Pre Test Questions

1. How long does a patient with a newly implanted BMS (Bare Metal Stent) need plavix?
   - A. 1 Week
   - B. 1 month
   - C. 6 Months
   - D. 12 Months

Pre Test Questions

2. How long does a patient with a newly implanted DES (Drug Eluting Stent) need plavix?
   - A. 1 Week
   - B. 1 Month
   - C. 6 Months
   - D. 12 Months
Pre Test Questions
3. In a patient with Atrial fibrillation, what is an indication for permanent pacing?
• A. Slow ventricular rates
• B. Rapid ventricular rates
• C. Paroxysmal Afib
• D. Symptomatic patients

Pre Test Questions
4. Which test has the highest yield in the work-up of unexplained syncope?
A. EKG
• B. Carotid ultrasound
• C. Holter Monitor
• D. Implantable Loop Recorder

Where have we been, where are we going?
• BMS, DES, POBA, Pacemakers, ICD's
• Early invasive strategy, D2B
• Lytic therapy
• CABG
• AVR/Stent Valves
• Goals for medical therapy
• Lipid goals
• Pay for performance
Drug Eluting Stents
Evolution and Application

CAD Treatment Evolution
Overview

POBA (Plain Old Balloon Angioplasty)
- Introduction 1977
- Post-POBA Restenosis ~ 40-60%

Stenting
- Coronary Stenting Keeping the Artery Open
  - Introduced 1987

Drug-Eluting Stents
- DE Stents
  - Decrease Restenosis
  - FDA Approved 2003

Restenosis Drivers
- Elastic Recoil
- Neointimal Hyperplasia

Physical Mechanism of Action
- Vessel Expansion
- Plaque Compression
**CAD Treatment Evolution**

**Mechanism of Action**
- Vessel Expansion
- Plaque Compression
- Compression Resistance

**In-Stent Restenosis Drivers**
- Minimal Elastic Recoil
- Neointimal Hyperplasia

- Disease State
- Post Stent
- Minimal Elastic Recoil
- Neointimal Formation

Stenting ~ 20-40% Binary Restenosis

**Drug Eluting Stents and Bare Metal Stents**

**Balancing the tradeoffs**
Benefit / Risk Profile of DES vs BMS

- Risk:
  - Stent thrombosis
  - MI, Death, TLR
  - Reduced restenosis

- Benefit:

How do these 2 risks (thrombosis vs. restenosis) balance out?

TAXUS® Express® Stent (SR)
Positive Benefit / Risk profile

TAXUS I, II, IV, V (N=2,797) – 5 year Meta-Analysis

- Risk:
  - p=0.46
  - p=0.14
  - p=0.92
  - p=0.76
  - p=0.46
  - p=0.91

- Benefit:
  - p=0.001
  - 21.0
  - 7.0

- 4K Stent/Plain Balloon
- SYNTAX
- ACUITY
- D-SHIELD
- E-SHIELD
- Death/MI
- Wave MI
- Q

DES Compared To BMS
Favorable Benefit / Risk balance

- Risk:
  - HSK reduced by >50%
  - Probable reduction in TLR-related MI/death*
  - Overall MI and death rates comparable or lower

- Benefit:

*BMS data show 9.5% of restenosis patients presented with acute myocardial infarction, and 26.4% presented with unstable angina.

The safety and effectiveness of the TAXUS ExpressStent have not been established in patients with lesions located at a bifurcation, in patients with a recent acute myocardial infarction where there is evidence of thrombus or poor flow, in patients with a recent acute myocardial infarction, in patients with diabetes, or in patients with renal failure.

Program Overview

• Three primary design elements impact drug-eluting stent (DES) performance:
  – The physical stent architecture or design
  – The polymer carrier
  – The anti-restenotic agent or drug

• These elements, while important on their own, work together to form a system; it is this combination or system, that should be carefully evaluated.
Stents

- DES (plavix 1 year)
  - Taxus
  - Cypher*
  - Promus
  - Xience
  - Endeavor

- BMS (plavix 1 month)
  - Driver
  - Liberte
  - Vision

Elements of DES Design

Stent Architecture Overview

- Three primary performance factors drive physical stent design:
  - Radial strength to minimize vessel recoil
  - Flexibility to improve deliverability
  - Conformability to maximize vessel wall contact or apposition
Why is Radial Strength Important?

- Stents provide scaffolding to resist compressive forces within the vessel.
- A number of factors contribute to radial strength:
  - Metal composition (or alloy)
  - Strut thickness
  - Stent geometry or pattern

Metal Composition

- Currently available stents use a variety of metals:
  - ASTM F38-03 (316L stainless steel)
  - ASTM F90-91 (L605 cobalt chromium)
  - ASTM F562-02 (IMP35N cobalt chromium)

Elements of DES Design

Polymer Carrier – The Drug/Stent Connection
Polymer Carrier Overview

- Polymers are critical elements in drug-eluting stent performance. More specifically, polymers:
  - Protect the drug during delivery to the target lesion
  - Control the dose of drug released into the artery
  - Control the timing of the drug released into the artery

Important Polymer Characteristics

- Drug delivery characteristics or “kinetics” can vary depending on which polymer design is used:
  - Low vs. high drug load
  - Controlled vs. fast drug release
- It is crucial to match the stent’s release kinetics to the drug – too little drug, too much drug or too fast or slow a release may impact DES performance

Polymer Carrier Summary

- Polymers play an integral role in the design of drug-eluting stents
- The type of polymer, the drug load combined with the polymer, and the single or multi-layer polymer configuration all have significant impact on drug-eluting stent performance
- No matter which polymer configuration is used the goal of the polymer carrier is to tailor the device’s release kinetics to the properties of the drug – too much drug, too little drug or too fast or slow release of the drug can all impair the performance of the drug-eluting stent.
Anti-Restenotic Agents

Definition

• Anti-restenotic agents or drugs used in conjunction with drug-eluting stents help to reduce restenosis by inhibiting the complex restenotic cascade.

• While the goal of all of these anti-restenotic drugs are the same, there are important and fundamental differences in the way these agents are believed to inhibit the restenotic response.

What is Paclitaxel?

• Paclitaxel was originally isolated from the Pacific Yew Tree, Taxus Brevifolia, in 1971.

• Paclitaxel, a naturally occurring agent, was found and developed by the National Cancer Institute.

• Paclitaxel was approved by the FDA in 1992 to treat cancer in the form of TAXOL®.
Paclitaxel Multifunctional Effects

- Paclitaxel is a multi-functional drug that:
  - Inhibits proliferation
  - Inhibits migration
  - Inhibits inflammation
  - Inhibits ECM (extracellular matrix) secretion
- Paclitaxel enables healing by selectively impacting the cells that cause restenosis while allowing healing of endothelial cells
  - TAXUS® Stent shows similar healing between control bare metal stent and paclitaxel.

What is Sirolimus?

- A naturally occurring immunosuppressive agent first found in the soil on Easter Island.
- Approved by the FDA in 1999 for the prevention of renal transplant rejection.
- Sirolimus is a cell cycle inhibitor.
- Sirolimus is lipophilic.

Sirolimus Mechanism of Action

- Sirolimus binds with mTOR
- mTOR is a critical signaling pathway that may be involved in restenosis

Sources referenced.
Everolimus and Zotarolimus

Mechanism of Action

• Everolimus and Zotarolimus also block mTOR signaling

What about PPI’s and Plavix

• clopidogrel and the PPI omeprazole and other drugs that inhibit the CYP2C19 enzyme
• Is it all just noncompliance?

Mixed data out
Switch patients or not?
We haven’t heard the final answer?

The Future of Stents
CAD Treatment Evolution
Stent Development

• FUTURE Stent Technology may include
• Different drugs on stents to combat restenosis
• Different drugs on stents to increase endothelial healing
• Combination of drugs on stents
• Bioabsorbable stents
• Stents with progenitor cells (stem cells)
• Gene therapy stents

Device Therapy

• Implantable Loop Recorders
• Pacemakers
• ICD’s

Differential Diagnosis

Cardiac
  - Arrhythmias: bradyarrhythmias, supraventricular or ventricular tachycardia, AV node dysfunction
  - Obstruction to Outflow: aortic stenosis, HCM, myxoma, dissection, pulmonary embolus, pericardial tamponade

Non-Cardiac
  - Neurologic: syncope, vertebrobasilar insufficiency, migraines
  - Metabolic: hypotension, hypoglycemia
  - Psychogenic: hyperventilation, panic disorder
Diagnostic Tests and Diagnostic Yields

<table>
<thead>
<tr>
<th>Test/Procedure</th>
<th>Yield</th>
</tr>
</thead>
<tbody>
<tr>
<td>ECG</td>
<td>2 - 11%</td>
</tr>
<tr>
<td>Holter Monitoring</td>
<td>2%</td>
</tr>
<tr>
<td>External Loop Recorder</td>
<td>20%</td>
</tr>
<tr>
<td>Tilt Table Testing</td>
<td>11 - 87%</td>
</tr>
<tr>
<td>EP Study without structural heart disease</td>
<td>11%</td>
</tr>
<tr>
<td>EP Study with structural heart disease</td>
<td>49%</td>
</tr>
<tr>
<td>Neurological (head CT scan, carotid doppler)</td>
<td>0 - 4%</td>
</tr>
</tbody>
</table>

Early Use of the Implantable Cardiac Monitor

<table>
<thead>
<tr>
<th>60 patients</th>
<th>Unexplained Syncope</th>
</tr>
</thead>
<tbody>
<tr>
<td>Conventional Testing (n=30)</td>
<td>Prolonged Monitoring (n=30)</td>
</tr>
<tr>
<td>Event Monitor/TTT/EPS</td>
<td>ILR</td>
</tr>
<tr>
<td>If (-), EPS/TTT</td>
<td>If (-), ILR</td>
</tr>
</tbody>
</table>

Diagnosis (n=7) | Diagnosis (n=22)

- CHB (event monitor)
- bradycardia (n=14)
- RVCA (n=3)
- NMS (n=3)
- SVT (n=2)
- Seizure (n=2)


The Sinoatrial (SA) Node

- The "natural pacemaker"
- High right atrium
- Unique ability to generate an electrical impulse
- In the healthy heart, the SA node fires (60-100 bpm) and the impulse
  - Causes atrial depolarization (p-wave)
  - Travels through the AV node
  - Conducts down to the ventricles and causes ventricular depolarization
The Atrioventricular (AV) Node

- Located in lower right atrium between the coronary sinus and the tricuspid valve’s septal cusp
- Electrical impulses from the SA node travel through the AV node, where they are briefly delayed (this gives the atria time to contract completely and extends passive filling time for the ventricles)
- The electrical impulse travels from the AV node down to the ventricles

Junctional Rhythm

- A rhythm disorder where the electrical impulse originates from the area around the AV junction
- Typically has a rate of around 40 to 60 bpm
- ECG may show an inverted P-wave, no P-wave, or P-wave obscured by a normal-looking QRS complex

Conduction Defects

- Defects in the conduction system affect one or more of the five areas of the conduction system
- Causes
  - Coronary artery disease (CAD)
  - Idiopathic degeneration
  - Calcification
  - Endocarditis
  - Heart surgery
  - Radiofrequency (RF) ablation
Indications for Pacing

- Pacing indications usually require both symptoms and documented evidence of arrhythmia
- Symptoms include: irritability, fatigue, forgetfulness, palpitations, chest pain, dyspnea, weakness, dizziness, presyncope, and syncope
- Documentation methods include:
  - Patient history
  - 12-lead ECG
  - Ambulatory Holter monitoring
  - Electrophysiologic (EP) testing in cardiac cath lab
  - Temporary pacing
  - Stress test

Sick Sinus Syndrome (SSS)

- Sick sinus syndrome (SSS) produces sinus bradycardia
  - Intrinsic rate is less than 60 bpm
  - ECG otherwise looks normal
  - Patients may or may not have symptoms
  - Not all patients with sinus bradycardia require a pacemaker! Symptoms are required.

Sinus Arrest/SA Node Exit Block

- Sinus arrest occurs when the SA node fails to produce an electrical impulse in a timely way
- SA node exit block occurs when the SA node produces the electrical impulse, but it fails to conduct normally
- Both produce pauses on the ECG
- Clinicians should notice the length and frequency of the pauses and the symptoms these pauses produce
Atrial fibrillation (AF) is characterized by very rapid and seemingly chaotic atrial activity. AF appears on the ECG as lots of atrial events, often in low-amplitude waves. The ventricular response to AF may be to try to keep up or, in this case, to respond with slower-than-normal activity. Digitalis toxicity can produce AF with slow ventricular response.
Tachycardia-Bradycardia Syndrome

- This rhythm disorder switches between very fast and very slow rates in an irregular way
- Appears as tachycardia and bradycardia episodes on an ECG
- Often treated with
  - Drug therapy to suppress the ventricular tachycardia
  - Pacing therapy to treat the bradycardia

AV Block

- AV block (a.k.a. heart block) is a rhythm disorder in which the impulses from the atria do not conduct properly down to the ventricles
  - The conduction may be delayed
  - The conduction may be partially blocked (only some impulses get through)
  - The conduction may be totally blocked (atria and ventricles are dissociated)
- AV block has three degrees
  - 1º AV block involves delayed impulses (not a pacing indication)
  - 2º AV block can be an indication for pacing
  - 3º AV block is most severe and is an indication even in the absence of symptoms!
Two Types of Second Degree AV Block

- Mobitz I or Wenckebach
  - PR interval lengthens progressively until a P-wave appears without a corresponding ventricular event
  - May or may not produce symptoms
- Mobitz II
  - PR interval remains stable
  - A QRS complex is “dropped” in a regular pattern
  - 2:1 AV block means there are two P-waves for every QRS complex
  - May or may not produce symptoms

Third-Degree AV Block

- Impulses are completely blocked in the AV node so that the atria have no connection to the ventricles (AV dissociation)
- Sometimes called “complete heart block”
- This means the ventricles have to beat on their own to pump any blood
- Most severe form of heart block
- This is the only rhythm disorder that is a pacing indication even if the patient has no symptoms

Mobitz I or Wenckebach ECG
SCD-Definition

"Death due to cardiovascular causes in patients with or without known preexisting heart disease in whom the mode and time of death are unexpected."*

The accepted temporal definition of such an event is a period of up to one hour between the onset of an abrupt change in clinical status and the loss of consciousness.

Etiology of SCA: Underlying Rhythm of SCA

- VT (62%)
- Torsades de Pointes (13%)
- Bradycardia (12%)
- Primary VF (8%)
Identifying the High-Risk Substrate

- Majority of SCA victims have coronary artery disease (CAD).¹
- Greater than 80% have no precipitating acute coronary occlusion²

Leading Causes of Death in the US

SCA is a leading cause of death in the U.S., second to all cancers combined.

ICD Clinical Trials Overview
### Evolution of ICD Clinical Trials & Indications

<table>
<thead>
<tr>
<th>Study</th>
<th>Incremental Indications</th>
<th>Prevention Strategy</th>
<th>Year of CMS Coverage</th>
</tr>
</thead>
<tbody>
<tr>
<td>SCD Survivors</td>
<td>All survivors of sudden cardiac death</td>
<td>Secondary</td>
<td>1991</td>
</tr>
<tr>
<td>AVI D</td>
<td>Sustained VT/EF ≤ 40%</td>
<td>Secondary</td>
<td>1999</td>
</tr>
<tr>
<td>MADIT I</td>
<td>Prior MI, EF &lt; 35-40%, non-sustained VT, inducible VT</td>
<td>Primary</td>
<td>1999</td>
</tr>
<tr>
<td>MADIT II</td>
<td>Prior MI, EF ≤ 30%, niv EP study required</td>
<td>Primary</td>
<td>2003</td>
</tr>
<tr>
<td>SCD-HeFT</td>
<td>No prior MI, EF ≤ 30%, Close RBB heart failure</td>
<td>Primary</td>
<td>2005</td>
</tr>
<tr>
<td>COMPENDIA</td>
<td>Class IV heart failure</td>
<td>Primary</td>
<td>2008</td>
</tr>
</tbody>
</table>

**Clinical Support**

#### Secondary Prevention ICD Clinical Trials

<table>
<thead>
<tr>
<th>Study</th>
<th>Trial Hypothesis</th>
<th>Conclusion</th>
</tr>
</thead>
<tbody>
<tr>
<td>AVI D</td>
<td>Initial ICD arrhythmic therapy prolongs patient life better than antiarrhythmic drug therapy</td>
<td>ICD group experienced 39% reduction in deaths in first year.</td>
</tr>
<tr>
<td>CIDS</td>
<td>Initial ICD therapy reduces risk of arrhythmic death compared to amiodarone in patients at high risk for arrhythmic death due to VT/VF</td>
<td>ICD group experienced a 20% absolute mortality reduction in 3 years.</td>
</tr>
<tr>
<td>CASH</td>
<td>ICDs reduce incidence of recurrence of CA, SCA, cardiac mortality and total mortality versus antiarrhythmic drugs</td>
<td>Mortality was reduced by 24% in ICD group.</td>
</tr>
</tbody>
</table>

#### Primary Prevention ICD Clinical Trials

<table>
<thead>
<tr>
<th>Study</th>
<th>Trial Hypothesis</th>
<th>Conclusion</th>
</tr>
</thead>
<tbody>
<tr>
<td>CABG PATCH</td>
<td>Prior MI, EF &lt; 35%, abnormal NSVT, abnormal SAECG, scheduled for CABG</td>
<td>Prophylactic ICD could reduce mortality compared to patient’s just undergoing bypass surgery, no significant survival benefit.</td>
</tr>
<tr>
<td>MADIT I</td>
<td>Prior MI, EF &lt; 35%, NSVT, EP positive</td>
<td>Prophylactic ICD improves survival compared with AA therapy. 54% reduction in overall mortality; 75% reduction in arrhythmic mortality over medication only group.</td>
</tr>
<tr>
<td>MADIT II</td>
<td>Prior MI, EF &lt; 30%</td>
<td>ICD reduces overall mortality in patients with previous MI and LV dysfunction vs. conventional pharmacological therapy. ICD with optimal drug therapy reduced overall mortality by 31%.</td>
</tr>
</tbody>
</table>
Clinical Support
Primary Prevention ICD Clinical Trials (continued)

<table>
<thead>
<tr>
<th>Trial</th>
<th>Population</th>
<th>Hypothesis</th>
<th>Conclusion</th>
</tr>
</thead>
<tbody>
<tr>
<td>DEFINITE</td>
<td>NICM, EF&lt;35% or PVCs or NSVT</td>
<td>ICD prevents fatal cardiac arrhythmias</td>
<td>ICD did not differ significantly in the ICD plus medication group vs. the medication only group. ICD did reduce arrhythmia-related deaths.</td>
</tr>
<tr>
<td>DINAMIT</td>
<td>Recent MI, EF&lt;35%</td>
<td>ICD prolongs life in recent MI patients compared to pharmacologic therapy alone</td>
<td>Overall mortality did not decrease. Arrhythmic mortality reduced by 45% with ICD.</td>
</tr>
<tr>
<td>MADIT-HoF</td>
<td>Prior or Recent MI, LV dysfunction</td>
<td>ICD improved survival in HF patients with reduced LV function but no history of near-fatal arrhythmia.</td>
<td>ICDs decreased mortality in HF dysfuction patients by 25% compared with patients on amiodarone alone.</td>
</tr>
<tr>
<td>SCD-HeFT</td>
<td>Prior MI or NICM, EF&lt;35%, LV dysfunction</td>
<td>ICD improves survival in HF patients with reduced LV function but no history of near-fatal arrhythmia.</td>
<td>ICDs decreased mortality in HF dysfuction patients by 25% compared with patients on amiodarone alone.</td>
</tr>
</tbody>
</table>

Trial Summary: Reduction in All-Cause Mortality with ICDs

![Graph showing reduction in all-cause mortality with ICDs]

Number Needed to Treat To Save A Life

<table>
<thead>
<tr>
<th>Trial</th>
<th>Number Needed to Treat</th>
<th>Lifesaving Benefits</th>
</tr>
</thead>
<tbody>
<tr>
<td>MADIT</td>
<td>23%</td>
<td>100 / (100 - 77)</td>
</tr>
<tr>
<td>MUSTT</td>
<td>23%</td>
<td>100 / (100 - 77)</td>
</tr>
<tr>
<td>MADIT-II</td>
<td>23%</td>
<td>100 / (100 - 77)</td>
</tr>
<tr>
<td>COMPANION</td>
<td>23%</td>
<td>100 / (100 - 77)</td>
</tr>
<tr>
<td>SCD-HeFT</td>
<td>23%</td>
<td>100 / (100 - 77)</td>
</tr>
</tbody>
</table>

ICD Therapy vs. Drug Therapy:

<table>
<thead>
<tr>
<th>Trial</th>
<th>5 Year</th>
<th>10 Year</th>
<th>Meta-analysis</th>
</tr>
</thead>
<tbody>
<tr>
<td>MADIT</td>
<td>4</td>
<td>8</td>
<td></td>
</tr>
<tr>
<td>MUSTT</td>
<td>6</td>
<td>9</td>
<td></td>
</tr>
<tr>
<td>MADIT-II</td>
<td>9</td>
<td>6</td>
<td></td>
</tr>
<tr>
<td>AKID</td>
<td>20</td>
<td>26</td>
<td></td>
</tr>
<tr>
<td>Marb-4</td>
<td>28</td>
<td>37</td>
<td></td>
</tr>
<tr>
<td>Merit-HF</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>4S</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Amiodarone Meta-analysis</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
General Cardiologist Refers ~35% While PCPs Refer Nearly ~75%
## Device Guidelines: ICDs & CRT

### Classification of Recommendation & Level of Evidence

**Implantable Cardioverter Defibrillators (ICD)**
- Seven (7) patient conditions with Class I recommendations
- Ten (10) patient conditions with Class IIa recommendations

**Cardiac Resynchronization Therapy (CRT)**
- One (1) patient condition with Class I recommendation
- Two (2) patient conditions with Class IIa recommendations

### Recommendations for ICD Implantation

#### Class I:
- Cardiac arrest secondary to VT/VF in absence of reversible cause
- Structural heart disease and sustained VT
- Syncopal of unknown origin and clinically relevant VT/VF @ EPS
- LVEF < 35%, CAD, 40 days post MI, NYHA II or III
- LVEF ≤ 35%, non-ischemic DCM, NYHA II or III
- LVEF < 30%, CAD, 40 days post MI, NYHA I
- LVEF < 40%, CAD, inducible VF or VT @EPS

#### Class IIa:
- Unexplained syncope, significant LV dysfunction, non-ischemic DCM
- Sustained VT and normal or near-normal LVEF
- HCM with 1 or more risks for SCD
- ARVD with 1 or more risks for SCD
- LQTS and syncope on beta-blocker, or VT/VF
- Non-hospitalized patients awaiting transplantation
- Brugada Syndrome and syncope or VT
- Catecholaminergic PMVT, syncope, and/or sustained VT while on beta-blocker
- Sarcoid, Giant-Cell Myocarditis, Chagas Disease
Recommendations for CRT Implantation

Class I:

- LVEF ≤35%, QRS ≥ 0.12 seconds, NYHA III or ambulatory IV and sinus rhythm

Class IIa:

- LVEF ≤35%, QRS ≥0.12 seconds, NYHA functional Class III or ambulatory IV and AF
- LVEF ≤35%, NYHA functional Class III & ambulatory IV symptoms and frequent dependence on ventricular pacing.

Use of Cardiac Resynchronization Therapy in Patients Hospitalized with Heart Failure

33,989 HF patient records analyzed (2005-2007). Only 12.4% of HF patients were discharged with a CRT device. Only 4.8% of HF patients with < 35% LVEF received a new CRT device. Lower CRT implant rates were noted in the NE US and African Americans.

“Studies have shown that, when used in combination with optimal medical therapy, CRT is associated with a 50 percent reduction in hospitalization for heart failure and a 36 percent reduction in mortality, or death. There are a lot of patients who potentially could benefit from the device who aren’t receiving it.”

Adrian F. Hernandez, M.D., M.H.S. Duke University Medical Center

Suggestions for Indentifying Patients At Risk of SCA

1) Place reminder stickers or inserts on charts for lipids.
2) Use a checklist to flag risk factors.
3) Commit to an EF that is valuable and interpretable.
4) Provide text field in the EHR to document EF, NYHA and patient discussion.
5) Ask all patients to “self report” on NYHA status.
6) Create a phrase in the EMR that, in patients with CHF or cardiomyopathy, places itself in a cardiology note and requires the identification of NYHA class and whether implantation of an ICD has been discussed.
7) Refer to an EP any patient that qualifies and hasn’t declined therapy.
8) Clear up ambiguous information regarding DNR and patient’s desire for “conservative treatment.”
Post Test Questions
1. How long does a patient with a newly implanted BMS (Bare Metal Stent) need plavix?
   • A. 1 Week
   • B. 1 month
   • C. 6 Months
   • D. 12 Months

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