EKGs, Stress Tests and Echos

Valerie R. Kaufman, MD, DBIM, FACC
Vice President & Medical Director

Colin DeForge
Executive Director, Underwriting

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Underwriting Cardiac Disease

- EKG
- APS
- Labs
- Stress Test
- BNP
- Echo
- Cardiac MRI
- Part 2

Template from www.presentationmagazine.com
. . . Putting the Pieces Together
Agenda

- Review of cardiac anatomy and physiology
- EKG overview
- Stress test overview
- Echocardiogram overview
- Case Studies
PreTest! True or False?

1. The EKG is no longer a useful risk assessment tool since echocardiograms and BNP are widely available.

2. An inadequate increase in the heart rate (less than 85% predicted maximum) invalidates the findings on a regadenosine (Lexiscan) stress test.

3. The echocardiogram gives a real time view of the motion of the myocardium and cardiac valves.
The heart is fixed within the body by the major vessels attached to it and by the fibrous skeleton provided by the heart valves and their supporting annuli. The ventricles and atria are free to enlarge and contract against the fixed plane.
The conduction system of the heart consists of specialized fibers that are adapted to the conduction of the electrical impulse at a much faster rate than that of muscle tissue. The SA node generates an impulse which spreads throughout the atria and on to the AV node. The AV node reconstitutes the impulse and delays transmission to the ventricles to allow the atria to finish contracting.
Cardiac Cycle

- **Electrical**
  - SA node fires
  - Impulse spreads throughout atria and to AV node
  - AV node reconstitutes/slows
  - Impulse enters Bundle of His, bundle branches, Purkinje fibers
  - Conduction system repolarizes

- **Mechanical**
  - Atria contract (systole)
  - Pressure increases in ventricles, closing mitral and tricuspid valves; atria relax (diastole)
  - Ventricles contract (systole); aortic and pulmonic valves open
  - Ventricles relax (diastole), aortic and pulmonic valves close
  - Mitral and tricuspid valves open and ventricles fill passively
  - Atrial “kick” contributes 10-15% of cardiac output

The Hard-Working Heart

- The myocardium is constantly working
  - Pumps about 5 liters of blood/minute
  - About 100,000 heartbeats/day
  - Pumps about 2,600 gallons of blood/day

- The coronary arteries must provide adequate oxygen and nourishment

- Cardiac reserve
  - Cardiac output may be increased to as much as 35 liters/minute
  - Normal coronary arteries can increase myocardial blood supply 5-6 fold if needed
  - Early stages of disease of the heart often associated with reduced cardiac reserve
  - Cardiac reserve diminishes with aging
  - Atrial fibrillation reduces cardiac output by 10-15%
Categories of Cardiac Disease

- Coronary artery disease
- Valvular heart disease
- Cardiomyopathy
- Congenital heart disease
- Hypertensive heart disease
- Primary Arrhythmias
The Electrocardiogram

EKG, ECG
Electrocardiogram (EKG, ECG)

- An EKG is a recording of the electrical activity of the heart.
  - 12 different leads “look” at this activity from different perspectives
  - Voltage is graphed against time
  - Voltage polarity (negative or positive) indicates direction of the flow of the electrical impulse.

- The EKG’s primary purpose is to determine the heart rate and rhythm
  - Rate: how fast the heart is beating
  - Rhythm: determines the origin of the pacing impulse and the integrity of the conduction system

- The EKG can give indirect information about other conditions
  - Ischemia
  - Infarction or other scarring
  - Hypertrophy
  - Electrolyte imbalance, drug effects
  - Pericarditis
  - Heart position in the chest (COPD, dextrocardia, chest wall deformities)
EKG Waves and Complexes

The **P wave** corresponds with atrial activation. The **PR interval** is the time from the activation of the atria to the activation of the ventricles. It indicates the time it takes the impulse generated in the SA node to travel through the atria, into and out of the AV node.

This is followed by the **QRS complex** (Q, R and S waves), which corresponds to the activation of the ventricle.

The **ST segment** and **T wave** correspond with ventricular repolarization.

The **QT interval** is the time it takes for ventricular activation and repolarization.
EKGs Were First Developed in 1903

EKGs were first developed by Willem Einthoven in Holland. Einthoven used a light projected on a moving mirror and reflected on a fluorescent plane to visualize the EKG.
The standard positioning of the EKG leads is not random. The leads are arranged in a standard way designed to provide adequate and consistent EKG readings. This consistency is required for pattern recognition and interpretation of the EKGs.

The EKG Waves

- **P wave**, atrial contraction starts
- **QRS complex**, ventricular contraction
- **T wave**, ventricular contraction ends
Underwriting EKG Findings

Considerations
- EKG findings provide direct information about the electrical activity of the heart only.
- Normal variants must be differentiated from potentially abnormal findings.
- EKG findings should be interpreted in context of other available information.

The Questions
- Is it a new finding?
- Any other findings on the EKG?
- Is there a known history of cardiac disease, and has there been any cardiac evaluation?
- What other medical history is there?
- What are the potential causes?
- What are the potential complications?
Case Study: Right Bundle Branch Block (RBBB)

- 62 year old man, nonsmoker, 6’2, 212 lbs (BMI 27.2)
- Sent to reinsurance due to EKG
  - Known to have a RBBB for at least 7 years (found on screening EKG)
  - No cardiac symptoms or history
  - No significant medical history
Rabbit Ears
Underwriting EKGs: The Questions

- Is it a new finding?
- Any other findings on the EKG?
- Is there a known history of cardiac disease, and has there been any cardiac evaluation?
- What other medical history is there?
- What are the potential causes?
- What are the potential complications?
Causes of RBBB

- Conduction system disease
- CAD, especially involving right coronary artery
- Right ventricular hypertrophy
- Pulmonary embolism
- Congenital heart disease (especially Tetralogy of Fallot, ASD)
- Myocarditis/Cardiomyopathy
- HTN
Underwriting EKGs: The Questions

- Is it a new finding? No, present x 7 years
- Any other findings on the EKG? No
- Is there a known history of cardiac disease, and has there been any cardiac evaluation? No history of cardiac disease, no evaluation
- What other medical history is there? Nothing significant
- What are the potential causes? ? Conduction system disease
- What are the potential complications? Progressive conduction system disease, underlying heart disease
Stress Tests
**Stress Testing**

- **Purpose:** to assess ability of coronary arteries to increase flow compared to rest/baseline. Can be used to screen for or to follow known CAD

- **Stress (increases demand or causes flow differential)**
  - Exercise by treadmill or bicycle - increases heart rate, BP and contractile state
  - Dobutamine – works much like exercise to increase heart rate, BP and contractile state
  - Vasodilators – adenosine, regadenosine (Lexiscan), dipyridamole

- **Monitoring for results**
  - EKG
  - Echo
  - Nuclear (MPI)
Vasodilator Stress Tests

- Includes adenosine/regadenosine and dipyridamole

- Mechanism
  - Dilate coronary arteries
  - Flow increases more in normal vessels than in diseased vessels
  - Relative differences in perfusion show up on imaging
  - Rarely induce actual ischemia
  - Often combined with low level walking as this may reduce side effects

- How differs from exercise stress test
  - Heart rate and BP not expected to increase significantly
  - EKG not expected to show ischemic changes
  - Symptoms more likely to be side effect of drug than due to ischemia
Stress Testing as a Screen for CAD

- Accuracy of any screening test depends on
  - Prevalence of disease
  - Sensitivity and specificity of test

- Sensitivity = True positive rate
- Specificity = True negative rate

- Exercise EKG: Sensitivity and Specificity in the range of 60 – 75%
- Imaging studies: Sensitivity and specificity in the range of 80 – 90%
Stress Testing in Known CAD

- Indicated to assess significance of new symptoms

- Could be considered
  - Evaluation of incomplete revascularization
  - Assessment of the adequacy of medical therapy
  - Need to evaluate coronary status in anticipation of major noncardiovascular surgery

- Should not expect “routine” follow up stress testing in stable CAD

The Exercise EKG – More Than ST Segments!

- Exercise capacity: duration and amount of exertion
- BP response
- Symptoms
- ST segment changes
- Arrhythmias
- Heart rate recovery
## Interpreting Stress Tests

<table>
<thead>
<tr>
<th>Type of Test</th>
<th>Component</th>
<th>Criteria for Positive Test</th>
<th>Other Adverse Findings</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Exercise or Dobutamine</strong></td>
<td>EKG</td>
<td>• ST depression</td>
<td>• ↓ exercise capacity</td>
</tr>
<tr>
<td></td>
<td></td>
<td>• Anginal pain</td>
<td>• Abnormal BP response</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>• Arrhythmias</td>
</tr>
<tr>
<td></td>
<td>Nuclear imaging</td>
<td>• Reversible perfusion defect</td>
<td>• LV dilatation</td>
</tr>
<tr>
<td></td>
<td></td>
<td>• Transient regional wall motion abnormality</td>
<td>• Fixed perfusion defect</td>
</tr>
<tr>
<td></td>
<td>Echo imaging</td>
<td>• Transient regional wall motion abnormality</td>
<td>• Failure to increase EF</td>
</tr>
<tr>
<td></td>
<td></td>
<td>• Failure to increase EF</td>
<td></td>
</tr>
<tr>
<td><strong>Vasodilator</strong></td>
<td></td>
<td>• Positive imaging findings</td>
<td>• Symptoms such as CP, SOB – often side effect from drug rather than due to ischemia</td>
</tr>
<tr>
<td></td>
<td></td>
<td>• Do NOT expect EKG changes</td>
<td></td>
</tr>
</tbody>
</table>
Not all ST Depression is CAD . . .

- Causes of ST depression on exercise EKG
  - CAD
  - Hypertensive blood pressure response
  - LVH
  - Cardiomyopathy
  - Bundle branch block
  - Other depolarization abnormalities, like preexcitation (such as WPW)
  - Electrolyte imbalance
The Echocardiogram
Echocardiogram

- Also known as cardiac ultrasound
- Noninvasive
- No radiation
- Readily available
- Provides real time images of the heart
- Cost is a consideration
Traditional Echo Modalities

- M mode – 1 dimensional “icepick” view through various planes of the heart
  - Measure chamber sizes and ventricular wall thickness

- 2 D – 2 dimensional, real time motion
  - Measure chamber sizes and ventricular wall thickness

- Doppler – assesses characteristics of blood flow
  - Assess valve function
  - Assess pressures
  - Assess for abnormal flow
Enhancements to Echocardiography

- Addition of color
- Echo contrast (microbubbles)
- Transesophageal approach
- Tissue Doppler
- 3 D imaging
- Speckle and strain imaging
The computer imaging system calculates sizes and displays them automatically in M mode. The left ventricle is being measured in this example. Also note the timing EKG tracing.
These are some of the echocardiographic views available through different transducer positioning. Each view shows only a narrow plane of tissue through the heart. Some of these views can be difficult to obtain depending on the size and shape of the patient’s chest. Some structures can be only be seen in specific views.
Doppler Echocardiogram

- Doppler of a ventricular septal defect showing the abnormal flow of blood across the defect in the septum.
- The EKG tracing indicates (red mark) the point of the cycle where this picture was taken.
- Very small defects with little flow may require microbubble contrast enhancement.

Doppler evaluation of the speed of flow across a valve or septal defect can be used to calculate the size of the valve opening or defect. Higher rates of flow indicate tighter openings and vice versa.
Echocardiogram 2D and 3D – in motion

Above: Sketch labeling the 3D picture. Mitral and aortic valves on right of image, tricuspid valve left.
Information Provided by the Echo

- Cardiac chamber sizes and wall thickness
- Appearance of the heart valves
- Assessment of valvular function and dysfunction
- Assessment of systolic and diastolic function of the myocardium
- Assessment of presence, direction and speed of blood flow
- Estimation of pressures within cardiac chambers
Limitations of the Echocardiogram

- **Body habitus**
  - Chest wall deformities such as pectus excavatum and kyphoscoliosis distort anatomy
  - Obesity makes it difficult for sound waves to penetrate
  - Sound waves don’t travel well in air – hyperinflated lungs make it difficult to acquire clear images (COPD, asthma, ventilator patients)

- Be careful when “technically difficult” study

- Skill and patience of the examiner and methodology of interpreter
## Normal Values

<table>
<thead>
<tr>
<th></th>
<th>Description</th>
<th>Range</th>
</tr>
</thead>
<tbody>
<tr>
<td>LVIDd, LVEDD</td>
<td>Left ventricular internal dimension in diastole</td>
<td>3.9 – 5.3 cm (women)</td>
</tr>
<tr>
<td></td>
<td>Left ventricular end diastolic dimension</td>
<td>4.2 – 5.9 cm (men)</td>
</tr>
<tr>
<td>LVIDs, LVESD</td>
<td>Left ventricular internal dimension in systole</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Left ventricular end systolic dimension</td>
<td></td>
</tr>
<tr>
<td>IVSd</td>
<td>Interventricular septum in diastole</td>
<td>0.6 – 0.9 cm (women)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>0.6 – 1.0 cm (men)</td>
</tr>
<tr>
<td>IVSs</td>
<td>Interventricular septum in systole</td>
<td></td>
</tr>
<tr>
<td>LVPWd</td>
<td>Left ventricular posterior wall in diastole</td>
<td>0.6 – 0.9 cm (women)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>0.6 – 1.0 cm (men)</td>
</tr>
<tr>
<td>LVPWs</td>
<td>Left ventricular posterior wall in systole</td>
<td></td>
</tr>
<tr>
<td>LA</td>
<td>Left atrium</td>
<td>2.7 – 3.8 cm (women)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>3.0 – 4.0 cm (men)</td>
</tr>
</tbody>
</table>

Lang RM, Bierig M et al. Recommendations for Chamber Quantification: a report from the American Society of Echocardiography’s Guidelines and Standards Committee and the Chamber Quantification Writing Group, developed in conjunction with the European Association of Echocardiography, a branch of the European Society of Cardiology. J Am Soc Echocardiogr 2005;18:1440-1463.
# Value of the Echo in Different Types of Heart Disease

<table>
<thead>
<tr>
<th>Type of Disease</th>
<th>How an Echo can Help</th>
</tr>
</thead>
<tbody>
<tr>
<td>Coronary Artery Disease</td>
<td>• Regional wall motion abnormalities</td>
</tr>
<tr>
<td>Valvular Heart Disease</td>
<td>• Valve structure</td>
</tr>
<tr>
<td></td>
<td>• Degree of dysfunction (stenosis, regurgitation)</td>
</tr>
<tr>
<td></td>
<td>• Compensatory changes (dilatation, hypertrophy)</td>
</tr>
<tr>
<td>Cardiomyopathy</td>
<td>• Type and severity</td>
</tr>
<tr>
<td>Congenital Heart Disease</td>
<td>• Type and severity</td>
</tr>
<tr>
<td></td>
<td>• Compensatory changes (dilatation, hypertrophy)</td>
</tr>
<tr>
<td>Hypertensive Heart Disease</td>
<td>• Compensatory changes</td>
</tr>
<tr>
<td>Primary Arrhythmias</td>
<td>• Excludes structural abnormalities</td>
</tr>
</tbody>
</table>
Review of Pretest

1. The EKG is no longer a useful risk assessment tool since echocardiograms and BNP are widely available. **FALSE**

2. An inadequate increase in the heart rate (less than 85% predicted maximum) invalidates the findings on a regadenosine (Lexiscan) stress test. **FALSE**

3. The echocardiogram gives a real time view of the motion of the myocardium and cardiac valves. **TRUE**
Putting the Pieces Together

- Consider all the different types of cardiac disease, not just CAD
- Consider all the pieces of evidence available
- Recognize the limitations of the various tests
- Give more weight to more predictive tests
- Go with the “preponderance of the evidence”
- Stay focused on assessing risk rather than making a diagnosis
. . . On to Case Studies