genetic defects will be found and that this is a genetically heterogeneous disease.

Due to the sodium channel involvement, the arrhythmias are based on phase 2 (of the action potential) re-entry.

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Electrophysiologic and Hemodynamic findings

1. Sinus node function is normal in the large majority of patients.
2. Isolated patients have manifest sinus node disease and are pacemaker dependent.
3. About 10% of patients have paroxysmal atrial fibrillation.
4. Inducibility of polymorphic ventricular tachycardia by programmed electrical stimulation in symptomatic patients.
   a. About 80% of them are inducible by giving 1 or 2 ventricular premature beats during ventricular pacing.
   b. In some patients three premature stimuli are required.
   c. The induced arrhythmia
      i. is sustained in practically all cases
      ii. results in hemodynamic collapse and
      iii. has to be terminated by an external DC shock.
5. Polymorphic ventricular tachycardia or ventricular fibrillation induced by programmed stimulation is a non-specific finding, because these arrhythmias can sometimes be induced in patients with a normal heart. There exist, however, major differences between the two situations:
   a. The clinical context, with symptomatic patients with Brugada syndrome having suffered from spontaneous ventricular arrhythmias
   b. The percentage of patients inducible to a sustained polymorphic ventricular arrhythmia in Brugada syndrome (80%) as compared to individuals without the syndrome where a sustained polymorphic ventricular tachycardia or ventricular fibrillation is only exceptionally induced.
6. The H-V interval is prolonged in about the half of the patients.
   a. The prolongation is not marked, rarely exceeding the 70 ms, but being clearly abnormal in this population with an average age of 40 years.
   b. The H-V prolongation explains the slight prolongation of the P- R interval during sinus rhythm.
7. Hemodynamic studies have been systematically normal.

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Volume 1: Number 13

Let’s Talk About Drugs

Drugs that are PRIMARILY eliminated through the liver (hepatic elimination)

<table>
<thead>
<tr>
<th>Quinidine</th>
<th>Mexiletine</th>
<th>Propafenone</th>
<th>Verapamil</th>
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<tr>
<td>Procainamide</td>
<td>Flecaainide</td>
<td>Moricizine</td>
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<tr>
<td>Lidocaine</td>
<td>Encainide</td>
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Drugs that are PRIMARILY eliminated through the kidneys (renal elimination)

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<th>Disopyramide</th>
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<td>Tocainide</td>
<td>Digoxin</td>
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<tr>
<td>Bretylum</td>
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</tbody>
</table>

Adenosine is eliminated through the vascular endothelium, erythrocytes (red blood cells).

Drug Absorption:

Rule of thumb - any drug that uses a sustained release formula should be avoided in patients with rapid transit times through the gut, such as colostomy patients. Likewise, and perhaps not so obvious (and, therefore, a good test question) timed released drugs should NOT be used in acutely ill patients where absorption may be impaired.

Some factors affecting absorption:

1. Gut flora - especially if the patient is on antibiotics - Digitalis is especially affected
2. Interaction with other drugs

Factors affecting distribution: (volume of distribution is defined as the difference between the dose and the plasma concentration)

1. Heart Failure - reduced volume of distribution
2. Elderly - reduced volume of distribution

For these patients, the dosage for drugs that are rapidly distributed should be reduced.

Half-life - the rate of elimination of a drug from plasma.

Composed of:
1. Distribution Half-Life where the drug is distributed systemically
2. Elimination Half-Life composed of:
   a. Metabolism
   b. Excretion

Antiarrhythmic Drug Interactions

Drugs that increase the digoxin level:

1. Quinidine
2. Flecaainide
3. Propafenone
4. Amiodarone
5. Verapamil

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Beta blocker effects:

1. Additive Negative Inotropic Effect
   a. Disopyramide
   b. Flecainide

2. Increase Beta Blocking effect
   a. Propafenone
   b. Amiodarone

3. Other
   a. Verapamil - additive bradycardia and negative inotropic effect
   b. Lidocaine - beta blockers increase Lidocaine level

Calcium Channel Blocker effects:

1. Additive negative inotropic effect
   a. Disopyramide
   b. Flecainide
   c. Propafenone

2. Other
   a. Moricizine - inhibits diltiazem metabolism
   b. Amiodarone - potential bradycardia

Vaughn Williams Antiarrhythmic Drug Classification

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<th>Type IB</th>
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J Am Coll Cardiol.2001;38:1231

Drugs that affect the Pacing Threshold:

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Drugs that affect the Defibrillation Threshold

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<td>Procainamide no change or ? increase</td>
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</tr>
<tr>
<td>Quinidine</td>
<td>Clofilium</td>
<td></td>
</tr>
<tr>
<td>Flecainide</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

I hope that's enough for today. Please let me know if you want additional info. I don't know of any easy way to memorize all the possible drug effects and interactions. Any suggestions? The drug section seems to cause the most stress for test takers.

Most of the info in this mailing came from Section 5 of the EPSAP. Principal author: Peter R. Kowey, MD

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1Not really. Motivation, intellect and a willingness to spend only 1 minute answering each question are also required. The real purpose of these newsletters is to STIMULATE thought and self-help research. Your comments and suggestions are welcomed.
The simple stuff?

Lead dislodgment

1. Loss of capture  
   a. complete  
   b. intermittent  
2. Change in morphology of captured beats  
3. Change in the dipole (spike polarity)  
4. Change in lead position on repeat CXR  
5. The lead impedance WILL NOT change significantly, in most cases

Signs of GOOD lead placement

1. Adequate "heel" on the intracardiac portion of the pacing lead  
2. 2 to 3 mV "current of injury" on the electrogram  
3. Proper fixation of the anchoring sleeve to prevent lead pullback into the pocket.

Twiddler's Syndrome

1. Most common cause - improperly fixed anchoring sleeve  
2. Other cause - patient twiddling (far less common than #1)  
3. Twiddler's is the most common cause of "Late Dislodgment"

Medscape article: http://www.medscape.com/viewarticle/502680

Exit Block

1. No change in the morphology of captured beats  
2. No change in lead position on CXR

Use of steroids in management of exit block

1. May be effective in about 50% of patients  
   a. Adult dosage - prednisone 60 mg/day in divided dosage  
   b. Test threshold prior to therapy  
   c. Test threshold 4 to 5 days after therapy initiation  
      i. If no change or rise in threshold, stop steroids (they are not working)  
      ii. If threshold has improved, continue  
   d. Continue dose for 1 month  
   e. Test thresholds every other week (biweekly)  
   f. Begin tapering the prednisone dosage  
      i. over a two month period  
      ii. monitor patient closely

Prednisone is a glucocorticoid. Glucocorticoids are adrenocortical steroids, both naturally occurring and synthetic, which are readily absorbed from the gastrointestinal tract.

Most of the information here came from "Cardiac Pacing" edited by Kenneth Ellenbogen. Blackwell Scientific Publications. 1994. Sorry about the age of this info (should not have changed, though). I have not yet purchased the latest edition by Ellenbogen and Kay. It is a massive volume, and IS on my wish list.

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Prefixes

<table>
<thead>
<tr>
<th>Latin prefixes make units smaller</th>
<th>Greek prefixes make units larger</th>
</tr>
</thead>
<tbody>
<tr>
<td>Deci = 1/10  ((10^{-1}))</td>
<td>Kilo = 1000  ((10^3))</td>
</tr>
<tr>
<td>Centi = 1/100  ((10^{-2}))</td>
<td>Mega = 1,000,000  ((10^6))</td>
</tr>
<tr>
<td>Milli = 1/1000  ((10^{-3}))</td>
<td>Giga = 1,000,000,000  ((10^9))</td>
</tr>
<tr>
<td>Micro = 1/1,000,000  ((10^{-6}))</td>
<td>Tera = 1,000,000,000,000  ((10^{12}))</td>
</tr>
</tbody>
</table>

Units of Measurement

<table>
<thead>
<tr>
<th>Unit</th>
<th>Unit of Measure</th>
<th>Definition</th>
<th>Example/Formula</th>
</tr>
</thead>
<tbody>
<tr>
<td>Energy</td>
<td>Joules</td>
<td>A measure of power expended over time. In this sense, one joule (1 J) is equivalent to one watt (1 W) dissipated or radiated for one second (1 s). Result of multiplying volts by current by time.</td>
<td>(E = \frac{V^2}{R} \times T) OR (E = V \times I \times T) OR (E = I^2 \times R \times T)</td>
</tr>
<tr>
<td>Impedance (aka: Resistance)</td>
<td>Ohms</td>
<td>Numerical sum of all resistances to the flow of electrons</td>
<td>(R = \frac{V}{I}) Voltage/Current</td>
</tr>
<tr>
<td>Ampere</td>
<td>Coulombs/sec</td>
<td>An ampere is a unit of measure of the rate of electron flow or current in an electrical conductor.</td>
<td>1 Amp.=1 Coulomb/sec. One ampere of current represents one coulomb of electrical charge moving past a specific point in one second.</td>
</tr>
<tr>
<td>Ampere-hour</td>
<td></td>
<td>A unit of battery capacity. An ampere hour is the amount of energy charge in a battery that will allow one ampere of current to flow for one hour.</td>
<td></td>
</tr>
<tr>
<td>Battery Capacity</td>
<td>Ampere-hours</td>
<td>Units of charge</td>
<td>1 Amp = 1000 milliamps 1 milliamp= 1000 micro amps; therefore 1 Amp= 1,000,000 microamps 1 amp hr. battery=1,000,000 micro amp. Available capacity</td>
</tr>
<tr>
<td>Battery Current Drain</td>
<td>Micro-amps</td>
<td>Relationship between battery capacity and current drain</td>
<td>1 Amp = 1000 milliamps 1 milliamp= 1000 micro amps; therefore 1 Amp= 1,000,000 microamps 1 amp hr. battery=1,000,000 micro amp. Available capacity</td>
</tr>
<tr>
<td>Capacitance</td>
<td>Farads</td>
<td>Relationship between charge stored on caps and voltage applied</td>
<td>Stored energy: (E (\text{Joules}) = 0.5 \times C \times V^2) Delivered energy: (E (\text{Joules}) = 0.5 \times C \times (V_{\text{initial}}^2 - V_{\text{final}}^2)) Note: Delivered energy depends on final voltage (which is dependent upon pts. Defib. Lead imped.)</td>
</tr>
</tbody>
</table>

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<table>
<thead>
<tr>
<th>Unit</th>
<th>Unit of Measure</th>
<th>Definition</th>
<th>Example/Formula</th>
</tr>
</thead>
<tbody>
<tr>
<td>Frequency</td>
<td>Hertz or cycles per second (archaic)</td>
<td>Rate of occurrence of a periodic phenomenon per second</td>
<td></td>
</tr>
<tr>
<td>Power</td>
<td>Watts</td>
<td>Rate at which energy is delivered or absorbed</td>
<td>1 watt=1 Joule/sec.</td>
</tr>
<tr>
<td>Magnetic Field</td>
<td>Gauss: The gauss is the centimeter-gram-second (cgs) unit of magnetic flux density. Gauss is used when expressing the flux density produced by magnets of the sort commonly encountered in consumer products. The tesla is the standard unit of magnetic flux density.</td>
<td>A magnetic field is generated when electric charge carriers such as electrons move through space or within an electrical conductor.</td>
<td>10-70 Gauss (1 Tesa=10,000 Gauss) The gauss is one ten-thousandth of a tesla (1 G = 10^-4 T).</td>
</tr>
</tbody>
</table>

**Formulas**

<table>
<thead>
<tr>
<th>Topic</th>
<th>Definition</th>
<th>Formula</th>
<th>Hints</th>
</tr>
</thead>
<tbody>
<tr>
<td>Slew Rate</td>
<td>Measurement of voltage change over time</td>
<td>dV/dt</td>
<td>Used for measuring intrinsic beats</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Atrium= &gt; 0.5 vS</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Vent. = &gt; 0.75 vS</td>
</tr>
<tr>
<td>Ohms LAW</td>
<td>Relationship of V (voltage), I (current) and R (resistance)</td>
<td>V= IR; R=V/I; I=V/R</td>
<td>See above under energy</td>
</tr>
</tbody>
</table>

**Calculations:**

TARP: Total Atrial Refractory Period

Sum of PV interval plus Atrial Refractory Period (if no sep. programmable PV delay, use AV. Note: If rate response is on, use shortest PV delay in calculations)

- Measured in ms
- When converted to BPM, denotes the pacemaker’s 2:1 Block point

Example: PV=200 ms/ARP=300 ms then TARP = 500 ms. Is PV shortening is allowed to 100 ms, then TARP = 400 ms

Wenkebach Window

Max Tracking Interval (in ms) - TARP (in ms)= ms Wenkebach interval or window

Example: If MTI = 500ms and TARP = 500 ms then device will 2:1 Block at 500 ms (120 bpm)
If MTI = 500ms and TARP = 400 ms, then device will Wenkebach at rates between 120 (500 ms) and 150 (400 ms) bpm
These values are calculated automatically by the programmer.
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Volume 1: Number 16

**Basic Radiation Safety**

**Note:** I'd like to take credit for this one, but it was actually written by Don Nemit. My sincere thanks goes out to him for his efforts. Also, radiation exposure and physics questions are more typical on the EP ExAM.

**Introduction**

This is a general overview of the characteristics, biological effects, and basic calculations involved with x-ray radiation.

**Why is this important to us?**

We are all exposed to x-rays during implants. Exposure to x-rays is a cumulative effect. While a necessary evil, there are safer ways to function under these conditions. Some of this material may be on the test!

**Types of Radiation**

**Non-Ionizing Radiation**

Electromagnetic waves given off by the sun and various electronic devices. Majority of the electromagnetic spectrum, more common than ionizing radiation. Does not have enough energy to cause ionization of atoms.

Types of Non-Ionizing Radiation

<table>
<thead>
<tr>
<th>Ultraviolet light</th>
<th>Visible light</th>
<th>Infrared radiation</th>
<th>Radiofrequency radiation</th>
<th>Microwaves</th>
</tr>
</thead>
</table>

**Ionizing Radiation**

Ionizing radiation is given off by the sun, radioactive materials, and high energy electronic devices (X-ray machines, etc.). All ionizing radiation has enough energy to cause atoms to lose electrons and become ions or charged atoms.

Types of Ionizing Radiation

<table>
<thead>
<tr>
<th>Alpha particles</th>
<th>Beta particles</th>
<th>Gamma rays</th>
<th>X-rays</th>
</tr>
</thead>
</table>

**Characteristics of Ionizing Radiation**

- X-ray radiation is a type of ionizing radiation which is an electromagnetic wave (electromagnetic radiation) or photon and has no mass and no electrical charge.
- X-rays are similar to waves of light in that their intensity is inversely proportional to the distance from the energy source. *The further you move away from the source of the radiation, exposure is decreased by the square of the distance.*
- However, since x-rays have no charge, they cannot be bent such as light waves.
- Therefore, x-rays travel in a linear path until contacting an object.
- Upon contact, x-rays interact at the atomic level generally "knocking out" atomic matter from either the nucleus or from the orbital region.
- Such interaction changes the function of the particular atom altogether. By taking this to a higher degree, if enough radiation is absorbed, cellular/tissue function is altered forever.
- Once a cell or localized tissue is distorted, it is never the same neither in function nor in form.
- Damage at the cellular level is irreparable and irreversible. That is why cumulative dose is important. Biological Effects of Ionizing Radiation
  - Ionizing radiation is known to cause DNA damage, that is, mutation.
  - Consequently, if male or female germ cells or gametes (egg or sperm) have been affected, the damage could be inherited in the next generation.
  - This kind of damage is called a germ cell mutation. Somatic Mutation

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• When a mutation occurs in cells other than germ cells it may be transmitted to descendant somatic cells (of the individual) if the cell undergoes division.
• Example: Liver, skin, or blood cells.
• However, a somatic mutation cannot be transmitted to the next generation of children.

**Cardinal Principles of Radiation Protection**

**Minimize Time**

The dose to an individual is directly related to the duration of exposure. If the time during which one is exposed is doubled, the exposure will be doubled. Exposure = Exposure rate x Time

**Exposure**

Exposure is a measure of source intensity x time.  
Exposure = Exposure rate x time
Exposure rate is measured in milliroentgens (mR).

**Time Exposure Example**

A radiation source has an exposure rate of 225 mR/hr at a position occupied by a radiation worker. If the worker remains at that position for 36 minutes, what will be the total occupational exposure?

Answer. Occupational exposure = (225 mR/hr)(36 min/60 min/hr) = 135 mR

**Distance**

As distance between the source of radiation and a person increases, the exposure decreases rapidly. This is calculated by the use of the inverse square law.

**Distance example**

With an exposure rate of 0.5 mR/hr at 5 ft. = 0.125 mR/hr at distance of 10 ft.

Formula: 0.5mR(5/10) = 0.5mR(0.25) = 0.125mR

**Shielding**

Shielding is necessary for the protection of all individuals involved in x-ray procedures. This includes the patient.  
Example: Pregnant patients, gonadal shielding for individuals of child bearing age  
Primary beam shielding from the x-ray source is achieved through the use of Aluminum plate shields.  
This contains the beam to its point of focus attenuating unnecessary exposure and also provides for a greater quality image.  
Personal shielding is achieved through the use of lead or lead equivalent aprons.  
Lighter (in weight) aprons provide adequate protection for use in short duration x-ray exams (chest x-rays, etc.)  
However, such material is not as efficient as lead aprons during fluoroscopic exams/procedures.

**Terms and Definitions**

**ALARA**

As Low As Reasonably Achievable  
The concept of maintaining radiation exposures at their lowest level to achieve a usable x-ray/fluoroscopic image.  
2 Inverse Square Law: I1/I2 = (d2/d1)

**Photon**

Energy "packets" which have no mass and no charge.  
Travel at the speed of light.  
Considered energy disturbances in space.
Rad (Radiation absorbed dose)

A unit of absorbed dose.
One rad is 0.01 Joule absorbed per kilogram of any material.
Being replaced by the gray (Gy)
One rad equals one hundredth of a gray.

Radiation
The emission and propagation of energy through matter or space by means of electromagnetic disturbances.
Display both wave-like and particle-like behavior.
In this context the “particles” are known as photons Radiation (Ionizing)
Any electromagnetic or particulate radiation capable of producing ions, directly or indirectly, by interaction with matter.
Examples are x-ray photons, charged atomic particles and other ions, and neutrons

REM
Roentgen Equivalent Man
The unit of dose equivalent or occupational exposure.
Used to express the quantity of radiation received by radiation workers (1 rem equals 1.07185 roentgen.).

Roentgen: the amount of radiation required to liberate positive and negative charges of one electrostatic unit of charge in 1 cm³ of air at standard temperature and pressure (STP).

The roentgen (R) is the unit of radiation exposure or intensity.
The output of x-ray machines is expressed in Roentgens or milliroentgens (mR)
The roentgen only applies to x-rays and gamma rays and their interactions with air.

Scatter Radiation
Phenomena in which an incident photon interacts with a target atom, causing it to become excited.
Target atom immediately releases this excess energy as a secondary, or scattered, photon with wavelength equal to that of the incident atom.
The direction of the secondary photon is different from that of the incident photon.

Scatter Radiation Formula
There is no exact formula for calculating scatter from a patient.
A “rule of thumb” does exist in that the scatter at 1 meter from the center of a patient field is approximately 0.1% of the primary beam dose rate at 1 meter from the center of the beam.

Scatter Example
If the exposure rate in the center of a primary beam were 1 R/min., the scattered exposure rate would be 0.1 R/min. at 1 meter from that point.
The inverse square law does apply here.
Review Questions

1. The output intensity of a radiographic unit is 4.2mr/mAs. What is the total output following a 200ms exposure at 300mA?
   Answer: 4.2mR/1mA s = x/60mA s x=252mR

2. What is the approximate patient skin dose following a 3.2-minute fluoroscopic examination of 1.5 mA?
   Answer: Exposure=(ExposureRate)(time) (1.5mA)(3.2 min)=xmA s (1.5mA)(192s)=288mA s

3. At 60 cm from the side of a fluoroscopic table (assume 90 cm from a point source) the exposure rate is 50 mR/hr. What is the exposure rate at 180 cm from the source?
   Answer: I1/I2=(d2/d1) 2 50 mr/hr /x = (180/90) 2 50 mr/hr = x(2)
   x=12.5 mr/hr

4. If an individual remained 30 cm from the table (60 cm from the source) for a total of 150 minutes of beam-on time during a week, what would be the expected personnel exposure?
   Answer:
   I1/I2=(d2/d1) 50 mr/hr /x = (60/90)
   50 mr/hr = x(.444)
   x = 112.5 mr/hr
   (112.5 mR/hr)(150 min)(1hr/60 min)=112.5 mR/hr)(2.5 hr)= 281.25 mR

5. What exposure will an individual receive when exposed for ten minutes at 4 m to a source with intensity of 100 mR/hr at 1 m while wearing a protective apron equivalent to 2 HVLs?
   Answer: 100mR/x=(4/1) = 6.25 mR/hr 2 [(6.25mR/hr)(10 min)(1hr/60 min)]/2 2 = 1.0416662/2 = .2604

6. The exposure from an x-ray tube operated at 70kVp, 200 mAs is 400 mR at 36 in. What will be the exposure rate at 72 in?
   Answer I1/I2=(d2/d1) 2 I1 = I2(d2/d1) 2 I1 = 400 mR (36in/72in)
   I1 = 400 mR (1/4)
   I1 = 100 mR

7. A radiographic unit shows patient skin dose to be 5.4mR/mAs at 70Kvp. What will be the appropriate skin dose following a technique of 84 Kvp, 120 mAs?
   Answer: I1/I2 = mAs1/mAs2(Kvp1/Kvp2) 2 x/5.4 mR/mAs =120mAs/1(84/70) 2 x/5.4 mR/mAs = 120 mAs (1.2)
   x = (5.4 mR/mAs)(120 mAs)(1.44)
   x = 933.12 mR/mAs
Today's topic is, pure and simple, the AV Delay interval as it applies to dual chamber pacemakers and ICDs.

When I speak of the "AV Delay," I mean the paced atrial to paced ventricular event. Sensed event timing will be discussed later.

The AV Delay interval begins when the atrial output escape interval (aka: pacing interval or atrial escape interval or AEI) is allowed to time out (nothing is sensed on the atrial channel). The device output first occurs on the atrial channel (hopefully causing depolarization of the right atrium). The period of time between the atrial output pulse and the ventricular output pulse is known as the AV Delay interval. This interval may be fixed or variable. During this interval, the atrial channel of the device is refractory (does not sense). The AV Delay interval is the first part of the Total Atrial Refractory Period (TARP). When the AV Delay interval times out without sensing an intrinsic ventricular event, a ventricular output pulse is delivered. NOTE: the ventricular output pulse CANNOT be delivered unless some atrial event occurs to start the AV Delay. In DDD pacing the ventricular output will never occur unless an AV Delay interval is started by some sort of atrial event. Atrial events include, but are not limited to, atrial output pulses, P waves, far field R waves, noise, make break signals, etc. These events may not be obvious on the surface ECG, but must occur in order to generate a ventricular output spike.

When AV pacing is present, in the non-rate responsive mode, the Atrial rate will always be at the base programmed rate.

In the rate responsive mode, an atrial rate at base rate indicates the sensor is not driving the device. If the atrial rate is faster than the programmed base rate, sensor drive is present. Some devices allow the user to enable a "rate responsive" AV interval that automatically shortens the AV delay interval during sensor drive. This feature is designed to mimic the way the heart shortens the intrinsic PR interval as the heart rate increases.

When performing a follow-up of a patient who is AV pacing:

1. To reveal intrinsic P waves, reduce the base rate
2. To reveal intrinsic R waves, increase the AV Delay or reprogram the device to a single chamber ventricular pacing mode, such as VVI, and reduce the rate until R wave become visible. Reducing the pacing rate in the DDD mode, reduces the ATRIAL pacing rate but usually does not affect the AV conduction to the point that R waves will emerge.
The PV Delay interval is another rather simple timing concept that must be thoroughly understood for a few questions on the ExAM. The PV Delay interval is also known as P Tracking Interval and PV Interval. It may be a fixed or variable value.

Modes that incorporate the PV Interval are DDD(R), VDD(R), DDT(R), and VAT. DDT and VAT are rare and will not be discussed.

When the atrial output escape interval (AEI), is interrupted by a sensed event on the atrial channel, the PV Delay interval begins. Note that I said a SENSED EVENT and not P Wave. Far field ventricular events and extraneous noise events MAY be sensed on the atrial channel and will start the PV Delay. When an event is detected, the atrial channel becomes refractory, a certain amount of time is allowed to elapse and the ventricular output is generated. An R wave or other perceived ventricular event will stop the PV interval to result in inhibition of the ventricular output pulse.

On the ExAM it will be important to note the relationship of the P wave to the ventricular output spike. Keep in mind that the P to V interval should not lengthen as the rate increases. Progressive lengthening of the PV Interval may indicate a condition known as pacemaker Wenkebach. Pacemaker Wenkebach is a NORMAL function in dual chamber pacing and occurs when the sensed P rate is faster than the programmed maximum tracking rate (or upper rate interval). In DDD pacing, P tracking occurs from rates just above the programmed base rate to the upper tracking rate limit. Rates at or below base rate should appear as AV or AR pacing.

Here’s where you need to be careful: in years past, at least a few questions concerned basic VDD timing. Remember, unlike DDD timing, there is NO atrial output pulse possible with VDD mode. If a rhythm strip showing P tracking at rates above base rate has episodes with what appears to be VVI (non-P Synchronous) at low rates, expect VDD as the mode (See GtP #11 for details). The rule you must remember is: in DDD mode, a ventricular output cannot occur unless an atrial event (sensed or paced) precedes it. In VDD pacing, if the atrial rate drops below the base rate, the mode will appear to be VVI.

Another idiosyncrasy of VDD pacing, although slightly off topic, is the behavior of below base rate ventricular pacing when the P rate is very near the programmed base rate. Here’s an example: if the device is programmed to 60 ppm (1000 ms) with an AV delay of 200 ms, and the sinus rate is 61 bpm (984 ms), the V - V interval will be 1184 ms (or about 51 ppm). This happens because the AV Interval is started when the P wave is sensed and delays the ventricular output. Below base rate ventricular pacing is NORMAL in VDD pacing.

As you can see from this short note, understanding of the NORMAL behavior of devices is mandatory.

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I thought I would spend a few minutes discussing the "AR" interval, today. The AR Interval begins when the atrial escape interval (AEI) is allowed to "time out" completely. An atrial output occurs at the end of the AEI, the AV Delay Interval is started but is interrupted by a sensed ventricular channel event (usually an intrinsic R wave). Pretty simple. Well, maybe not.

There are two types of pacemaker timing schemes: ventricular based timing and atrial based timing. In ventricular based timing, the sensed R wave causes the atrial escape interval timer to start. This means that the next atrial output is based on when and where the R wave (or other ventricular channel event) is sensed. The clinical ramification of this is, whenever the AR state is present, and the timing is ventricular based, the next atrial output will occur at a rate that is faster than the programmed base rate. For example, if the AV Delay Interval is programmed to 200 ms and the sensed R wave occurs at 150 ms, the next atrial output will occur 50 ms earlier than the calculated base rate. For a base rate of 60 ppm (1000 ms) the atrial output will occur at 950 ms causing the rate to be measured at about 63 ppm (calculated by measuring A spike to A spike). If the timing is Atrial based, the AEI timing begins with the atrial event.

In the presence of PVCs, Atrial Based Timing reverts to Ventricular Based for one cycle. Following the PVC, the next atrial output is scheduled at the pacing interval minus the AV Delay interval. In a device programmed to 60 ppm (1000 ms), with an AVD of 200 ms, the PVC would initiate a clock cycle of 800 ms for the next atrial output pulse.

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The final state of DDD pacing (in case you did not know, the 4 states are: AV, PV, AR, PR) that I'll be describing is the "PR" or "inhibited" state. In this state, the atrial escape interval (AEI) is interrupted by a sensed event on the atrial channel, a PV interval begins but is stopped by a sensed ventricular event. No pacing occurs, the device remains inhibited. This is the "normal" way the PR interval occurs.

A normal, but anomalous appearing PR interval can occur at fast sinus rates. Let's say the patient has first degree heart block with a long PR interval, and the pacemaker is set up with an AV interval of 175 ms and a maximum tracking rate (a.k.a. Upper Rate Limit) of 110 ppm (545 ms). If the sinus rate is greater than 110 bpm the P wave will be sensed, the PV interval will start, but at the end of the PV interval the ventricular output will not occur because the Upper Rate Limit of Ventricular Pacing is set for 110 ppm. This means that the ventricular channel cannot output a spike until the upper rate interval timer completes its cycle. If an intrinsic R wave occurs before this cycle is completed, the V output will remain inhibited and one will see a P wave followed by an R wave at an interval GREATER THAN the programmed AV or PV delay.

Notice how the Upper Rate Limit (URL) is interrupted by the sensed R wave. Since it is never allowed to complete, ventricular outputs are not permitted. On the ECG recording, the rhythm looks like 1st degree heart block at a rate greater than the programmed upper rate of the device.

The 4 States of DDD Pacing

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1Yeah, right, and maybe I could interest you in some prime swamp real estate in Florida, too. The real purpose of these newsletters is to STIMULATE thought and self-help research. Your comments and suggestions are welcomed.
Volume 1: Number 21

Easy to Remember Longevity Calculation/Some Q&A

Here's an FYI on a "rule of thumb" to use for longevity. It may be desirable for those who are "algebraically challenged" or experience "exam lockupitis". For device longevity the "magic number" is 114. That's all I try to remember. The formula is

\[ 114 \times \frac{(Ahr\ battery\ capacity)}{(current\ drain\ in\ uA)} = Longevity\ (yrs) \]

That way one doesn't have to work with algebra during the exam.

Next, some Q & A...

1. Do you know what Ashman's Disease or Syndrome is?

NOTE: I'm not sure if the following answer completely answers the question. If anyone has a better or more appropriate answer, I will be happy to publish it.

Atrial fibrillation is usually a narrow-complex tachycardia in which the rhythm is "irregularly irregular", P waves are not consistently seen, and untreated patients typically have ventricular rates > 100 beats/minute. Occasional wide complex beats with a right bundle branch morphology are due to aberrant conduction and are called "Ashman's beats." They are not VPCs and do not imply increased risk.

Here is a good definition of Ashman's Phenomenon: "Ashman's phenomenon occurs in sinus rhythms as well as in atrial fibrillation. It's simply a predisposition to both ectopy and aberration when a longer R-R interval precedes a shorter one. The bundle branches (especially the right) reset their repolarization time for the longer period and are caught off guard by the shorter cycle length. This often causes aberration through the bundle that hasn't repolarized. The same situation predisposes the patient to bigeminal rhythm by allowing the ectopic focus time to increase its spontaneous automaticity."

2. Do you know what a Bisping Coaxial Lead is?

Bisping refers to an extendable/retractable helix type lead. The helix (or screw) is active. The Bisping mechanism patent is owned, I believe, by Medtronic. Essentially, any extendable/retractable lead uses the Bisping patent for which royalties are paid (I believe).

3. When looking at an X-Ray, how can you determine Dextrocardia vs. a flipped X-Ray?

Usually by the x-ray marker. The "L" or "R" will be reversed if the film is flipped. Also, check out the "gastric bubble." It may be on the left with a plain vanilla dextro but, it could be on the right with a full blown situs inversus. Go here for a looksee and a fairly full description:

http://www.bcm.tmc.edu/radiology/cases/pediatric/text/6a-desc.htm

The first part of this answer may seem a little goofy, but I have heard that at least one of the CXR identification questions uses a CXR with a reversed "L" or "R" - please be careful when viewing the images.

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Volume 1: Number 22

Lead Insulation Failure

Keep in mind the two types of pacing leads: unipolar, consisting of an outer insulator and a conductor (the cathode); bipolar consisting of an outer insulator, an outer conductor (the anode), an inner insulator, and an inner conductor (the cathode).

In a unipolar system when the outer insulator, by erosion, incision, accident or other, becomes compromised, the patient may present with any (or all) of the following:

1. Muscle stimulation (near the site of the insulation break)
2. Myopotential inhibition
3. Myopotential tracking
4. Reduced lead impedance (may be dramatic)
5. Noisy electrogram (if the pacing device has IEGM capability)
6. Possible attenuation (damping) of the pacemaker artifact
7. Capture threshold may appear to increase
8. Ventricular lead insulation defects above the tricuspid valve may result in P wave sensing.

In a bipolar system, outer insulator defects may appear as:

1. All of the above except #6
2. Unipolarization of the output spike (larger than normal spike)
3. Programming the device to unipolar sensing/pacing should eliminate the effects.

If the inner insulator should become defective

1. "Make/break” signals may be visible on the IEGM
2. Pauses due to detection of the make/break signals may occur
3. Spikes may be attenuated due to short circuiting
4. Capture threshold may appear to rise

Keep in mind that all of the above can be constant or intermittent. Since many of the effects of a lead insulation defect and occur with other device conditions, getting a chest X-ray can be very important. In some cases of intermittent lead insulation defects, a Holter monitor may be required to appreciate the condition.


A United States General Accounting Office (CAO) report cited the bipolar coaxial design and Pellethane® 80A insulation as factors associated with a high risk of lead failure. We reported the incidence of inner insulation failure of the Pacesetter 1016T and 1026T leads (Pacesetter, Inc, Sylmar, CA, USA) and the possible contributory role of compression by retention sutures over a thin anchoring sleeve. Others have suggested that "crush" injury caused by lead entrapment by the scalenus muscle or costoclavicular ligament may be important, suggesting a benefit of cephalic or axillary venous access. Hayes et al., however, found no differences in the incidence of failure between leads inserted via the cephalic versus direct subclavian venous approach.

From: Brinker, Jeffrey, PACE, Vol. 18, pp. 953-4; May 1995

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P Wave Oversensing from a Ventricular Lead Insulation Defect

This was reported by van Gelder, et al, in the February 1995 issue of PACE (Vol. 18).

P wave sensing, in VVI pacemakers, always has been associated with bipolar leads in case of endocardial stimulation even with the lead properly positioned in the right ventricle. P wave sensing under these conditions, is caused by the position of the proximal electrode in the vicinity of the tricuspid valve area. In our patient, a different mechanism appears involved, because the lead had functioned properly for 20 years. We assumed that P wave oversensing was caused by an insulation defect in the ventricular lead at the level of the high right atrium, most likely caused by friction between the abandoned lead and the functioning lead. Arguments for this hypothesis are the following.

1. ECG interpretation channel telemetry confirms P wave sensing.
2. The morphology of the P wave recorded from the unipolar ventricular lead showed a negative P wave with a sharp initial deflection. The sharp initial deflection occurs simultaneously or even just before the onset of the P wave in the surface ECG. This implies that the P wave is recorded in the high right atrium.
3. A decrease in lead impedance from 540 Ω to 390 Ω is suggestive of an insulation defect.
4. An insulation defect at high right atrium level is confirmed by chest wall stimulation.
5. Fluoroscopy revealed movement of both leads touching each other at the high right atrium level.
6. Retraction of the lead failed because the locking stylet could not be advanced further than the intersection of both leads, also at the level of the high right atrium. This can be explained by penetration of blood the conductor coil enabled by an insulation defect at this place.
7. Because P waves are sensed at the onset of the P wave in the surface ECG, oversensing bears no relationship to atrial contraction, thereby excluding oversensing of spurious signals generated by intermittent contact of the functioning electrode with an insulation defect and an inactive lead.
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Lead Fractures

As with yesterday's discussion, most of the info in this post comes from "Cardiac Pacing" edited by Kenneth Ellenbogen. The Chapter entitled "Differential Diagnosis, Evaluation, and Management of Pacing System Malfunction" by Paul A. Levine, MD. Blackwell Scientific Publications.

In his discussion, Dr. Levine appropriately titles the section dealing with lead fractures, OPEN CIRCUITS. The truth is, many times what is assumed to be a "lead fracture" turns out to be something else. That "something else" could be an improperly tightened set-screw, or a device well past EOL and, hence, no output. For this report, I'll try to stay on topic and discuss only lead fractures.

Lead Fractures may demonstrate themselves as constant or intermittent events. It is possible for a complete lead fracture to have each end of the wire remain well proximated, resulting in intermittent contact, thus, intermittent capture.

Bipolar wires: When the inner coil (cathode) fractures, the result is an open circuit. No pacing will occur, no sensing will occur. Reprogramming the device to unipolar WILL NOT help.

When the outer coil (anode) fractures, the result is still an open circuit, however, reprogramming the device to unipolar may help. Programming to unipolar makes the pacemaker can the anode. If the fracture is caused by stress from the suture, the inner insulation may open causing a short circuit between the anodal and cathodal wires - result...no pacing, no sensing (or if the defect is intermittent, intermittent capture and sensing in unipolar configuration).

Diagnosing the problem.

1) Get a Chest X-ray (AP and Lateral)
2) Fractured unipolar wires are easier to see than bipolar wire fractures.
   a) Fractures may not always appear on CXR (especially on bipolar leads)
   b) Take a look at the device based electrograms (if available)
3) Look for make/break signals
4) Look for total signal drop-out

Be very careful with provocative testing using arm and/or shoulder motion - an intermittent fracture could become a complete fracture leaving the device dependent patient with NO rhythm!

"The usual sites of transvenous lead fracture are: at the site of venous entry (40%), between the site of venous entry and the generator (28%), and close to the lead insertion into the generator (23%), but only 7% of fractures are intravascular." From: Alt E, Volker R, Blomer H. Lead fracture in pacemaker patients. [Thorac Cardiovasc Surg 1987; 35:101-104."

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The two images at the left illustrate “pseudo fractures” of ICD leads. Pseudo fractures are not limited to ICD leads, in some bifurcated bipolar pacing leads, similar ‘apparent’ discontinuity of the coil is commonly seen on chest x-ray.

I’ve presented these examples on normal leads that appear damaged, just in case similar images appear on the exam.


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Volume 1: Number 24

AMS vs Fallback

Automatic Mode Switching (AMS)

AMS uses an algorithm to determine when the atrial rate exceeds a predetermined value, say 170 beats per minute. When the atrial rate (or, more likely, the filtered atrial rate) reaches the magic number, the device switches from a tracking mode (DDD or DDDR) to a non-tracking mode such as DDI(R) or VVI(R). The device will remain at the mode switched mode until the atrial rate (filtered) drops to (or below) the upper tracking rate of the pacemaker, at which time it reverts to the tracking mode. At rates between the upper tracking rate and the mode switch rate, the device will exhibit pacemaker Wenkebach behavior until it reaches the 2:1 block point, it will then begin 2:1 behavior. This could result in a sudden drop in the pacing rate. NOTE: some devices allow the physician to select a "mode switch base rate." This is a rate higher than the normal brady base rate of the device, so the sudden switch to a non-tracking mode will not be so noticeable to the patient. The atrial rate continues to be detected and AMS will occur when the Atrial Rate reaches the AMS rate. AMS is primarily used in patients who are prone to atrial arrhythmias such as atrial fibr and atrial flutter.

Fallback

Fallback is not associated with mode switching during an episode of atrial tachycardia. The main objective of fallback function is to avoid rapid and prolonged ventricular pacing initiated by the sensing of atrial arrhythmias. In normal DDD mode, the maximum ventricular rate initiated by sensed atrial arrhythmias is equal to the lowest rate lying between the upper rate limit (URL) and the 2:1 point. The latter limit corresponds to the total atrial refractory period (TARP), which is the smallest possible AV synchrony interval. **Fallback is used to prevent a sudden rate drop when the atrial rate exceeds the upper rate limit of the device.** For example, lets say that a device is programmed to a 200 ms AV delay and a 300 ms post-ventricular atrial refractory period. This makes the total atrial refractory period equal 500 ms (or a rate of 120 bpm). Lets also say that the device is programmed to a upper rate limit of 120 bpm. What happens when the sinus rate reaches 120? Answer: 2:1 pacer block with an effective pacing rate of 60 ppm (assuming the lower rate is programmed to 60). Not a good thing. Patient symptoms could occur. Fallback prevents the sudden rate drop to base rate by slowly and progressively slowing the ventricular paced rate. It disassociates the ventricular output from the sinus rate until the sinus rate drops to the upper rate limit or the Fallback rate. It continues to track the atrial rate, but at longer and longer AV intervals. Fallback is used in patients who are prone to fast atrial rates (not atrial tachycardias) and who would otherwise find themselves experiencing slow ventricular rates at inopportune moments.

Ela Chorus behavior of the fallback function upon sensing of an isolated premature atrial contraction (PAC). The pacemaker initiates a window of atrial rate acceleration detection (WARAD) equal to 75% of the preceding P-P interval upon sensing of a sinus event. The pacemaker reinitiates an atrial escape interval (AEI), but no AV delay on any atrial event sensed during the WARAD. At the end of this interval, AV pacing is delivered with a 3I-ms AV delay designed to raise the 2:1 point of atrial sensing to a maximum.

An alternative to Fallback is proper programming of the sensor in rate responsive devices. If the sensitivity of the sensor is properly setup, sensor drive response lags just below the native sinus rate. This provides a pseudofallback function with the advantage of sensor drive during periods of activity.

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Rate Smoothing

The following information comes from an old (very old) issue of PACE. I believe it to be accurate, but I am not certain if modern Guidant/Boston Scientific devices use exactly the same algorithm.

Rate smoothing values (RSV) may be set at 3%, 6%, 12.5%, or 25%. When the rate smoothing option is on, the pulse generator stores in its memory the most recent RR interval and does not allow the next RR interval to be less than the preceding RR interval minus the programmed percentage nor does it allow it to be greater than the preceding RR interval plus the programmed percentage.

Based on the previous RR interval and the elected smoothing value, the pulse generator sets up two windows—one for atrial events and another for ventricular events:

\[
\text{atrial window} = (\text{previous RR interval} \pm \text{RSV}) - \text{AV delay}
\]

\[
\text{ventricular window} = \text{previous RR interval} \pm \text{RSV}
\]

The timing for both windows is initiated at the end of a ventricular event. When an atrial vent occurs before the atrial synchronization window (e.g., APB), the ventricle is paced after interval that equals the previous RR interval RSV. When an atrial event occurs within the atrial synchronization window (e.g., sinus beat), the programmed AV delay period is initiated and the ventricle is paced at the end of the AV delay period. When no atrial event occurs during the time represented by (previous RR interval + RSV) - AV delay, the atrium is paced at the end of this interval and the ventricle at the end of the programmed AV delay period. When a ventricular event occurs before the ventricular window (e.g., VPB), the timing circuit is reset at an interval equal to the previous RR interval - RSV and a new cycle begins. When a ventricular event occurs within the ventricular synchronization window, ventricular output of the pulse generator is inhibited and the next RR interval will be equal to the previous RR interval ± RSV.


From “New Perspectives in Cardiac Pacing” edited by Serge Barold: “The fact that a rate-smoothing algorithm will not allow a rapid change in ventricular rate can easily result in VA conduction. Whenever a “fast” atrial beat comes along, the ventricular response is delayed until the rate-smoothing algorithm can be satisfied. If, for instance, the last R-R interval was 1000 msec and the rate-smoothing algorithm was set to 6%, the shortest R-R interval allowed would be 1000 minus 6% (1000) = 940 msec. If the atrial event happened to occur at a 600 msec rate, then the AV delay would be 340 msec. With a 340 msec AV delay, it is quite likely that VA conduction and subsequent PMT could occur. This PMT would be unusual because it would start slowly and gradually speed up (6% per cardiac cycle) until it reached either the absolute upper rate limit or the sum of the programmed AV delay plus the VA conduction time.”

In both NASPExAMs I’ve taken, there have been questions relating to the definition of “Fallback” and “Rate Smoothing.” Additionally, if I recall correctly, there have been a couple (maybe only 1) of questions showing either Rate Smoothing or some form of Fallback on a rhythm strip. Remember, the exam is a general knowledge test. It assumes you walk into the room with some knowledge of a great many devices from several manufacturers.
Chagas Disease or “Let me introduce you to my little friend!”

This may only be worth one question on either the AP or the EP exam, but what the heck, you may need the answer to pass.

FYI, the vector for this disease is an insect, hematophagous assassin bugs of the subfamily Triatominae, also known as the "Kissing Bug." This insect bites the patient, usually at night, and deposits feces near the bite. The feces contain the parasite Trypanosoma cruzi that is rubbed into the bite by the victim. The disease is most prevalent in Central and South America, although it is becoming more common in North America. In case you think this is not an important disease read this:

"Profound economic and social changes in the last four decades are stimulating rural-urban migration in most of endemic areas, with more than 60% of the population presently settled in urban centers. It is estimated that, because of migration, about 300,000 infected individuals are living today in the city of São Paulo and more than 200,000 in Rio de Janeiro and in Buenos Aires. In addition, chagasic patients are migrating northward to the USA and even eastward to Europe: nowadays, around 100,000 infected individuals are living in the USA, most of them immigrated from Mexico and Central America." From: http://www.dbbm.fiocruz.br/tropical/chagas/chapter4.html

The following quote was found at: www.thirdworldtraveler.com/Disease/Chagas.html - go there for more info. Better still, go to google.com and do a search using the term "chagas disease" for a huge database on this disease.

"In adults, an acute infection of the heart is the main result, damaging the heart muscle. Most of the victims of Chagas' disease survive the acute heart infection, the symptoms subside within four to eight weeks, and the person continues to live an apparently healthy life. The disease however progresses and goes on unrecognized, remaining undiagnosed until a routine blood test may disclose it. The disease finally surfaces after 10 to 20 years in the form of chronic heart disease, as the infected heart muscle fibers are slowly replaced by scar tissue, thinning the walls of the heart, severely affecting heart function, and ultimately resulting in death. The nervous system may also be affected causing convulsions, paralysis, and brain damage."

Typical clinical features of Chagas' disease in a 42-year-old man from rural Brazil who presented with palpitations. (A) Electrocardiogram showing right bundle-branch block, left anterior hemiblock, anterior Q waves, and frequent premature ventricular contractions. (B) Two-dimensional echocardiogram showing an apical left ventricular aneurysm. (From: http://www.clinicalcardiology.org/productcart/pc/briefs/200012briefs/cc23-883.review.html)
Back in the day (2002), unipolar left ventricular (nee: coronary sinus) leads were the only option for cardiac resynchronization therapy (CRT). In the December 2003 issue of PACE (PACE, Vol. 26, pp. 2264-71) Mayhew, et. al. from The University of Alabama at Birmingham, published a report entitled “Electrical Characteristics of a Split Cathodal Pacing Configuration.” In this article, “split cathodal pacing” is described as a parallel circuit using the tips of the RV and LV leads as the common cathode.

The study found that the total impedance measured with a pacing system analyzer in the bipolar split Cathodal configuration was consistently greater than would be predicted by a parallel circuit using the impedances of the RV and LV leads where the total impedance ($R_T$) is given by the equation $1/R_T = 1/R_{LV} + 1/R_{RV}$. For example, using the bipolar configuration, the mean LV lead impedance (vs the RV ring electrode) was 874Ω, the bipolar RV impedance was 705Ω, and the predicted total impedance of these leads in parallel would have been 390Ω. This compares with a measured impedance in the bipolar split cathodal configuration of 516Ω. Although some variables that determine impedance remain constant when two cathodes are combined, the size and shape of the cathode is dramatically changed. While the size and shape of the individual electrodes are unchanged, the combined cathode presents a different effective electrode size, surface area, and (presumably) current density that influence both impedance and threshold.

The study concluded: “A split cathodal configuration increases the apparent stimulation threshold for the LV and RV. The pacing threshold is further increased by programming from the unipolar to the bipolar split cathodal configuration.”

In the “ExAM” world, this means the capture threshold increased and the lead impedance decreased.

**Serial Connections:** In electrical terms, a serial connection (or, more accurately a connection in series) uses a single impedance to determine the output current. When you have one lead, such as the RV lead, delivering the current (mA), you are using a serial connection. The current path in a bipolar lead is lead tip (negative or cathode) to ring (positive or anode). In a unipolar connection the ‘can’ is the anode. When you know the impedance, you can easily calculate the delivered current (mA) by using Ohm’s Law (mA = voltage/impedance). The single impedance is actually a sum of the impedances. For example, if the RV impedance is 500 ohms and the LV impedance is 600 ohms, the measured serial impedance will be 1100 ohms which may result in a slightly higher capture threshold.

**Parallel Connections:** Parallel connections occur when there are two independent impedances. In parallel circuits, the sum of the output CURRENT is used to determine the final lead impedance. Here are two examples:

<table>
<thead>
<tr>
<th>Output voltage = 2.5 V</th>
<th>Output voltage = 5 V</th>
</tr>
</thead>
<tbody>
<tr>
<td>LV impedance = 700 ohms</td>
<td>LV impedance = 700 ohms</td>
</tr>
<tr>
<td>RV impedance = 400 ohms</td>
<td>RV impedance = 400 ohms</td>
</tr>
<tr>
<td>Using Ohm’s Law...</td>
<td></td>
</tr>
<tr>
<td>2.5V/700 Ohms = 3.6 mA</td>
<td>5V/700 Ohms = 7.1 mA</td>
</tr>
<tr>
<td>.5V/400 ohms = 6.3 mA</td>
<td>5V/400 ohms = 12.5 mA</td>
</tr>
<tr>
<td>3.6 mA + 6.3 mA = 9.6 mA @ 260Ω</td>
<td>7.1 mA + 12.5 mA = 19.6 mA @ 255Ω</td>
</tr>
</tbody>
</table>

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A term to remember: Pulse Droop

"Pulse Droop" and is caused by the inability of a device's output capacitors to handle high current drain efficiently. Pulse Droop is the difference between the leading edge voltage of the output waveform and the trailing edge voltage (similar to "tilt" when discussing ICDs). When the current is high (impedance low), the trailing edge voltage may be only 1/2 (or less) the amplitude of the leading edge voltage. This will result in HIGHER capture thresholds from the device since the delivered current will be less as compared to the capture threshold determined by the PSA. Remember, the capacitors within the PSA are far larger than the capacitors in the pacemaker and, thus, can handle the increased demand of low current drain without the effect of pulse droop.

All first generation CRT-P devices used a parallel circuit for connecting the leads to the output stage of the pulse generator.

In real terms, most of this information may seem to be of little importance in day to day practice. However, the examiners at the Heart Rhythm Society may take a different view when one considers the ExAM is designed to be an evaluation of overall knowledge of pacing, defibrillation and all the implied electrical aspects of each.

Diagram to the right is a simplified example of a Series Circuit.

Diagram to the left is a simplified example of a parallel circuit. It is important to try to remember these circuits as examples in diagram form sometimes appear on the exam.

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Take a good long look at this strip…

Can you identify this rhythm? The mode is DDD, the base rate is programmed to 70 ppm (857 ms), the AVD is 165 ms. What is happening here?

This is an example of pacemaker crosstalk. In today’s world, this presentation of crosstalk has been virtually eliminated by the use of algorithms that prevent the inhibition of the ventricular output in the presence of noise from the atrial channel. The algorithms are called by several names, including, Ventricular Safety Standby (VSS); Ventricular Safety Pacing; non-physiologic AV delay, etc.

**Crosstalk defined:** Crosstalk is defined as the inappropriate detection of a pacemaker-generated event in one channel by the sensing amplifier of the other channel that causes inhibition of the second channel’s output. Most commonly, the atrial output is detected by the ventricular channel.

Here is the annotated version of the rhythm above…

**Box 1:** Atrial output occurs. The AVD begins, but is stopped by the presence of crosstalk (sliver of red after pacer spike). Crosstalk starts both the ventricular refractory period (VRef) and an atrial escape interval. After the completion of the VRef, an intrinsic R wave is sensed, stopping the original AEI and then resetting a new Atrial Escape Interval (note: this is ventricular based timing). **Note the A-A interval is considerably slower than the programmed rate.**

**Box 2:** Atrial output occurs. The AVD begins, but is stopped by the presence of crosstalk. Crosstalk starts both the ventricular refractory period (VRef) and an atrial escape interval. This time, the intrinsic R wave occurs within the VRef where it is not sensed, thus allowing the AEI to complete and allowing an atrial output to occur. **Note the A-A interval is considerably faster than the programmed rate.**

This patient is fortunate he/she has an underlying intrinsic rhythm. Crosstalk that occurs in truly pacemaker dependent patients can be fatal.

**What are the causes of crosstalk?** In general, crosstalk occurs when the atrial output is at a high value (usually >5 volts), the ventricular sensitivity is high (usually <1 mv). Programming a lower atrial output or lower ventricular sensitivity (if possible) may correct this condition. Unfortunately, it is not always possible to provide adequate programming.

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Additionally, crosstalk is seen when the insulation has been rubbed away from two leads in very close proximity and the energy is transferred to the ventricular lead at the time of an atrial output. In this occurrence, the leads must be replaced to prevent the crosstalk from occurring.

From time to time, interrogation of a device with no leads attached may demonstrate crosstalk due to the absence of load on the output stage of the device. This is usually corrected automatically with a load (impedance) is applied to the circuit, and should not be of concern.

Automatic preventative methods

Fortunately, device manufacturers have long recognized Xtalk as problem in dual chamber devices. All modern pacemakers are equipped with automatic systems to detect and provide ventricular back-up pacing when the condition occurs.

While the actual algorithm varies from company to company, the principles are the same. The device provides a detection window immediately after the atrial output pulse. If Xtalk noise occurs within this window, the device resets the AVD interval to approximately 120 ms (varies w/company). At the end of the shortened AVD a ventricular output is issued. Some devices allow a return to normal sensing after the crosstalk detection window closes. This allows inhibition of the non-physiologic RV output.

A general rule of thumb for interpreting rhythms on the exam would be to look for AV Delay intervals in the range of about 100 to 120 ms.

1. If the test question suggests high atrial output, low impedances, and/or high ventricular sensitivity…crosstalk is the probable answer.

2. Atrial outputs in the absence of ventricular output coinciding with rates faster than programmed indicate Xtalk in non-rate responsive modes.

Correct solutions to the cross talk problem include the following…

1. Turn on the anti-crosstalk algorithm…

2. increase the ventricular blanking period (most probable answer from 2005)…

3. reduce the atrial output (unlikely)…

4. reduce the ventricular sensitivity (also unlikely).

Keep in mind that some devices with automatic capture capability may automatically reduce the AVD during the routine automatic capture update. Read the question carefully before answering.

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Volume 1: Number 28

I’ve compiled (from a couple of sources) a breakdown of question topics from the 2005 Pacing ExAM. Over the next couple of months, I will attempt to create a GtP for as many as possible. It’s not very likely that I will be able to cover all the topics, but at least you now have an idea of what to expect.

**Fundamentals of electrophysiology (5 questions)**

- Pre and Perioperative Practice (34 questions)
- Surveillance: security system interaction
- SVT
- Timing intervals
- Ventricular high rate
- T wave oversensing
- Ventricular auto capture
- Ventricular output
- Ventricular sensing
- Wedensky effect.
- Wedensky, ventricular refractory
- Clinical trials (3 questions)
- Clinical trials
- Outcomes
- Randomized trials

**Applied Science and Technology (48 questions)**

- Congenital Heart disease
- ICD Complications
- Indications
- Indications for pacing
- LV Lead
- Lyme disease
- RV Apex Lead Placement
- Sick sinus syndrome and syncopy
- Surgical technique
- Tamponade
- Upper rate limit.

**Pre and Perioperative Practice (34 questions)**

- Cardioversion
- Device EMI interaction
- EMI/Extracardiac signals
- ICD therapy
- Radiation safety

**Safety (8 questions)**

- Auto capture in a non-dependent patient
- Charge time
- ECG interpretation:
  - Sinus beat undersensing
  - Retrograde Conduction
- Electrocardiography of AAI mode
- Far field R wave sensing
- Histograms
- ICD sensitivity
- Inappropriate shock
- Lead noise
- Mode switching
- Pacemaker reprogramming
- Pacemaker syndrome
- Pacing lead complications
- Programming
- Sudden brady response

**Pharmacology (3 questions)**

- Atrial capture
- Atrial pacing preference
- Atrial R oversensing
- Atrial sensitivity programming
- Atrial tachycardia
- Charge time
-ECG interpretation:
- Sinus beat undersensing
- Retrograde Conduction

**Roentgenology (4 questions)**

- Coronary Sinus structures
- Pacing lead connection
- Persistent left SVC

**Cardiac Life support (4 questions)**

- Transcutaneous pacemaker

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Volume 1: Number 29
Transcutaneous External Pacing (TEP)

Most of the following information was lifted from this web site: http://www.emedicine.com/EMERG/topic699.htm
Author: Joseph Bocka, MD, Director, Shelby Hospital, Department of Emergency Medicine, Med Central Health System (Mansfield and Shelby, OH)

Inventor: Paul Zoll, MD (1911 – 1999)

In 1981, Zoll patented and introduced a transcutaneous external pacemaker with a longer pulse duration (40 msec) and a larger electrode surface area (80 cm2). This reduced the current required for capture and increased comfort for the patient. Additionally, this model could be applied much more rapidly than earlier TEPs, paving the way for renewed interest in TEPs. In 1982, the FDA approved use of the Zoll TEP for patients with heart rates less than 40 beats per minute and asystole. The current AHA ACLS guidelines included the use of TEPs for symptomatic bradycardias.

Application of Therapy: Electrodes/pads and monitor leads, if necessary, are placed on the patient. About 2-3 cm of space should be left if separate defibrillation pads are required, and the second pad should be placed posteriorly, just below the left scapula. The desired heart rate is chosen and the current is set to zero milliamperes (mA). The TEP is then turned on and the current is increased as tolerated until capture is achieved.

Pulse Duration: Current TEPs deliver 40 (Zoll) or 20 (all others) msec pulses. Zoll found that increasing the duration from 1 to 4 msec resulted in a 3-fold reduction in threshold (the current required for stimulation). Increasing the current from 4 to 40 msec further halves the threshold.

Current: Human studies have shown that the average current necessary for external pacing is about 65-100 mA in unstable bradycardias and about 50-70 mA in hemodynamically stable patients and volunteers. At this current, more than 90% of patients tolerated pacing for 15 or more minutes.

Using a longer pulse duration and larger electrodes permits patients to tolerate higher applied current. One hundred milliamperes of current applied over an average (50-ohm resistance) chest for 20 msec will deliver 0.1 Joules. This is well below the 1-2 Joules required to cause an uncomfortable tingling sensation in the skin. The force of skeletal muscle contraction, not the electric current, determines TEP discomfort. Current TEPs are capable of delivering up to 140-200 mA tolerably.

Electrodes: Most commercially available electrodes are 80-100 cm2. TEPs generally perform best with their own pads, but different combinations may be helpful. Pain is a function of the current delivered per unit of area. Pain sensation is minimized by electrodes with a surface area of at least 5 cm2. The amount of pain for a current of a given strength reaches a plateau once the electrode surface area exceeds 10 cm2.

Hemodynamics: Varghese reported that external pacing simultaneously stimulated all 4 heart chambers in dogs. Madsen, however, echocardiographically demonstrated in humans that atrial stimulation was retrograde without opening the mitral valve.

Studies have shown no difference in hemodynamics between transcutaneous and transvenous pacemakers, using comparable rates in complete heart block and cardiac arrest.

The atrial-pacing threshold in humans is generally much higher than that for the ventricles; thus, current needed to stimulate all 4 chambers is not tolerated, even by sedated patients. This results in loss of the "atrial kick" and a reduction in cardiac output. Talit studied healthy volunteers and found, via Doppler measurements, that both stroke volume and cardiac output were reduced even when pacing at a rate 15-30% higher than the sinus baseline. Thus, external pacing must be used cautiously in patients with sinus bradycardia to ensure blood pressure is preserved.

Minimizing discomfort: Placing electrodes over areas of least skeletal muscle can minimize discomfort.

CPR: CPR can be performed with the TEP pads in place. However, turning the unit off during CPR is advisable.

[On the 2005 ExAM for Pacing, there were 4 cardiac life support questions. At least one pertained to Transcutaneous External Pacing.]
Persistent Left Superior Vena Cava

* Incidence-uncommon
  o 0.3% of general population:
  o 4.3-11% of patients with CHD
* Two types
  o Persistent left SVC connecting to right atrium via coronary sinus is only common anomaly of SVC (90% of this anomaly)
  o In other 10%, persistent SVC connects to left atrium
    + Most with connection to left atrium have associated ASD or heterotaxy syndromes
    + This produces a right-to-left shunt of a rather small magnitude
* Etiology
  o Failure of regression of left anterior and common cardinal veins and left sinus horn
* Course of persistent left SVC
  o Draining into right atrium
    + Starts at junction of left subclavian vein and left internal jugular
    + Passes lateral to aortic arch
    + Receives left superior intercostal vein
    + Anterior to left hilum
    + Joined by hemiazygous system
    + Crosses posterior wall of left atrium
    + Receives great cardiac vein to become coronary sinus (common)
  o Draining into left atrium
    o Starts at junction of left subclavian vein and left internal jugular
    o Passes lateral to aortic arch
    o Receives left superior intercostal vein
    o Anterior to left hilum
    o Joined by hemiazygous system
    o Passes between the left atrial appendage (anteriorly) and the left superior pulmonary vein posteriorly
* Absent / small left brachiocephalic vein (65%)
* Really this abnormality produces bilateral SVCs
* In small percentage, right SVC is absent (10-18%)

Freedom, Culham and Moes, Angiography of Congenital Heart Disease, 1984

(A) Pulmonary artery chest X ray following implantation of the tripolar RV defibrillation lead through a persistent left SVC. showing the typical a shaped loop of the lead in the right atrium, which is caused by the close proximity of the orifice of the coronary sinus through which the leads enter the atrium and the tricuspid valve. The ICD generator in pectoral position serves as the defibrillation anode (active can principle). (B) Lateral chest X ray. The lead follows the course of the persistent left SVC along the posterior part of the left atrium, enters the heart through the coronary sinus at the bottom of the right atrium, and passes the tricuspid valve near the peak of the loop in a more anterior and superior position. From: ANDREAS MARKEWITZ, SOREN MATTKE (1996) Right Ventricular Implantable Cardioverter Defibrillator Lead Implantation Through a Persistent Left Superior Vena Cava Pacing and Clinical Electrophysiology 19 (9), 1395–1397.
Coronary sinus angiogram in a patient with ischemic cardiomyopathy (right anterior oblique 30° view). This angiogram highlights lateral segmental branch and the multiple second-order tributaries originating in the basal (B), mid (M), and apical (A) regions, coursing in the anterior (apical branch) and posterior direction (i.e., branches from the basal and mid-region). LV = lateral marginal vein; GV = great cardiac vein; AV = anterior interventricular vein.

I hope you find these radiographic images helpful in your quest to understand the coronary venous anatomy. A few details about site selection are located at the end of the review.

<table>
<thead>
<tr>
<th>Lead position</th>
<th>n</th>
<th>Pacing threshold</th>
</tr>
</thead>
<tbody>
<tr>
<td>1 Mid-cardiac vein</td>
<td>8</td>
<td>0.9 (0.2)</td>
</tr>
<tr>
<td>2 Posterior vein</td>
<td>7</td>
<td>0.9 (0.7)</td>
</tr>
<tr>
<td>3 Posterior lateral vein</td>
<td>22</td>
<td>1.0 (0.7)</td>
</tr>
<tr>
<td>4 Lateral vein</td>
<td>39</td>
<td>1.2 (0.9)</td>
</tr>
<tr>
<td>5 Great cardiac vein</td>
<td>16</td>
<td>1.6 (0.9)</td>
</tr>
<tr>
<td>6 Anterior lateral vein</td>
<td>10</td>
<td>0.8 (0.4)</td>
</tr>
</tbody>
</table>

Above: Left ventricular lead position and implant pacing thresholds. From: Six year experience of transvenous left ventricular pacing lead implantation for permanent biventricular in patients with advanced heart failure: technical aspects: C Alonso, C Leclercq, F Revault d'Allonnes, D Pavin, F Victor, P Mabo and J-C Daubert, in Heart
Rotational venous angiogram, with still frames in anteroposterior and left anterior oblique 30°. PV = posterior vein; MV = middle cardiac vein; GV = great cardiac vein; AV = anterior cardiac vein. Along side are three-dimensional reconstructed images of the coronary venous tree, facilitating segmental visualization of the coronary venous anatomy. The coronary venous ring is divisible into three segments: posterior (C), lateral (B), and anterior (A), in both views. (B) The mid-ventricular position of a lateral segmental branch (1), closely corresponding to the second-order lateral tributary from an anterior segmental branch, extending over the mid-ventricular region (2).

From: The Coronary Venous Anatomy: A Segmental Approach to Aid Cardiac Resynchronization Therapy by Jagmeet P. Singh, MD, PhD, Stuart Houser, MD, E. Kevin Heist, MD, PhD, Jeremy N. Ruskin, MD Boston, Mass. (Jam Coll Cardiol 2005; 46:68-74) © 2005 by the American College of Cardiology Foundation

To achieve the therapeutic goal of cardiac resynchronization, it is critical that the LV epicardial lead be positioned appropriately in the region with delayed electrical activation and mechanical dyssynchrony (1). So far, the approach for lead positioning has been rather simplistic and has been mostly directed at placement of the lead along the lateral wall of the LV (2). Recent data have suggested that mechanical asynchrony is variable and that a simplistic approach may not always provide the maximal hemodynamic benefit. The optimal site for LV lead implantation may vary depending on the region and/or extent of dyssynchrony (3,4).


“Lyme disease is the most common tick-borne disease in the United States and Europe. In the United States, Lyme disease is most often acquired from the bite of the Ixodes scapularis tick, with the spirochete Borrelia burgdorferi the sole cause. In Europe, Lyme disease is more commonly caused by B. afzelii than by B. burgdorferi.”

Presentation:
- Erythema migrans - an expanding red rash, usually round or oval in shape, but varying greatly in size and appearance.
  - develops 7 to 14 days (range, 3 to 30) after tick detachment
  - 75 to 80 percent of patients in the United States have only a single (primary) lesion
- Viral Type Symptoms
  - Arthralgias
  - Fatigue
  - Headache
  - neck pain
  - fever may or may not accompany these symptoms.

Without treatment, erythema migrans resolves spontaneously within a median of approximately four weeks. The more serious clinical sequelae of Lyme disease develop as a consequence of the hematogenous spread of the spirochete to extracutaneous sites. Spirochtemia can be found in about 45 percent of patients with erythema migrans at the time of presentation, irrespective of the size or duration of the skin lesion. Approximately 60 percent of patients with erythema migrans who are not treated will go on to have a monoarticular or oligoarticular arthritis, typically involving the knee; approximately 10 percent will have a neurologic manifestation, the most common of which is facial-nerve palsy; and approximately 5 percent will have a cardiac complication, usually varying degrees of atrioventricular block.

Treatment: 10 or 20 day course of doxycycline is usually effective for mild cases. Parenteral therapy (with ceftriaxone) is much more expensive and has greater potential for serious adverse effects. Therefore, such therapy is not recommended for patients with erythema migrans, except in unusual circumstances (i.e., in patients with advanced heart block from Lyme carditis or with neurologic manifestations of Lyme disease, aside from uncomplicated facial-nerve palsy).

From: Tick-Borne Diseases, Lyme: Jonathan A Edlow, MD, Associate Professor of Medicine, Department of Emergency Medicine, Harvard Medical School; Associate Chief, Department of Emergency Medicine, Beth Israel Deaconess Medical Center: http://www.emedicine.com/EMERG/topic588.htm

Cardiovascular involvement
- Cardiovascular involvement occurs in fewer than 10% of patients with untreated Lyme disease and is more common in male patients than in female patients.
- Palpitations, lightheadedness, and syncope may be a manifestation of varying degrees of heart block, including complete heart block, which occurs in 50% of patients with cardiac involvement. Lyme disease is an important reversible cause of heart block.
- Chest pain and dyspnea can occur in the setting of Lyme pericarditis, myocarditis, and myopericarditis. Tamponade has been reported.
- In patients with complete heart block, Canon A waves may be observed in the neck. A slow or irregular pulse may be palpated.
- A cardiac rub, S3 and/or S4, may be auscultated in patients with myocarditis or pericarditis. Signs of tamponade very rarely can occur. In patients with chronic cardiac involvement with congestive heart failure, typical signs of congestive heart failure may be present.
- Occasional patients with Lyme disease-related heart block will require temporary cardiac pacing. The indications for cardiac pacing are the same as for any other patient with varying degrees of heart block. Permanent wires are very rarely needed.
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**ATP: Antitachycardia Pacing** - Introduction of a sequence of pacing impulses, faster than the tachycardia rate in an attempt to terminate an arrhythmia. In theory, a single critically timed pulse will terminate reentry tachycardia. Placing the pacing lead close to reentry site may increase ATP effectiveness. There is some suggestion that ATP is most effective for VT rates <200 bpm. The results of ATP may be: 1) no effect; 2) acceleration of the tachycardia (including to ventricular flutter or ventricular fibrillation); or 3) termination of the arrhythmia.

The advantages of ATP include minimal symptoms for the patient (when effective, most patients remain unaware of the therapy) and immediate delivery of therapy.

The disadvantages are: 1) multiple bursts may be required (patient could become symptomatic); 2) the arrhythmia may be accelerated or changed to ventricular fibrillation; 3) the time to high voltage therapy can be prolonged. Note: all devices terminate ATP and commence High Voltage therapy when the rhythm is identified as accelerating into the “Fib” zone.

In past years, the ExAM has asked the participant to distinguish between “RAMP” and “BURST” pacing.

**Burst pacing** stimuli are delivered at a fixed cycle length. Care should be taken to keep the number of stimuli and the burst rate as low as possible to minimize the risk of VT acceleration.

**Ramp pacing** successively decreases the intervals between pulses within a burst. If Ramp pacing is Off, all of the stimuli in a burst are delivered at the same cycle length. If Ramp pacing is On, each interval after the first in a burst is decremented by the programmed Ramp Step. If the minimum burst cycle length is reached during Ramp pacing, the remaining stimuli are delivered at the minimum burst cycle length (BCL). **Ramp Step** is the amount by which each pacing interval within a burst is decremented during Ramp pacing. If the minimum BCL is reached during a burst, the pulse generator delivers the remaining stimuli at the minimum BCL.

**BCL** (burst cycle length) may be either:

- **Adaptive** - BCL is calculated as a percentage of the average tachycardia interval.
  
  For example, if the adaptive cycle length is 75% and the average tachycardia interval at the time of diagnosis is 400ms, the calculated BCL is 300ms (provided that the minimum BCL is less than or equal to 300ms). If more than one antitachycardia pacing burst is delivered in an episode, the BCL is not recalculated based on the tachycardia interval detected between bursts unless Readaptive is set to On.

- **Fixed** - the programmed BCL is used, regardless of the cycle length of the tachycardia.

The first stimulus is delivered synchronously with a sensed event. The remaining stimuli in the burst are delivered as VOO pacing at the programmed BCLs; they are not affected by the patient's rhythm.

All bursts use the same programmed number of stimuli unless added stimuli per burst is chosen.

Min BCL (minimum burst cycle length) is the shortest cycle length the pulse generator will deliver during an antitachycardia pacing burst. If Adaptive BCL, Readaptive scanning, or autodecremental pacing decrease the BCL such that the minimum BCL is reached, remaining pulses are delivered at the minimum BCL.

Additional stimuli per burst after the first burst are usually a programmable feature.

SCANNING controls the BCL between bursts.

If Scanning is On, the initial BCL of each burst decreases by the programmed Scan Step from one burst to the next. If Scanning is Off, the initial BCL of each burst is the same. When Ramp and Scanning are both On, the initial BCL of each burst decreases by the programmed Scan Step and each pacing interval in a burst (after the first interval) decrements by the Ramp Step. Scan Step is the amount the cycle length changes from one burst to the next during scanning.
Torsades de Pointes

This is from…ready for this…Wikipedia…”Torsades de pointes or torsades is a French term that literally means "twisting of the points". It refers to a specific variety of ventricular tachycardia and its name is derived from a maneuver in ballet. On the electrocardiogram, torsades is irregular, polymorphic, and often exhibits a "streamer" effect as the QRS complex transitions from positive to negative, and back again. Clinically speaking, the difference between polymorphic ventricular tachycardia and torsades de pointes is the presence of a **prolonged QT interval** in the underlying rhythm.

Common causes for torsades de pointes include hypomagnesemia (not enough magnesium) and hypokalemia (not enough potassium). It is commonly seen in malnourished individuals and chronic alcoholics. Diarrhea, dietary supplements, and various medications may also contribute.

<table>
<thead>
<tr>
<th>Familial long QT syndrome</th>
<th>Hypomagnesemia</th>
<th>Heart failure</th>
</tr>
</thead>
<tbody>
<tr>
<td>Family history of Sudden Death</td>
<td>Hypokalemia</td>
<td>Left ventricular hypertrophy</td>
</tr>
<tr>
<td>Congenital Deafness</td>
<td>Hypoxia</td>
<td>Slow heart rate</td>
</tr>
<tr>
<td>Class IA antiarrhythmics</td>
<td>Acidosis</td>
<td>Female gender</td>
</tr>
</tbody>
</table>

Factors that are associated with an increased tendency toward torsades de pointes

Torsades de pointes, complete heart block, on quinidine for a ventricular arrhythmia. The bottom strip shows rhythm shortly after use of quinidine.

Myerburg, R.J., M.D., Castellanos, A., M.D., Kessler, K.M., M.D., Recognition, Clinical Assessment and Management of Arrhythmias and Conduction Disturbances, Hurst's The Heart, 8th ed, p 745.

**Familial long QT syndrome**

Jervell and Lange-Nielson syndrome (genetic disease that causes profound hearing loss and arrhythmia, it is a type of long QT syndrome.

Romano Ward syndrome the most common form of inherited long QT syndrome inherited in an autosomal dominant pattern

Class IA antiarrhythmics: quinidine, procainamide, disopyramide

Class IC antiarrhythmics: encainide, flecainide

Class III antiarrhythmics: sotolol, amiodarone.

**Astemizole and terfenadine** (removed from U.S. market), in high dosages or when used in combination with the azole antifungal drugs or the macrolide antibiotics, have been reported to precipitate torsade and sudden death. **Grapefruit juice** has been shown to slow the hepatic metabolism of these antihistamines as well as other drugs and to prolong the QT interval in patients taking astemizole or terfenadine.

**Hypokalemia and hypomagnesemia**, cause a delay in phase III of the action potential and formss the substrate for emergence of the dysrhythmia. Patients with cirrhosis or hypothyroidism are particularly affected.
Okay, those of us involved in bradycardia pacing know the phenomenon seen at implant when stepping down voltage for capture thresholds, and there is a difference between the voltage at the point of loss of capture and the voltage when the capture reoccurs. This is known as the Wedensky Effect. For example, we lose capture at .5 volts, step up the voltage and regain capture at .8 volts. The actual capture threshold is then recorded as .8 volts. If only that were the end of it, I could stop this now and have history’s shortest GtP…

Here is the true definition of the Wedensky Effect. “a prolonged lowered threshold of excitability induced by strong stimulus (ed: that explains why you lose capture at a lower voltage than you recapture it); that under certain conditions one impulse could trigger a second impulse without invoking vulnerability or supernormality. And that such a phenomenon, and not a re-entry mechanism, could adequately explain the origin of extrasystoles occurring late in the cardiac cycle. Extrasystoles were due primarily to a disturbance of cardiac excitability, not of conductivity.” It shouldn’t surprise you to learn that this was first described in neuromuscular studies.

Here it is in action…

Exposure of the Wedensky effect in the human heart during intermittent paired electrical stimulation. Numbers indicate interspike (S↓-S↑) intervals in hundredths of a second. The first step in this process consists in identifying the supernormal phase. The driving rate was around 60 per minute. Numbers in the top strips indicate the distance between driving and testing stimulus artifacts. The latter did not stimulate the ventricles when occurring at intervals shorter than 0.425 sec or longer than 0.615 sec. Hence, testing stimuli produced a propagated response only during the supernormal phase, that is, towards the end of the T wave and slightly afterwards. In a second step, the intensity of the testing stimuli was lowered below the level required to detect super-normality. Note that when the intensity of the driving stimuli was increased from twice to 15 times above diastolic threshold, the second (previously subthreshold) impulse is now able to produce a propagated response. This is a classic Wedensky effect.


Still awake? There’s more… This mechanism was first documented on the nerve-muscle preparation and was defined as a “stronger” stimulus, where in the case of AV block, a ventricular premature or paced beat, is followed by transient antegrade conduction by decreasing the refractoriness of the AV conduction. Similarly to supernormal conduction, its existence in humans remains controversial.” Transient unexpected improvement of AV conduction: What is the mechanism? Meir Friedman, MD and Paul Schweitzer, MD; Beth Israel Medical Center, New York, NY; Indian Pacing Electrophysiol J. 2006 July–September; 6(3): 182–183.

A final note, for what it’s worth, one can eliminate the Wedensky Effect by beginning the threshold measurement at zero (0) volts and increasing pulse output until capture is attained. Since, traditionally, this is never done, the Wedensky Effect will be something observed at implant and ignored at follow-up until a new paradigm emerges.
Some of these sensors will be very familiar to you, unfortunately, the ones that show up on the ExAM won’t be those. However, fountain of knowledge that I am, I will attempt to touch on each sensor type that may appear.

First, some terms with which you will need to be familiar…

Open loop: this type sensor USUALLY does not respond to a specific physiologic function. Examples of open loop sensors are the piezoelectric crystal and accelerometer. Some sensors may be partially open loop. Examples of partially open loop sensors are the temperature sensor and respiration sensor.

Closed loop: this type sensor responds to physiological change, causes a rate increase or decrease according to bodily needs, measures the effect and modifies its output (rate modulation) as required. Examples of closed loop sensors are stimulus to T wave – aka: QT sensors - sensing devices (most likely to show up in a question), dP/dt of RV contraction sensors, pre-ejection interval (PEI) sensors and oxygen saturation sensors. The dP/dt, PEI and Oxygen sensors will not be discussed. There is no clear clinical advantage to using a closed loop sensor over an open loop sensor.

A question appeared a few years ago about device response to intellectual activity and/or emotion. Activity, temperature, and QT sensors DO NOT respond proportionately to either.

**Motion or Activity Sensors**

**Piezoelectric Crystal:** this sensor is bonded to the inside of pulse generator can and is highly sensitive to activity associated with walking – aka: heal strike rate – this in not a physiologic sensor. These sensors detect frequencies in the range of about 10 Hz, the so called resonant frequency of the human body. Watch carefully for the phrase “maximally sensitive to up and down motion” as this is how it is described in Furman’s “A Practice of Cardiac Pacing. 3rd Edition.” The first commercially available piezo device was the Medtronic Activitrax. It was actually developed by using their VDD pacemaker and modifying the atrial sensing input channel to detect output from the crystal.

**Accelerometer:** essentially, this is (but certainly not always) a piezoelectric crystal attached to the circuit board within the device can.

These devices are characterized by:

1. Rapid response to onset of exercise
2. Functionally simple
3. Compatible with any pacing lead

Negatives associated with motion sensors include:

1. No direct relationship between detected levels of activity and metabolic demand
2. Susceptible to environmental noise (highway vibrations) – *primarily piezoelectric sensor*
3. Susceptible to rate increases caused by pressure on the can – *primarily piezoelectric sensor*

**Respiratory Sensors**

**Minute Ventilation (MV):** the use of thoracic impedance to estimate the respiratory rate and tidal volume. The relationship between minute ventilation and cardiac rate during exercise is nearly linear. Rate modulation is closely correlated to VO₂.

MV is estimated using the transthoracic impedance between the pacing can and the pacing lead in a tripolar fashion.

1. Low frequency, 1 mA pulses with a 15 microsecond pulse duration are delivered from the anodal ring of the pacing electrode.
2. The voltage between the tip electrode and the pacing can is measured and resistance calculated (impedance pulses are delivered every 50 msec – *note this is a very important number to remember*).
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3. Transthoracic impedance
   a. Increases with inspiration
   b. Decreases with expiration

4. The transthoracic impedance is most closely related to the volume and resistivity of blood in the right heart chambers and systemic venous system.

Other Physiologic Sensors

The QT Interval (Vitatron): Although it is insinuated by the name that this sensor uses the QT Interval, it does not. It actually measures the time from the pacemaker stimulus until the peak of the T wave. This means that in order for the sensor to work, pacing must occur. The principle of QT Interval sensors is the natural physiologic shortening of the heart's qT interval with exercise or increased sympathetic tone. Periods of reduced activity concurrent with emotional stress, like, say taking the NASPExAM, can cause this device to increase the paced heart rate. This rate may not be proportional to the stress.

Problems associated with QT Interval devices include:
1. Marked changes in the QT interval due to medications.
2. Electrolyte concentrations may affect the QT Interval
3. Early devices suffered from...wait for it...T-wave undersensing
4. Additionally, degradation of the QRs may occur over time.
5. Low polarization are required for consistent accuracy.

It must be pointed out that 2nd and later generation devices were able to overcome problems 3 thru 5 by various methods. Improvements were made to the bandpass (T-wave) filter, and a fast recharge pulse was incorporated to neutralize the afterpotentials associated with high polarization leads.

These devices have a fixed absolute refractory period of 250 ms and programmable T-wave sensing windows from 250-450 msec.

Temperature (Cook)...no pun intended...: Uses changes in the central venous temperature to modulate rate. It does not use a temperature per se. For example, 98.6 F may be the steady state temp, but the device only knows it as baseline. If the temperature rises ½ a degree, the device does not know the CV Temp is 99.1, it only associates the rise as a fluctuation from baseline. This would begin a rate increase.

Important: When the body begins exercise from a relatively sedate state, such as sitting in a chair, or sleeping, the very first thing that occurs is a dip in the body temperature as venous blood pooled in the legs and arms surges into the thoracic cavity. When the pacemaker detects this dip, it uses an algorithm to begin pacing at an increased rate. This prevents a lag from occurring between the initiation of exercise and the central venous temperature increase. The device also contains algorithms that compensate for long periods of fever (again, a steady state will eventually occur), or forms of diurnal variation.

Blended Sensors

Boston Scientific (Guidant) sells a “blended sensor” device that incorporates an accelerometer for fast response to changes in motion, and a minute-ventilation sensor for response to changes in breathing due to exercise, stress, and emotion. The MOST study concluded: Quality of life analyses demonstrated that patients with blended sensors had significantly worse (P < 0.01) physical function than did patients with the other two sensor systems. Moreover, patients receiving blended sensors had the poorest absolute scores, without reaching statistical significance, on 9 of 13 quality of life measures after adjusting for differences in the groups.

Clinical and Quality of Life Comparison of Accelerometer, Piezoelectric Crystal, and Blended Sensors in DDDR-Paced Patients with Sinus Node Dysfunction in the Mode Selection Trial (MOST)

Authors: SHUKLA, HIMANSHU H.1; FLAKER, GREG C.1; HELLKAMP, ANNE S.2; JAMES, ERSKINE A.1; LEE, KERRY L.2; GOLDMAN, LEE3; ORAV, E. JOHN4; LAMAS, GERVASIO A.5

Source: Pacing and Clinical Electrophysiology, Volume 28, Number 8, August 2005, pp. 762-770(9)

Publisher: Blackwell Publishing
The upper limit of vulnerability (ULV) is the weakest shock strength at or above which VF is not induced when the shock is delivered at any time during the vulnerable period. There is a close correlation between the ULV and defibrillation threshold (DFT).

From Swerdlow, et al: The vulnerable zone displayed as a bounded, homogeneous region in a two-dimensional space defined by time (coupling interval) on the abscissa and shock strength on the ordinate. The ULV is the weakest shock strength at or above which VF is not induced when the shock is delivered at any time during the vulnerable period. The upper border (ULV) and the lower borders (VF threshold) are defined by shock strength. The inner (left) and outer (right) borders are defined by time (coupling interval).

A strict definition of the ULV would be the strongest shock that induces VF. Since the clinician is most interested in the weakest shock strength that results in reliable defibrillation, others and we have defined the ULV as the weakest shock that does not induce VF. Strictly, this might better be described as the “lower limit of invulnerability.” In general, investigators who define the ULV as the strongest shock that induces VF identify a lower value of ULV relative to DFT than those who use the alternate definition.

(1) The ULV provides a patient-specific measure of defibrillation efficacy with a single fibrillation–defibrillation episode. (2) The safety margin for implantable cardioverter defibrillator (ICD) shocks can be assessed without inducing VF.

All of the above information was directly quoted from: Using the Upper Limit of Vulnerability to Assess Defibrillation Efficacy at Implantation of ICDs in PACE 2007; 30:258–270.

A side note: most of us are familiar with the terms X Axis and Y Axis as related to graphs. This article uses the terms abscissa and ordinate to describe the Shock Strength and Time axes. The Exam may query you to identify each axis by its proper name.

The abscissa is commonly referred to as the “X Axis.” The horizontal coordinate of a point in a two dimensional coordinate system.

The ordinate is commonly referred to as the “Y Axis.” The vertical coordinate of a point in a two dimensional coordinate system.
If you can’t tell an IS-1 from an IS-4A, you may want to read on. Today’s menu items are leads and connectors.

Let’s work our way back to the connector pin from the tip of the electrode.

**Lead Tip Material**

**A tip:** The people who write the exam know you are too young to remember the “good ol’ days” so they sometimes ask questions about the “classics.” One such classic, other than moi, is a lead tip material that was developed by the Elgin Watch Company for their watch bands. This material is called Elgiloy. For the metallurgists among you, the material is an alloy of cobalt, iron, chromium, molybdenum, nickel, and manganese. A veritable soup d’jour of elements just to keep your wrist from turning green, or getting electricity through a wire into your heart.

Now on to the list…

1. Platinum/Iridium: platinum with 10% iridium
2. Elgiloy
3. Silver/Stainless Steel Combo (no substitutions, please)
4. Activated vitreous carbon
5. Platinum with Titanium Nitride (TiN) Coating

These days, nearly all leads contain a steroid eluting capsule containing dexamethasone, a potent synthetic member of the glucocorticoid class of steroid hormones.

Solid metal electrodes, such as uncoated platinum/iridium, tend to be smooth and, consequently, have smaller surface areas. Newer technology leads tend to be sprayed or “sputtered” with a fine coating of TiN, activated vitreous carbon, Elgiloy, silver, or other substance to provide…

1. Lower polarization
2. Fibrous ingrowth
3. A smaller fibrous capsule
4. Lower impedance which may improve sensing
5. Lower current and voltage to improve the stimulation threshold

**Lead Insulation**

By far the most common material for lead insulation is some form of silicone rubber. It is well tolerated by the body and not overly prone to wear or lead deterioration. In general, silicone lead bodies tend to be thicker than polyurethane leads.

In the past, polyurethane leads were available in two basic varieties, P80A (more commonly referred to as 80A) and P55D (a.k.a. 55D). 80A polyurethane is no longer used because it tends to biodegrade, resulting in cracks and insulation failure.

Obviously, newer formulations of lead insulation materials have been developed. It is not likely that questions pertaining to insulation material developed and put into use within the past year or two will find their way onto the exam.

The following table is adapted from “Cardiac Pacing” edited by Kenneth Ellenbogen

<table>
<thead>
<tr>
<th>Pacemaker Lead Insulation</th>
<th>Polyurethane (55D)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Pros</strong></td>
<td><strong>Pros</strong></td>
</tr>
<tr>
<td>Proven History</td>
<td>Proven History</td>
</tr>
<tr>
<td>Repairable</td>
<td>High tear strength</td>
</tr>
<tr>
<td>Low process sensitivity</td>
<td>Resistant to cutting</td>
</tr>
<tr>
<td>Easy to fabricate</td>
<td>Low friction</td>
</tr>
<tr>
<td>Very Flexible</td>
<td>Abrasion resistant</td>
</tr>
<tr>
<td></td>
<td>Thin</td>
</tr>
<tr>
<td></td>
<td>Relatively nonthrombogenic/fibrotic</td>
</tr>
</tbody>
</table>
ExAM Tidbits in easy to digest, bite sized morsels

<table>
<thead>
<tr>
<th>Cons</th>
<th>Cons</th>
</tr>
</thead>
<tbody>
<tr>
<td>Tears/Cuts easily</td>
<td>Not repairable</td>
</tr>
<tr>
<td>Low abrasion resistance</td>
<td>Stiff</td>
</tr>
<tr>
<td>High friction</td>
<td>Difficult to build</td>
</tr>
<tr>
<td>Thicker than polyurethane</td>
<td></td>
</tr>
<tr>
<td>More thrombogenic/and fibrotic</td>
<td></td>
</tr>
<tr>
<td>Subject to cold flow failure (micro holes in the insulation)</td>
<td></td>
</tr>
<tr>
<td>Calcifies due to absorption of lipids</td>
<td></td>
</tr>
</tbody>
</table>

### Lead Conductor Configurations

Become familiar with the term *filar*, it represents the number of filaments associated with the conductor design. Most modern leads that continue to use the wrapped filament design are either trifilar or quadrifilar.

The drawing to the left represents an example of a unifilar lead design. There is only a single wire filament used as the conductor. Should this wire fracture at any point, the electrical connection between the patient and the pacemaker would be broken.

The image at the right represents a lead conductor configuration using a trifilar (3 filaments) design. The use of multiple filaments assures that if one wire fractures, the electrical connection between the heart and the pacing device will remain intact.

In addition to the number of filaments associated with the pacing lead, internal designs also vary greatly. Below are some examples of internal lead construction.

Examples of four possible insulator/conductor configurations for pacing electrodes. The Coaxial configuration at the left consists of an inner coil (the cathode or negative pole), an insulator wrapped with a conductor (the anode or positive pole) and the outer insulation. Next to the coaxial lead is a lead consisting of two insulated, unifilar conductors surrounded by lead body insulation. The 7 French bipolar illustration is fairly typical of most bipolar lead configurations. This lead has a quadrifilar inner coil (cathode) insulated by a thin layer of silicone or polyurethane, next is the quadrifilar anodal coil that is insulated with the outer coating of the lead. The 5 fr. Unipolar lead is, also, typical of unipolar lead construction. In this case a quadrifilar coil (cathode) is surrounded by insulator. The inner electrode in both the unipolar and bipolar configuration, is hollow, which allows for insertion of a stylet to assist in lead placement.

### The Connector

For several years the standard connector pin designation for new implants of pacemakers and pace/sense leads in defibrillators has been the IS-1, or International Standard-1. As shown at the left, the IS-1 connector body incorporates a maximum diameter of 3.23 mm. The cathodal pin has a diameter of 1.59 mm, or .34 mm larger than the connector pin for the DF-1 Connector (shown at right). This prevents the low voltage lead from being accidentally connected to a high voltage port in a defibrillator.
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While there are some variations, one should remember these points…

**IS-1/VS-1** (VS-1 refers to “voluntary standard”)
- 3.2 mm diameter
- no sealing rings in header
- short receptacle for lead terminal

**VS-1A**
- 3.2 mm diameter
- no sealing rings in header
- long receptacle for lead terminal

**IS-1B/VS-1B**
- 3.2 mm diameter
- sealing rings in header
- long receptacle for lead terminal

It is important to note some variations. For example, some ICD headers do not have sealing rings and do not accept long pins, but still comply with the IS-1 requirements.

A the right are examples of 5 mm connectors. The top image (color enhanced for illustrative purposes only) is a bifurcated bipolar connector. The white rectangle represents (but is not indicative of actual markings) the distal electrode segment of the lead. In addition to the marker, which may be seen inside the insulation, rather than as illustrated, the distal pin port will be designed to receive a positioning stylet. The anodal port may, or may not be notched, and will not accept a stylet. The lower image represents a unipolar connector.

Finally, and not likely to show up on the exam, this year, is the new IS-4 configuration.

As you can see, this lead connector allows for 4 poles to be connected through a single port. This may be useful for future lead designs and device configurations for 4 chamber pacing, or multichamber leads.
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Volume 1: Number 39

I will try to hit the high points of a few congenital heart problems you may encounter on the exam. In no way should you consider this a complete review.

**Tetralogy of Fallot**

**What It Is**

Tetralogy of Fallot has four key features. A ventricular septal defect (a hole between the ventricles) and many levels of obstruction from the right ventricle to the lungs (pulmonary stenosis) are the most important. Also, the aorta (major artery from the heart to the body) lies directly over the ventricular septal defect, and the right ventricle develops thickened muscle.

Because the aorta overrides the ventricular defect and there's pulmonary stenosis, blood from both ventricles (oxygen-rich and oxygen-poor) is pumped into the body. Sometimes the pulmonary valve is completely obstructed (pulmonary atresia). Infants and young children with unrepaired tetralogy of Fallot are often blue (cyanotic). The reason is that some oxygen-poor blood is pumped to the body.

**Surgical Treatment**

*Temporary Operation*

In small and very blue infants, a shunt operation may be done first to provide adequate blood flow to the lungs. This lets the baby grow big enough to have a full repair. The shunt is built between the aorta and the pulmonary artery. The shunt is removed when a complete intracardiac repair is done later.

*Complete Repair*

To do a complete repair, the surgeon closes the ventricular septal defect with a patch and opens the right ventricular outflow tract by removing some thickened muscle below the pulmonary valve, repairing or removing the pulmonary valve and enlarging the peripheral pulmonary arteries that go to both lungs. Sometimes a tube is placed between the right ventricle and the pulmonary artery. This is sometimes called a Rastelli repair.

**Transposition of the Great Arteries**

**What It Is**

In transposition of the great arteries, the aorta and pulmonary artery are reversed. The aorta receives the oxygen-poor blood from the right ventricle, but it's carried back to the body without receiving more oxygen. Likewise, the pulmonary artery receives the oxygen-rich blood from the left ventricle but carries it back to the lungs.

**Surgical Treatment**

All patients with transposition of the great arteries require surgery early in life to survive. Many infants undergo a procedure in the catheterization laboratory to "buy time" and delay the surgery until they can handle it better. The procedure enlarges a naturally occurring connection between the right and left upper chambers (the atria). This lets the blood mix so some oxygen-rich and oxygen-poor blood can be pumped to the correct side.
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Two major types of surgery can correct the transposition. The first creates a tunnel (a baffle) between the atria. This redirects the oxygen-rich blood to the right ventricle and aorta and the oxygen-poor blood to the left ventricle and the pulmonary artery. This operation is called an atrial or venous switch. It’s also called the Mustard procedure or the Senning procedure.

The second type is called the arterial switch operation. The aorta and pulmonary artery are switched back to their normal positions. The aorta is connected to the left ventricle, and the pulmonary artery is connected to the right ventricle. The coronary arteries, which carry the oxygen-rich blood that nourishes the heart muscle, also need to be re-attached to the new aorta.

Truncus Arteriosus (http://www.americanheart.org/presenter.jhtml?identifier=11073#)

What It Is

Truncus arteriosus is characterized by a large ventricular septal defect over which a large, single great vessel (truncus) arises. This single great vessel carries blood both to the body and to the lungs.

Surgical Treatment

Surgery is required to close the ventricular septal defect and separate blood flow to the body from blood flow to the lungs. This is generally done early in infancy to prevent high blood pressure from damaging the lungs’ arteries. A patch is used to close the ventricular defect. The pulmonary arteries are then disconnected from the single great vessel and a tube (a conduit or tunnel) is placed from the right ventricle to these pulmonary arteries. This is sometimes called a Rastelli repair.

Other common congenital heart defects to review

Aortic Stenosis: the aortic valve is stiffened and has a narrowed opening. It does not open properly, which increases strain on the heart because the left ventricle has to pump harder to send blood out to the body. Sometimes the aortic valve also does not close properly, causing aortic regurgitation.

Atrial Septal Defect

Atrioventricular Canal Defect: also known as endocardial cushion defect or atrioventricular septal defect. Typically there is a large hole between the atria and, often, an additional hole between the ventricles. Instead of two separate valves allowing flow into the heart (tricuspid on the right and mitral valve on the left), there is one large common valve, which may be quite malformed. Atrioventricular canal defect is commonly seen in children with Down syndrome.

Coarctation of the Aorta: This is a narrowing of a portion of the aorta, and often seriously decreases the blood flow from the heart out to the lower portion of the body.
Hypoplastic Left Heart Syndrome: When the structures of the left side of the heart (the left ventricle, the mitral valve, and the aortic valve) are underdeveloped, they are unable to pump blood adequately to the entire body.

Patent Ductus Arteriosus (PDA): The ductus arteriosus (DA) is a normal blood vessel in the developing fetus that diverts circulation away from the lungs and sends it directly to the body. If the DA doesn't close, then a condition called patent ductus arteriosus (PDA) results, which can result in too much blood flow to a newborn's lungs. PDA is common in premature babies.

Pulmonary Atresia: This defect is one where the pulmonic valve does not open at all and may indeed be completely absent. The pulmonary artery also may be malformed and the right ventricle can be abnormally small.

Pulmonary Stenosis: The pulmonic valve is stiffened and has a narrowed opening.

Total Anomalous Pulmonary Venous Connection: The pulmonary veins don't join the left atrium during development. Instead they deliver blood to the heart by other pathways, which may be narrowed. Pressure builds up in this pathway and in the pulmonary veins, pushing fluid into the lungs, decreasing the amount of oxygenated blood that reaches the body. These infants often have difficulty breathing and appear bluish.

Tricuspid Atresia: The tricuspid valve is replaced by a plate or membrane that does not open. The right ventricle therefore does not receive blood normally and is often small. Often corrected using the Fontan Procedure. The Fontan Procedure is a type of surgery that is used for children with complex congenital heart disease. This procedure is only used for children who cannot have surgery that gives them two pumping chambers. The procedure is designed to direct the blood coming back from the body directly to the lungs, without being pumped to the lungs by the heart.

Ventricular Septal Defect (VSD): One of the most common congenital heart defects. Some of the smaller defects may gradually close on their own.
Unipolar coronary sinus leads can be programmed in a "common ring" configuration in which the anodal electrode of the right ventricular lead is used as anodal electrode for the coronary sinus lead. In this configuration, a left ventricular (LV) stimulus can give rise to anodal stimulation at the right ventricular (RV) lead, causing simultaneous biventricular activation. If the LV capture threshold is greater than the RV anodal capture threshold, one may observe either biventricular capture, or RV anodal only capture. This may interfere with programming of an interventricular (VV) pacing delay.

When right ventricular anodal stimulation occurs during biventricular (BiV) pacing, it has been hypothesized that three wave fronts of ventricular depolarization occur from LV tip and RV tip plus RV proximal electrode (the triple-site pacing phenomenon). Apparently, pacing from three different sites produces a hemodynamic benefit detected by echocardiography.

Anodal RV stimulation can render timing of a VV interval ineffective when the LV is stimulated before the RV, resulting in unaltered simultaneous biventricular stimulation over a wide range of VV intervals. At shorter V-V intervals a change in morphology is observed which is due to anodal as well as cathodal RV stimulation.

The 12 Lead ECG

In general, a non-paced 12 lead ECG obtained prior to implant (or post-implant with intrinsic only rhythm) is the best guide to assessing LV vs. RV pacing. This baseline ECG may be compared with RV only, LV only, and BiV pacing to confirm if capture is present. The typical RV only pacing pattern is a wide complex, left bundle branch block (LBBB) configuration, usually easily identified in leads V1 through V4 as a negative deflection of the QRS. Depending upon the position of the LV lead, the surface ECG may indicate a right bundle branch block (RBBB), LBBB, or variation of either. From “Advanced ICD Troubleshooting: Part II” by Charles Swerdlow and Paul Friedman in PACE Vol. 29, Jan 2006: “During determination of the LV pacing threshold, the QRS complex usually widens with loss of LV capture. It becomes less negative in lead I and more negative in lead III as ventricular excitation originates from the inferior RV as opposed to the lateral LV.”

When Biventricular pacing is present the ECG tends to narrow somewhat (but, it must be emphatically pointed out, NOT ALWAYS), and may appear as a variant of LBBB, RBBB or normal impulse.

The Device Based Intracardiac Electrogram (IEGM)

Using device based IEGMs may be of significant help in assessing LV only capture. Suggestions for IEGM programming include, RV Coil to Can (BiV capture verification utilizing RVcoil to Can evoked response in CRT-D patients; F. Philippon, et al; Europace, June 2005); SVC Coil to Can; observation of changes in impulse to IEGM timing, etc.

“At least seven possible states of ventricular pacing can occur in cardiac resynchronization systems that use an extended bipolar pacing configuration. These are no capture (intrinsic conduction) and capture from six specific pacing configurations:

RV cathodal + LV cathodal (the intended state);
LV (cathodal) only;
RV cathodal only;
RV anodal only;
RV anodal + LV cathodal; and
RV (cathodal + anodal) + LV (“triple site” pacing).

Most of these can be distinguished by analysis of intracardiac electrograms combined with a 12-lead ECG, which is invaluable for follow-up of resynchronization pacing systems.” (Swerdlow & Friedman)

The morphology change associated with LV versus RV capture is best examined in the ECG lead that is perpendicular to the axis shift. A change from BV to LV capture was best identified as increasing positivity of the QRS in lead III, while a change from BV to RV capture was best recognized as increasing positivity of the QRS in lead I. (A new and reliable method of individual ventricular capture identification during biventricular pacing threshold testing.: P. Yong and C. Doby; PACE, Nov. 2000.)
An interesting aspect of the multiple choice test is not having to know everything about every aspect of a particular topic. For the IBHRE Exam, it’s doubtful that every conclusion from every article published on heart failure will need to be committed to memory. Much more likely is having to know the basis or reason the research was performed, and the conclusion(s) reached. To get this information, one only needs to read the first and final paragraphs of the article. For example a quick search of the journal PACE for the term “Cardiac Resynchronization Therapy” returned several articles. Since it is not likely the exam will include information from 2007, because they just can’t put the thing together that quickly, and items older than a couple of years may be dated, it’s fairly simple to select one or two to review.

Examples (note: the highlights are mine):

“Cardiac Resynchronization Therapy (CRT) is currently an established therapy for patients with congestive systolic heart failure and intraventricular electrical or mechanical conduction delays. It is based on synchronized pacing of the atrium and the two ventricles. The resynchronization task demands exact timing of the cardiac chambers so that the overall stroke volume is maximized for any given heart rate (HR). Optimal timing of activation of the atrium and the right and left ventricles is one of the key factors in determination of the cardiac output. The timing parameters that are programmable in a CRT device that determines the pacing intervals are the atrioventricular (AV) delay and interventricular (VV) interval. Clearly, optimizing resynchronization is patient dependent as well as time and activity dependent. Intuitively, the best combination of pacing time intervals that restores optimal synchrony will change considerably during normal daily activities. CRT devices must be individually optimized, “fine-tuned” to provide optimal benefits. In addition to being time consuming and expensive, echo-guided AV and VV interval programming is limited to a static, sedentary activities. The impact of HR, body position, medications, and many other variables on these programmable variable is unknown. Determining these programmable variables should ideally be automatic and adaptive to the patient’s activities.

“Conclusion: The present simulation model suggests that an online adaptive CRT device based on a neural network—learning module has the potential to solve major weaknesses of the current CRT devices. The adaptive CRT device with feedback control from a hemodynamic sensor can provide a solution for the need for auto-programmability and auto-adjustment capabilities in a CRT device.

“In addition, an external adaptive CRT device is expected to significantly simplify the follow-up procedure, to optimize pacing intervals and to select a better lead position and hence to identify and improve response to CRT, and subsequently reduce costs. In vivo data are needed to prove the clinical benefits expected from the adaptive CRT device.” From: Adaptive Cardiac Resynchronization Therapy Device: A Simulation Report; Rami Rom; et al; Pace 2005; 28:1168-1173.

“Cardiac resynchronization therapy (CRT) has been shown to be an effective therapy in selected patients with advanced drug-refractory heart failure. Nevertheless, about 20–30% of patients in randomized trials do not respond clinically. This may be due to a variety of reasons, such as patient selection, inappropriate lead positioning, or suboptimal device programming. We recently reviewed the utility of echocardiography for patient selection. Follow-up of patients with CRT implies additional considerations as compared to conventional pacing. These aspects are reviewed in this article, which focuses on biventricular pacing (issues with ICD follow-up having been reviewed recently.

Conclusion Optimal follow-up of patients with CRT plays an important role in ensuring that patients will derive maximum benefit from this treatment. In addition to ensuring adequate therapy delivery by evaluating standard parameters such as pacing thresholds and percentage of ventricular stimulation, AV and VV delays may be optimized. Echocardiography has been the most frequently used technique for this application, but is relatively time consuming, especially as concerns VV optimization. Clinical benefit of AV and VV interval optimization still has to be demonstrated in prospective randomized studies. With the current growth in number of CRT implantations, many centers will not have the means to optimize devices in all their patients. Ideally, devices should be able to program optimal settings automatically, or suggest optimal settings to the physician via device-based algorithms. A pragmatic approach would be to verify adequate transmirtal inflow at baseline by echocardiography (with AV optimization only if required, e.g., if the A wave is truncated) and focus individual AV and VV optimization at follow-up on nonresponders or in patients who may have responded initially with secondary clinical deterioration.” From: Optimization of Device Programming for Cardiac Resynchronization Therapy; Haran Burri; et al; PACE 2006; 29:1416-1425.
“Cardiac resynchronization therapy (CRT) has proven beneficial in patients with heart failure and electrical conduction disturbances. Ventricular dissynchrony is corrected by pacing both ventricles through leads placed in the right (R) and left (L) ventricles (V). Optimization of CRT requires the adjustment of atrioventricular and interventricular (VV) pacing delays.

“In LV pseudobipolar (i.e., LV tip to RV proximal electrode) configuration, unexpected RV anodal capture (AC) has recently been reported during LV pacing. Furthermore, when RV AC occurs during biventricular (BiV) pacing, it has been hypothesized that three wave fronts of ventricular depolarization occur from LV tip and RV tip plus RV proximal electrode (the triple-site pacing phenomenon). Apparently, pacing from three different sites produces a hemodynamic benefit detected by echocardiography. The aim of this study was to establish the prevalence and characteristics of RV AC in CRT devices and to evaluate how this phenomenon may affect the device programming. Finally, we analyzed whether triplesite pacing mode produces QRS variations.

“Conclusions: RV AC through pseudobipolar LV pacing may be obtained in a significant proportion of patients. In around half of them, this pacing configuration obtains the narrowest QRS. When BiV pacing plus RV AC is used, ventricular depolarization occurs from the RV ring (as pseudobipolar LV pacing) or from the RV tip (as conventional BiV pacing) depending on the VV interval programmed.” From: Anodal Capture in Cardiac Resynchronization Therapy Implications for Device Programming: DAVID TAMBORERO; et al; PACE 2006; 29:940-945.

Please accept my apology for the “wordy” presentation, today. However, a huge amount of relevant and up-to-date information can be obtained by assigning oneself a fairly simple reading task. You may be surprised at how much you can learn with such a minimum of effort.
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From: Mode Switching of Dual Chamber Pacemakers from Activation of a Blanked Flutter Search Algorithm by a Single Atrial Event; S. Serge Barold, Carsten W. Israel, and Bengt Herweg; PACE 2005; 28:917–920.

“Failure of automatic mode switching (AMS) may occur in dual chamber pacemakers during atrial flutter when alternate flutter waves coincide with the post-ventricular atrial blanking period (lock-in phenomenon)... For this reason, some dual chamber pacemakers offer two different AMS algorithms, one for detection of atrial tachycardia, including atrial fibrillation and a supplemental algorithm for the detection of atrial flutter unrecognized by the primary algorithm. In the Medtronic Kappa 700 and 900 DDDR pacemakers, the second algorithm (Blanked Flutter Search) is automatically activated as soon as the pacemaker senses a high atrial rate suggestive of atrial flutter with a 2:1 lock-in response... We have observed activation of this special algorithm in circumstances unrelated to atrial flutter, atrial tachycardia, or sinus tachycardia. This report describes 5 such cases involving AMS initiated by a single atrial event.

“Activation of AMS (based on the Blanked Flutter Search algorithm) by far-field pre-ventricular atrial sensing (short AR-AS cycle). (A) The 5th sinus beat (arrow) falls in the extended PVARP and is registered as an AR event. It is immediately followed by a ventricular extrasystole (in the AV delay) that exhibits pre-ventricular far-field atrial sensing so that an AS event (beyond the PVARP) occurs before ventricular sensing (VS). Note the decrease of the pacing rate (AP-AP) at the onset of AMS a response of the device in the DDIR mode at the onset of AMS. (B) The 7th sinus beat (arrow) falls in the extended PVARP and is registered as an AR event. This sinus beat or AR event gives rise to a conducted QRS complex which then exhibits pre-ventricular far-field atrial sensing (beyond the PVARP) depicted as AS before VS. The device stored the sequence as AMS because it made the diagnosis of atrial flutter. However, the diagnosis of the DDIR mode cannot be made on the stored tracing because the pacemaker was inhibited by the spontaneous rhythm.

“On the basis of our observations, we conclude that the essential element of the Blanked Flutter Search algorithm is the detection of AR anywhere in the total atrial refractory period (excluding blanking times) of the cycle where the PVARP is periodically extended provided AR is either preceded or followed by an AS event.”

Here’s one more from Drs. Israel and Barold… Failure of Atrial Flutter Detection by a Pacemaker with a Dedicated Atrial Flutter Detection Algorithm; Carsten W. Israel and S. Serge Barold; PACE 2002; 25:1274–1277.

“Automatic mode switching (AMS) is used in patients with dual chamber pacemakers and paroxysmal atrial tachyarrhythmias. AMS changes the pacing mode from the atrial tracking (VDD, DDD, DDDR) to nontracking (VDI, DDI, DDIR) mode upon atrial tachyarrhythmia detection. AMS failure may occur during atrial flutter when alternate flutter waves coincide with the post-ventricular atrial blanking period (locked-in phenomenon). For this reason, some dual chamber pacemakers offer two different AMS algorithms, one for detection of atrial tachyarrhythmia including atrial fibrillation and a supplemental algorithm for the detection of atrial flutter unrecognized by the primary algorithm. In the Medtronic Kappa 701
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DDDR (Minneapolis, MN, USA) pacemaker, the second algorithm (Blanked Flutter Search) is automatically activated as soon as the pacemaker senses a high atrial rate suggestive of atrial flutter with a 2:1 locked-in response.

Schematic drawing of Blanked Flutter Search operation (simultaneous representation of lead II surface electrocardiogram [ECG], atrial electrogram [AEGM], and marker diagram [MD], 25 mm/s). In this example, every second atrial flutter wave is not sensed by the pacemaker due to coincidence with the postventricular atrial blanking period (PVAB) (black boxes in MD). However, if atrial cycles are sensed at an interval shorter than twice (atrioventricular [AV] delay 1 PVAB) the tachycardia detection interval for eight consecutive cycles (annotations À – Ç), the pacemaker suspects blanked atrial flutter. The Blanked Flutter Search algorithm now extends the postventricular atrial refractory period (PVARP), (white boxes in MD) to 400 ms for one cycle (arrow). By this, the formerly tracked atrial signal (atrial sensed [AS]) becomes refractory (AR) (atrial refractory sensed) and is thus not followed by a ventricular paced (VP) event. The formerly blanked flutter wave becomes detectable in the unblanked portion of the PVARP and reveals a short atrial cycle (*). On detection of this short atrial cycle, the pacemaker activates the AMS. The atrial sensed events are no longer tracked, and using a rate smoothing algorithm, the ventricular rate is driven towards the sensor rate (DDIR). If the Blanked Flutter Search does not disclose atrial flutter, it is automatically deactivated for 90 seconds.

Finally, this article is a MUST READ...


“The clinical behavior and programmability of the various types of AMS algorithms are reviewed in this part of the article. Current AMS algorithms can be classified according to the way atrial tachyarrrhythmias are detected: (1) “Rate cutoff” criterion: the sensed atrial (As) rate exceeds a programmable value; (2) “Running average rate” criterion: the atrial rate exceeds a mean atrial rate calculated by the pacemaker from the duration of the preceding atrial rate; (3) “Sensor-determined” physiological rate to distinguish sinus rhythm from atrial tachyarrhythmia; and (4) Complex algorithms that combine one or more of the above criteria, with or without additional methods such as examining the atrioventricular (AV) relationship.”
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Volume 1: Number 43

ICD Capacitors

The IBHRE exam will expect you to have some basic knowledge of electrical components, and how they work. Since nearly all basic pacing classes teach the “fluid” dynamics of basic electricity, I will endeavor not to be too redundant, here. Keep in mind, electrical engineering is a four year (or longer) curriculum at Georgia Tech, so one or two pages of GtP is not going to make you an expert. I do hope this information stimulates you to continue researching the principles on your own.

Capacitor: a device that stores an electrical charge on closely spaced conductors (plates). When voltage is applied to the capacitor, electric charges of equal magnitude, but opposite polarity, build up on each plate. A capacitor has been described as “A tank full of electrons.”

Capacitance is a measure of the amount of electric charge stored (or separated) for a given electric potential. The capacitor's capacitance (C) is a measure of the amount of charge (Q) stored on each plate for a given potential difference (the voltage present between two points) or voltage (V) which appears between the plates:

\[ C = \frac{Q}{V} \]

The farad is defined as the capacitance of a dielectric for which a potential difference of one volt results in a static charge of one coulomb. Note: ICD capacitors are generally rated at between about 90 and 150 microfarads.

A dielectric, or electrical insulator, is a substance that is highly resistant to electric current.


“Theoretical models predict that defibrillation thresholds (DFTs) may be lower for smaller output capacitors than the 120- to 150-µF capacitors used by current implantable cardioverter defibrillators (ICDs)… Capacitors in the range of 90 µF may provide a compromise between the potential benefits of smaller capacitors and the requirement for excessively high voltages. In the present study, we prospectively compared DFTs for 120 µF -65% tilt pulses, 90 µF -65% tilt pulses, and 90 µF -50% tilt pulses. The 90 µF -50% tilt pulse was selected because the pulse duration is half that of the 120 µF -65% tilt pulse. The time constant of pulses used by ICDs is the product of the resistance of the defibrillation pathway and the capacitance of the output capacitor.”

So…what does all this mean? Essentially, the smaller the capacitor, the greater the amount of voltage required to deliver an equal charge to the tissue. The greater the voltage, the faster the charged is delivered (remember, voltage is electrical pressure). A fast discharge is closer to the “membrane time constant” and hence more efficacious.

For example: a 155 µF capacitor would require 600 volts and deliver 29 joules and an 86 µF cap would require 830 V to deliver 30 J. Since the voltage on the 86 µF cap is greater than the voltage on the 155 µF cap, the charge will be delivered faster.

Whew! This is hard stuff!!
The following information is from an internal communication by Mark Kroll, PhD. It describes how a 21 joule output from an 830 V device delivers equivalent defibrillating efficacy to a 27 J output from a 600 volt unit.

“This is a comparison of an optimal waveform to the waveform primarily used in the MADIT 2 trial. The optimal has a peak voltage of 830 volts. The phase one duration is 4 ms and phase two is 2 ms. The MADIT 2 waveform has a low voltage of around 600 volts. Because of this low voltage it requires a great deal of time to deliver its energy. And, the durations are not programmable but are fixed with a 60% phase one tilt and a 50% phase two tilt. For a 50 ohm patient that gives durations of 7 and 6 ms respectively. These durations are about twice optimal. In fact, the first phase is longer than both phases of the optimized waveform.

“Based on the cellular, animal, and human research, we can confidently calculate the effectiveness of this waveform and state that it has the equivalent defibrillation capability of 21 J from a shock of optimal durations.”

As you can see, this can be complicated to comprehend. I will endeavor to send out a few clarifying documents in the weeks to come. In the meantime, I would highly recommend you spend some time becoming familiar with electronic principles as they apply to pacing and, especially, defibrillation.


Whew! This is hard stuff!!
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Volume 1: Number 44

The following information may be completely useless, most likely will not appear on the exam, but its kind of fun, if not important to know.

Pacemaker Batteries

These days, all pacemaker batteries use Lithium/iodine cells. There are two very good reasons for this.
1. The self discharge rate is very low, which gives the pacemaker a long shelf life.
2. The voltage characteristics remain stable throughout most of the useful life of the device allowing a clear delineation between working voltage, recommended replacement time, and end of service.

(note: End of Service is the recommend description for the predicted end of the useful service life of the device. The term End of Life should no longer be used.)

In the “old” days, other compounds were used, some with great success, some…ummm….not so much.

**Lithium Cupric Sulfate:** Excellent energy density. Used only in Cordis devices. Highly corrosive and, therefore difficult to contain in an enclosed environment. No longer used.

**Lithium Thional Chloride:** Used by ARCO Corporation (yes, the petroleum company made pacemakers, for a while). This compound failed due to very unstable end of service characteristics.

**Nickel-Cadmium:** believe it or not, this rechargeable type battery was used in the first ever permanent pacemaker implant (Sweden, October 1958). It was also used in a product made by Pacesetter Systems. NiCad batteries are very heavy and must be recharged at frequent intervals. Many of the PSI devices lasted well over 15 years, with patients donning a vest once a week to recharge the implanted device using inductance. Ultimately, the light weight and stability of lithium/iodine cells caused the NiCad to fade into history.

**Plutonium:** Yep, nuclear power. These devices required the implanting and following physician(s) obtain a license from the Atomic Energy Commission (AEC, now NRC – Nuclear Regulatory Commission). Additionally, the follow-up physician had to agree to follow-up the patient until the device was removed or the patient died, and the pacemaker had to be disposed of according to AEC regulations. Burying the device with the deceased patient was highly discouraged. A second form of nuclear fuel for devices was promethium. Believe it or not, promethium powered devices had an expected longevity of a middle of the road lithium-iodine device. Not many were ever implanted. Plutonium, on the other hand, had an expected longevity of 20 to 40 years (the cumulative survival rate was 82% after 16 years). I always wondered why anyone would agree to a 40 year pacemaker, when lead longevity was around 12 years.

**Mercury-Zinc:** These cells were used in pacemakers until the lithium compounds began to appear. Mercury-Zinc cells were wired in series to provide about 4 to 8 volts. Unfortunately, they were wired in series. This meant that when one cell failed, the others tended to dump their charges into the failed cell, causing early failures of many of these devices. Additionally, mercury-zinc cells produced gas that had to be vented. Some devices were implanted without venting, and like a can of spoiled meat, the gas build-up caused the cans to become inflated and eventually “explode” – more of a poof, really – under the skin. Not a good thing. A typical mercury-zinc cell could be expected to last about 2 or 3 years (but many only last about 2 or 3 weeks). Ah! Those were the days.
Battery Capacity

Battery capacity is measured in Ampere Hours (Ah). A typical pacemaker battery can be expected to have about 1 to 2 Ah capacity. Here’s where reading between the lines becomes important… The Ah can be described as either TOTAL or USEABLE. These are not the same thing. The Total battery capacity includes the unusable capacity and overestimates the usefulness of the cell by about 10 percent. The Useable capacity takes into account that lithium cells are only useable for about 85 to 90% of their total capacity. Watch out for this on the exam. You must read carefully and understand the question fully if battery capacity is part of the answer.
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Volume 1: Number 45

The following are some clinical studies that may appear, in various types of questions on the exam. Unfortunately, there is no way to determine which of these, if any, will be considered worthy of inclusion in the exam. Keep in mind, this list is in no way complete and other clinical studies may show up on the exam.

**Companion:** *N Engl J Med* 2004;335:2140-50
- Results: **risk reduction** for the endpoint by CRT/CRT-ICD Rx
  - Primary endpoint: Total mortality or total hospitalization: - 20% CRT/CRT-ICD (p=0.01)
  - Secondary endpoint: Total mortality: -24% CRT (p=0.059) / -36% CRT-ICD (p=0.003)
- **36% Reduction in mortality in the CHF-Defibrillator group.**

- Heart failure class II or III CAD or DCM + LVEF <35%
- Endpoint: all-cause mortality: Placebo vs Amiodarone vs ICD
- Intention to treat: 2521 pts. 148 centers, FU 45.5 months
- Mortality: Placebo 29%, Amiodarone 28%, ICD 22%
- Amiodarone has no effect on mortality
- **23% decreased risk of death for ICD therapy**

**MADIT I:**

MADIT I randomized (1:1 ratio) 196 patients to conventional antiarrhythmic drugs (N=101) or an ICD (N=95). Amiodarone was the predominant treatment in the drug limb of this study. The 2-year mortality was 32% in the antiarrhythmic drug limb and 10% in patients who received an ICD. The hazard ratio comparing the risk of death per unit of time in the ICD group with that in the conventional therapy group was 0.46, which indicates a relative reduction of 54% in the risk of death. The difference in survival between the two treatment groups was significant (P=0.009).

**MADIT II:** *Circulation. 2003;108:1779*

MADIT II was designed to evaluate the survival benefit of a prophylactic ICD in patients with prior myocardial infarction and left ventricular ejection fraction = 30%. The study enrolled 1,232 patients who were randomly assigned in a 3:2 ratio to receive an ICD (N=742) or conventional medical therapy (N=490). Invasive electrophysiology studies were not required for risk stratification. During an average follow-up of 20 months the mortality rates were 19.8% in the conventional therapy group and 14.2% in the ICD group. Thus, the absolute reduction in mortality was 5.6%. The hazard ratio for the risk of death from any cause in the ICD group as compared with the conventional therapy group was 0.69 (p=0.016), which indicates a relative reduction of 31% in the risk of death at any interval among patients in the ICD group. Kaplan-Meier estimates of survival for the groups are shown in Figure 3. The study was stopped early because of the benefit in survival for patients receiving the ICD.
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AVID: Circulation. 1999;99:1692-1699

The Antiarrhythmics vs Implantable Defibrillator (AVID) study is the largest of these trials (N=1,016), but the mean follow-up was shortest (1.5 yrs). Although a small number of patients in the drug therapy limb were treated with sotalol (3%), most received amiodarone (96%). The study was stopped early because of significant reductions in total mortality and arrhythmic death. The total mortality death rates (deaths per year) were 16.5% in the amiodarone group and 10% in the ICD group. The corresponding arrhythmic death rates were 7.4% and 3.0%. A retrospective analysis of the AVID data concluded that in patients with left ventricular ejection fractions \( \geq 35\% \) survival was comparable in the antiarrhythmic drug therapy and ICD groups, but for those with left ventricular ejection fractions \( <35\% \) survival from all cause and arrhythmic death was superior in patients who received ICD therapy.


MUSTT randomized 704 patients with inducible ventricular arrhythmias to no antiarrhythmic therapy (N=353) or electrophysiologically guided therapy (N=351). In patients who were assigned to no antiarrhythmic treatment, the 5-year mortality was 48%. In the group randomized to electrophysiologically guided therapy, the 5-year mortality was 24% among patients who received an ICD and 55% for those who did not (P<0.001). The lower mortality rate in patients with ICDs was largely attributable to a reduction in the rate of arrhythmic deaths. In the electrophysiologically treated group, the 5-year rate of cardiac arrest or arrhythmic death was 9% for those who received an ICD and 37% for those who did not. Figures 1 and 2 compare the total mortality and deaths from arrhythmias in patients who did not receive antiarrhythmic therapy, patients who received electrophysiologically guided therapy without an ICD, and those with electrophysiologically guided therapy who received ICD therapy. An important conclusion from MUSTT was that ICDs reduced the risk of sudden death, but antiarrhythmic drug therapy was not effective.


The Cardiac Arrest Study Hamburg randomized 288 patients to ICDs (N=99), amiodarone (N=92), and metoprolol (N=97). Although CASH was a smaller study, the mean follow-up was 4.5 years. Compared with AVID, the higher mean left ventricular ejection fraction (45%) in CASH probably reflected a large number of patients without organic heart disease. Total mortality rates were 9.4% for those treated with amiodarone and 7.7% in the ICD group (not significant). There was a significant reduction in arrhythmic deaths in patients who received an ICD (1.5% vs those treated with amiodarone 5.1%). Mortality in the metoprolol group was similar to amiodarone. The percent reduction in all cause mortality with ICD therapy was 39% at 2 years and 28% at 3 years, but the survival curves converged at 6-7 years of follow-up because of nonarrhythmic deaths. The authors concluded that the benefit of ICD therapy is more evident during the first five years after the index event.
The Canadian Implantable Defibrillator Study (CIDS) randomized 659 patients with VF or VT to treatment with an ICD (N=328) or amiodarone (N=331). The mean duration of follow-up was three years. Death rates for total mortality were 10.2% in the amiodarone limb and 8.3% in the ICD limb. The corresponding arrhythmic death rates were 4.5% for amiodarone and 3.0% for ICD therapy. At 2 years the relative risk reduction with ICD therapy was 29.7% for total mortality and 31.4% for arrhythmic deaths. A recent analysis of CIDS was presented at the 2002 AHA Scientific Sessions. The analysis determined that in a subset of CIDS, the benefit of ICD over amiodarone increased over time. Most of the amiodarone die, have recurrences, or developed side effects by 11 years follow-up.
Interestingly, most of the literature surrounding pacemaker infections occurred in the early to mid-1980s. In those reports *Staphylococcus aureus* and *Staphylococcus epidermidis* were the two most common causative agents.

The most recent article I could easily find is from October 1994 (Br Heart J. 1994 Oct; 72(4):339-3; Antibiotic prophylaxis in permanent pacemaker implantation: a prospective randomised trial; Mounsey, et. al.). This study found:

1. Infections were significantly more common when the operator was inexperienced (< or = 100 previous patients),
2. the operation was prolonged, or
3. after a repeat operation for non-infective complications (principally lead displacement).
4. Infection was not significantly more common in patients identified preoperatively as being at high risk (for example patients with diabetes mellitus, patients receiving long term steroid treatment), although there was a trend in this direction.
5. Antibiotic prophylaxis significantly reduced the incidence of infective complications requiring a repeat operation after permanent pacemaker implantation.
6. It is suggested that antibiotics should be used routinely.

In “A Practice of Cardiac Pacing, 3rd Edition,” David Holmes states in his chapter “The Implantable Cardioverter Defibrillator” that

1. Infections occur in about 5% to 6% of ICD implantations
2. There is usually a long delay (weeks or months) before symptoms appear
3. Symptoms include local erythema (an abnormal redness of the skin caused by capillary congestion), tenderness, or a draining sinus
4. Fever and leukocytosis (an elevated number of white cells in the blood) are relatively common
5. Sepsis is rare

Mark Midei, MD and Jeffrey Brinker, MD, in their chapter “Pacemaker Implantation” (Cardiac Pacing, Kenneth A. Ellenbogen, MD Editor) name *Staphylococcus aureus* as the most common agent for acute infections. They give the honor of most common for infections appearing months or years after implantation to *Staphylococcus epidermidis*.

They go on to say that antibiotic therapy, alone will not be sufficient, in most cases, to cure the infection. Complete removal of all the implanted hardware (device and leads) is usually necessary.
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Volume 1: Number 47

Lead Extraction

Much of the following information was extracted from this site: http://cogprints.org/4258/01/bracke.htm as was found in the Indian Pacing and Electrophysiology Journal 2003;3(3):101. “The Lead Extractor's Toolbox: A Review Of Current Endovascular Pacemaker And ICD Lead Extraction Techniques” by Bracke FA, MD.

Extraction Methods

1. Laser Assisted
   a. 308 nm XeCL excimer laser
   b. Pulsed light at a max fluence of 60 milliJoules/mm²
   c. 40-80 Hz repetition rate
   d. Used photochemical destruction of cellular structures
      with explosive photothermal vaporization of cellular water.
      i. Transient micro-bubbles mechanically disrupt the tissue
      ii. Ablation depth is approximately 2 to 15 microns per pulse
   e. the blunt tip of the laser sheath is not suited for direct mechanical disruption of the fibrous scar
   f. applying more force than necessary to assure good contact with the tissue does not improve efficacy but increases the risk of complications

2. Conventional Intravascular counter traction
   a. As the leads may be too fragile to withstand traction it is necessary to use a locking stylet
   b. Counter traction is applied to the distal electrode
   c. The force is thus concentrated at a small area of the scar tissue without gross displacement of the myocardium
   d. counter traction prevents invagination of the myocardium
   e. perforation of the myocardium is still possible
      i. the lead tip may have been incorporated into the myocardium
      ii. the possibility of increasing the force using counter-traction can lacerate the myocardium especially in the thin-walled atrium

3. Locking stylets
   a. Used to avoid disintegration of the lead during traction
   b. stylet is introduced into the central lumen of the lead
   c. a straight non-expandable wire is locked into the coil close to the tip of the lead
   d. The force exerted via a locking stylet is almost directly applied at the tip
   e. Some stylets can be unlocked and repositioned if necessary
   f. Limitations
      i. If the conductor is broken or distorted it is not possible to introduce the stylet
      ii. Excessive force can dislocate the stylet
      iii. the distal conductor coil can unwind or disconnect from the electrode
      iv. risk of invagination of the myocardium
      v. may not be able to remove the lead tip through the fibrous sheaths along the lead body
4. Traction
   a. Direct traction without additional tools is the most basic technique
   b. 5-10 times rotation of the lead with simultaneous gentle traction may be helpful
   c. Prolonged graded traction using increasing weights that are connected to the proximal end of the lead and guided over a pulley mounted on the bed of the patient
   d. To keep the patient ambulatory, prolonged traction fixed under tension to the skin using rubber bands and adhesive tape.

The Transfemoral Retriever (Snare)
A retriever is inserted through the sheath to grab and secure the lead as close to the tip as possible. The lead (with the connector cut off) is pulled inferiorly by the retriever whilst the outer sheath is advanced over the doubled up lead. The proximal part of the lead is pulled down through the fibrous envelope.
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Volume 1: Number 48
Pacemaker Syndrome

If someone asked me to guess the question I’m asked most by nurses and technicians in the pacemaker field, I would have to answer…“Just what exactly is pacemaker syndrome?” Questions about pacemaker syndrome were on each NASPExAM I took – AP Exam in 1990 or 91 (who remembers?) and the EP Exam in 1999 or 2000 (ditto).

Here is a simplified answer, taken from Cardiac Pacing, edited by Kenneth Ellenbogen. Section 3 “Hemodynamics of Cardiac Pacing” by Dwight Reynolds, MD

1. Symptoms
   a. Related to blood pressure and cardiac output
      i. Syncope
      ii. Malaise
      iii. Easy Fatigability
      iv. A sense of weakness
      v. Light-headedness
      vi. Dizziness (related to blood pressure and cardiac output)
   b. Related to higher atrial and venous pressures
      i. Dyspnea – frequently at rest
      ii. Orthopnea
      iii. Paroxysmal nocturnal dyspnea
      iv. Fullness and/or pulsations in the neck and chest
      v. Palpitations
      vi. Forceful heart beats

2. Associated with Loss of AV Synchrony
   a. VVI Pacing Mode
   b. Loss of atrial capture or sensing in DDD mode
   c. Retrograde conduction and entrainment of the P wave

3. Careful questioning may be necessary – pt. may deny symptoms

4. A careful physical exam may reveal...
   a. Neck vein distension w/prominent –Cannon- “A” waves
   b. Pulmonary rales
   c. Peripheral edema (rare)
   d. Hypotension (relative or absolute/continuous or fluctuating)

Definition: “Any combination of the variety of symptoms and signs occurring with ventricular pacing that are relieved by restoration of AV Synchrony” – Direct Quote from Cardiac Pacing.

Note on the “DAVID Study”: this study pursued the hypothesis that dual-chamber ICDs provide improved patient prognosis and reduced health care costs as opposed to single-chamber ICDs. 506 people with indications for ICD therapy participated in this multi-center, randomized clinical study. All participants had an ICD with dual-chamber, rate-responsive pacing capability implanted, which was randomly programmed to VVI or DDDR. For patients with standard indications for ICD therapy, no indication for cardiac pacing, and LVEF of = 40%, dual-chamber pacing offered no clinical advantage over ventricular backup pacing and may be detrimental by increasing the combined endpoint of death or hospitalization for heart failure.

I felt the DAVID information was important to include in the Pacemaker Syndrome section to illustrate the paradox between DDD pacing (Good) and the DDD ICD in pts. w/NO indication for pacing (BAD). Three are advantages to dual chamber ICDs, so don’t be mislead by these results.

Remember to use all resources available to prepare for the exam. There is no substitute for experience and practical knowledge.
Okay, let me be the first to admit it...I’m getting a little thin on ideas for the GtP Newsletters. Please recognize this one for what it is, a hunch that there may be one or two questions regarding the microvolt T-wave alternans test. When I prepare the Newsletters for next year’s “EP” exam, I plan to expand on this topic.

Alternating amplitude and/or morphology from beat to beat on the ECG, referred to as electrical alternans, has been recognized for almost a century, only in the last 25 years has this phenomenon been linked to SCD. Numerous reports propose that electrical alternans can precede the development of ventricular arrhythmias. T-wave alternans (TWA) is linked quantitatively to electrical instability in the heart. Fluctuations in the morphology of the T-wave during alternating beats, termed TWA or repolarization alternans, has been associated with the development of ventricular arrhythmias in animal models. These subtle alterations in T-wave morphology usually are not apparent on the surface ECG but are reflective of physiologically important abnormalities in repolarization that may be present on a microscopic, or microvolt, level. That is, the variation in T-wave morphology is only a few microvolts in amplitude and TWA cannot be detected by visual inspection of the surface ECG. Sophisticated signal processing techniques have been developed to measure TWA at the microvolt level (i.e., microvolt TWA [MTWA]). The original studies required the use of atrial pacing to unmask the alternans signal, but subsequent studies substituted atrial pacing with exercise to accelerate the heart rate to the optimal level, making the assessment of MTWA completely noninvasive. Typically, the onset heart rate for MTWA occurrence is lower in patients with structural heart disease and a history of sustained ventricular arrhythmia, than in patients with no structural abnormalities or history of arrhythmia (Rosenbaum, et al., 1994; Cambridge Heart, Inc., 2005; Barron, 2000; Costantini, et al., 2000; Cohen, 2001; Armoundas, et al., 2002; Gold, Spencer, 2003; Walker, Rosenbaum, 2003; Sarzi, et al., 2004).

The amplitude, or magnitude, of TWA is measured in microvolts (µV), or MTWA.

1. A value of $= 1.9 \mu V$ is considered a positive result.
2. The threshold onset heart rate must be < 110 beats per minute (bpm), and
   a. it must be sustained above that heart rate for the test to be considered positive
   b. this provides a means of establishing the statistical confidence of the alternans measurement.

The alternans ratio: divide the noise-corrected alternans voltage by the standard deviation of the noise.

1. This value indicates the number of standard deviations by which the alternans magnitude exceeds the noise level, and
2. is positive if it is $= 3$ (Costantini, et al., 2000; Armoundas, et al., 2002; Gold, Spencer, 2003).

Test Limitations

1. Cannot be administered if the pt. is in atrial fibrillation
2. Frequent atrial or ventricular ectopy
3. Paced ventricular rhythm
4. Inability to achieve a heart rate >105 bpm
5. Allergy to skin electrodes

Factors that may result in False Negatives or False Positives

1. High Signal to noise ratio that cannot be reduced appropriately
2. Frequent PVC’s

These factors cause about 15% to 40% of MTWA test results to be indeterminate. (Costantini, et al., 2000; Armoundas, et al., 2002; Gold, Spencer, 2003).

The latest information can be found at:
http://phx.corporate-ir.net/phoenix.zhtml?c=106685&p=irol-newsArticle&ID=977902&highlight=
Volume 1: Number 50

Signal Averaged ECG (SAECG) has mostly appeared on the EP exam. I’m not sure if any question regarding this diagnostic tool has ever appeared on the pacing exam. As with MTWA (GtP #49), this is only basic information, and the topic likely will not appear on the AP exam.

Signal averaged ECG (SAECG) is a technique involving computerized analysis of small segments of a standard ECG in order to detect abnormalities, termed ventricular late potentials (VLP), that would be otherwise obscured by "background" skeletal muscle activity. VLPs reflect aberrant, asynchronous electrical impulses arising from viable isolated cardiac muscle bordering an infarcted area and are thought to be responsible for ventricular tachyarrhythmias. Late potentials indicate slow conduction within the ventricular myocardium.

In 1996, the American College of Cardiology published an expert consensus document which concluded that SAECG had an established or valuable role in clinical care in the following situations:

1. Stratification of risk of developing sustained ventricular arrhythmias in patients recovering from MI who are in sinus rhythm without electrocardiographic evidence of bundle branch block or intraventricular conduction delay.
2. Identification of patients with ischemic heart disease and unexplained syncope who are likely to have inducible sustained ventricular tachycardia.
4. Assessment of success of operation for sustained ventricular tachycardia.

### Suggested Normal SAECG Criteria

<table>
<thead>
<tr>
<th>Equipment</th>
<th>Gender</th>
<th>fQRS ms</th>
<th>LAS ms</th>
<th>RMS µV</th>
</tr>
</thead>
<tbody>
<tr>
<td>Marquette (general electric)</td>
<td>M</td>
<td>=124</td>
<td>=42</td>
<td>=16</td>
</tr>
<tr>
<td>ART (Corozonix)</td>
<td>F</td>
<td>=116</td>
<td>=42</td>
<td>=15</td>
</tr>
<tr>
<td></td>
<td>M</td>
<td>=115</td>
<td>=47</td>
<td>=11</td>
</tr>
<tr>
<td></td>
<td>F</td>
<td>=107</td>
<td>=43</td>
<td>=13</td>
</tr>
</tbody>
</table>

Weighted means

On the basis of published data, our suggested normal values for SAECG parameters (40 Hz high-pass filtering) according to recording equipment and gender shown above. Normal values are estimated as the weighted average of the gender- and equipment-specific 90th percentile reported in Tables 1a and b. Males have longer fQRS duration than females, whereas limits for LAS and RMS do not differ significantly by gender, but remain equipment-specific. 


Criteria for normal values for a SAECG are those published in 1991 and 1996 in the Journal of the American College of Cardiology for risk stratification in postinfarction patients.1,2 Usually, 300–400 beats are averaged to meet the criteria of a noise level <0.3 µV. In these publications, it was suggested that for 40 Hz high-pass filtering, the normal value for filtered QRS (fQRS) duration is <114 msec, the normal value of low-amplitude signal duration below 40 µV (LAS 40) is <38 msec, and the root-mean square voltage in the last 40 msec of the QRS (RMS 40) should be >20 µV.

It has become common practice to state that the SAECG is positive if two of the three parameters are abnormal, although this categorization is not based on evidence. Marcus, Frank; et al. J Cardiovasc Electrophysiol, Vol. 18, pp. 231-233, February 2007

\[ f_{QRS} = \text{Filtered QRS} \]
The Endotak SQ Model 0048 is an electrode system consisting of three conductive elements (a so-called "Lead Array"), that can be subcutaneously inserted. The array was initially developed to replace the SQ patch in most configurations, acting as an anode or as a part of the anode. The SQ Array depolarizes a significantly greater cardiac surface area than the original subcutaneous patch (28 cm$^2$ for the patch vs a minimum of 51 cm$^2$ with the SQ Array). The conductive elements consist of an electrically common multifilar coil. The three elements (each measuring 25 cm in length and 6 French in diameter) are joined in a silicone yoke that connects the system to a terminal pin, which can be connected to the defibrillator. The three legs are separately introduced with a lead tunneler and peel-away sheaths.

Implantation Technique

An incision of about 3 cm is made to position the yoke and to introduce the lead elements with three separate sheaths after tunneling with a dilator to the left lateral part of the chest. The three elements are individually sutured after removal of the disposable sheaths.

Fluoroscopy during implantation: (A) initial lead position, with failing shocks; (B) after dispersion of the three branches of the array the patient could be defibrillated with biphasic bi-directional shocks of 10 J.
These chest radiographs (from 2 patients) illustrate a typical location for Endotak and SQ Array leads. The posteroanterior radiograph (left panel) shows the distal array leads extending posteriorly in the subcutaneous tissue of the chest wall. The leads are inserted through an incision at approximately the mid-clavicular line anteriorly. The lateral view (right panel) shows the three SQ Array lead elements somewhat splayed but paralleling the ribs.

“The results with the SQ Array were superior to the lead-alone, however, reduction in the DFT was not seen in all patients. Twenty percent of our patients had an identical DFT with the lead-alone and the lead-SQ Array. We also observed one patient (5%) in whom the DFT was lower with lead-alone intraoperatively, but postoperatively, the lead-alone was incapable of successful defibrillation.” The Subcutaneous Array: A New Lead Adjunct for the Transvenous ICD to Lower Defibrillation Thresholds; Higgins, S.L.; et al; PACE Vol 18; 1540:48; August 1995.

“In 2001, there was a report of subcutaneous arrays positioned in the right and left chest walls having good defibrillation thresholds in a 2-year-old child with ventricular tachycardia and repaired congenital heart disease. There are other reports of ICD implantation in a child using a single subcutaneous array lead and an abdominal active can and implantation from an ileofemoral approach. The major concern about these approaches at our institution was the potential for lead trauma and damage. These alternative techniques expose the fragile ICD coils to external trauma because they are not protected underneath the rib cage. In a study of subcutaneous ICD leads by Kettering, 6% of patients with a subcutaneous array had a major complication with the most common major complication being a lead fracture, which occurred in approximately 4% of patients. In the same study, an additional 6% of patients had minor defects of the lead insulation that were insignificant at the time of investigation but “might affect the future performance of these leads and result in serious complications in the future.” In children who are more prone to falls and sustaining blunt trauma to the stomach or chest, the potential for damaging a superficially placed lead is high. Innovative Techniques for Placement of Implantable Cardioverter-Defibrillator Leads in Patients with Limited Venous Access to the Heart; Cannon, B.C.; et al; PACE Vol 29; 181:187; Feb 2006.
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Volume 1: Number 52

Remember this number: 330 801…it is the model number of the Telectronics Accufix pacing lead that was the subject of a recall in 1994. Yep, 1994 and thirteen years later I have to bring it to your attention in my little newsletter.

“The Accufix series of atrial active-fixation permanent pacemaker was formally recalled on November 10, 1994, at a time when there were 4 recognized injuries, 2 fatal and 2 nonfatal, as a result of fracture and protrusion of a J-wire embedded in the distal end of the lead that was intended to help retain the J-shape of that portion of the lead…The recommendations for patient management left the decision to extract or not extract a particular lead to the physician with the aid of guidelines based on risk factors for J-wire fracture, injury from the J-wire, and injury from lead extraction.

Discussion
1. The number of deaths and major complications associated with lead extraction procedures exceeds the number of deaths and injuries attributed to J-wire fracture.
   a. There were 15 deaths and 113 major complications for lead extractions,
   b. while for J-wire related injuries the current totals are 6 deaths and 39 injuries.
   c. The number of J-wire related injuries which would have occurred over this period, had all of the extracted leads been left in situ, is unknown.

2. Some of the extracted leads may be assumed to have been at high risk of fracture, such as those with an L or straightened configuration.

3. Several of the deaths associated with lead extraction occurred in patients with no evidence of J-wire fracture and no other clinical Indication for extraction than the need to undergo elective pulse generator replacement.”


Examples of Accufix failures from:
Left: Lead Explantation Late After Atrial Perforation; Trigano, A and Caus, T. PACE 1996; 19:1268

Right: Interelectrode (Accufix) Lead Fracture; Trigano, A; et al; PACE 1999; 22:1705-1706

This is not highly likely to appear on the exam, but it has appeared there in the past.