Cardiac CTA
Disclosures

• None
Importance of Primary Prevention

- Over 1 million M.I.’s per year
- For almost 1/2, infarction is first symptom
- For 150,000 first symptom is sudden death
- 68% of Infarcts are in vessels with < 50% stenosis
Early Appearance of Coronary Atherosclerosis: Bogalusa Heart Study

Prevalence of Fibrous Plaque Lesions

P = .001 for trend

Age (Years)

Our current tests for dx CAD

• Stress test 60% accurate
• Cardiac SPECT Imaging (MPI)
  o 40% have Attenuation Defects
  o Sensitivity 87%
  o Specificity 75%
  o 80-85% accurate for ischemia
  o Negative ETT (8 mets) and MPI both associated with “good” short term outcomes
Our current tests for dx CAD (Cardiac Cath)

- Cardiac Cath (lumanogram) can miss CAD...is good for % stenosis
- ~25% -50% are Normal
- ~8 Million Caths/yr at $4000/Cath
- ~$4 Billion/yr
We need better tools
Who was at greater risk for the early development of heart disease?

Jim Fixx - marathon runner, exercise advocate, author - dead at 53 of a heart attack

Sir Winston Churchill - broke every tenet of “healthy life style” - dead at age 91
Chest Pain in the ED
Chest pain in the ED

- 8-10 million ED chest pain presentations/yr in US
- 90% have a final diagnosis other than MI or USA
- 60% will have unnecessary admission and testing to exclude ACS
- High cost to society ($5 billion/year), hospital resources not available for those who need it
- But, big downside for missed diagnosis (up to 5%)
- General agreement in US, <1% miss rate is needed
- National average rate of revascularization in standard therapy group ≈ 42%
Acute Chest Pain Presentation (Issues)

• Physician wants to know:
  o Is the patient having an ACS/USA?
  o Risk of short term morbidity?
  o Need for acute intervention?
  o Is there opportunity for long term risk assessment?

• Practical considerations:
  o When is this patient getting out of ER?
  o Will something bad happen if sent home?
  o Should we defer the decision?
    • Admit
    • Put in observation unit
Our current tests for dx CAD

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We need better tools…

CCTA?
Can Cardiac CTA help us manage chest pain presentations better?

- There have been numerous studies evaluating the use of CTA for patients presenting with acute chest pain.
- It has been shown to be safe and cost effective.
- CMS and most payers have endorsed this specific application of CT.
Coronary CTA

MPR of the LAD & 1st Diagonal

MPR of the RCA

Curved Reformat of LAD
Mixed Plaque LAD

1st Diagonal Branch

RCA

LCX

LMA

LAD

Curved Reformat LAD
## CT Evidence

<table>
<thead>
<tr>
<th>Study</th>
<th>Risk Group</th>
<th>N</th>
<th>Follow Up</th>
<th>30 Days</th>
<th>60 Days</th>
<th>Methodology</th>
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<tbody>
<tr>
<td>Goldstein, JACC 2007</td>
<td>low-intermediate risk</td>
<td>99</td>
<td>6 months</td>
<td>0%</td>
<td>11%</td>
<td>RCT, stress 26-70%</td>
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<tr>
<td>Hoffmann, Circ 2006, JACC Imaging 2011</td>
<td>low-intermediate (very few high)</td>
<td>368</td>
<td>2 years</td>
<td>0% 30-days, 4.6% 2 years</td>
<td>9%</td>
<td>prospective obs, CT not for decisions</td>
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<tr>
<td>Hollander, Ann Emer Med 2007</td>
<td>low-intermediate (most low)</td>
<td>588</td>
<td>1 year</td>
<td>0% 30 days, 0.2% 1 year</td>
<td>not recorded</td>
<td>observational</td>
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<tr>
<td>Rubenshtein, Circ 2007</td>
<td>intermediate-high</td>
<td>58</td>
<td>15 months</td>
<td>0% 30 days, 3% 15 months</td>
<td>0%</td>
<td>observational</td>
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<tr>
<td>Beigel, Isr Med Assn J. 2010</td>
<td>low-intermediate</td>
<td>445</td>
<td>mean 236 days</td>
<td>unknown, overall</td>
<td>7%, retrospective</td>
<td>observational</td>
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<tr>
<td>Winchester, Int J Card, 2010</td>
<td>low to intermediate</td>
<td>50</td>
<td>3 months</td>
<td>0</td>
<td>0%</td>
<td>observational</td>
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<tr>
<td>Goldstein, JACC 2011</td>
<td>low-intermediate</td>
<td>376</td>
<td>6 month</td>
<td>0.6%</td>
<td>unknown</td>
<td>RCT, multictr</td>
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<tr>
<td>Shuman, AJR 2010</td>
<td>low-intermediate</td>
<td>75</td>
<td>12 months</td>
<td>0%</td>
<td>8%</td>
<td>observational</td>
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<tr>
<td>Hansen, Heart Lung Circ 2010</td>
<td>intermediate</td>
<td>85</td>
<td>mean 355 days</td>
<td>0%</td>
<td>unknown</td>
<td>observational</td>
</tr>
</tbody>
</table>

Penn Medicine
CT-STAT

- 700 patients
- 54% reduction in length of stay
- 38% reduction in cost
ACNH-PA (NEJM 2012)
CTA c/w usual care

- Higher ER discharge rate 50% vs. 23%
- Shorter length of stay 18 vs. 24 hours
- Higher rate of detection of CAD 9% vs. 3.5%
- Total cost similar
NCR Data

• National average rate of revascularization in standard therapy group ≈ 42%

• Average rate of revascularization in group incorporating CTA ≈ 75%
SCOT Heart – Lancet 2015

• 4,146 patients evaluated in ED with CTA

• At 3 years, coronary events reduced by 50%

• Explained by identifying patients with non obstructive CAD and instituting risk factor modifications
Small, Vulnerable Plaques are responsible for Causing MI

RCA
Why Now?

• Equipment:
  o Faster Scanner, Larger detector, less artifacts
  o Able to do patients with faster HR
  o Major reductions in radiation and contrast dose
  o Computer speed allows almost instant acquisition, processing and image construction

• Automated work station with offsite access for reading

• Acceptance by payers / Reimbursement
Summary 1

• CTA in ER patients ↓ length of stay and ↓ ED cost
• CTA incorporation with chest pain management may not change number of PCI secondary to greater sensitivity c/w spect
• CTA significantly reduces number of normal caths (intervention rate 75% vs. 42%) secondary to greater accuracy
Summary 2

• Decreases 30 day revisits to ER and 30 day readmissions (patients and doctors believe results)
• 1-2 yr warranty (<1% event rate)
• CTA decreases 3 yr event rate by 50% secondary to detection of early CAD and institution of risk factor modification
Summary 3

- ↓ long term costs on a population basis
- PCI rate may not change (greater detection rate of CAD)
- Future potential of the “triple r/o”
- Numerous non CCTA applications
Coronary Calcium Score and Treatment of Dyslipidemia
Atherosclerotic Plaque Development

Stary Classification of Atherosclerotic Plaques

I        II                      III         IV        V_{a,b,c}        VI_{a,b,c}

Regression Possible

Potentially Vulnerable

Normal  Early  Lipid rich  Internal rupture  Calcified shell  Calcified plaque  Vulnerable  Rupture  Thrombus  Myocardial infarction  Obstructive

Fatty streaks  White blood cells  Red blood cells  Lipid rich plaque  Calcium  Scar  White blood cells  Platelets and fibrin  Inflammation and calcification  Scar development with calcification
NCEP CAD Risk Factors

- High LDL
- Cigarette smoking
- Hypertension (BP ≥140/90 mmHg or on antihypertensive medication)
- Low HDL cholesterol (<40 mg/dL)†
- Family history of premature CHD
  - CHD in male first degree relative <55 years
  - CHD in female first degree relative <65 years
- Age (men ≥45 years; women ≥55 years)
- Diabetes
<table>
<thead>
<tr>
<th>Risk Category</th>
<th>LDL-C Goal</th>
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<tbody>
<tr>
<td>Low (no CVD)</td>
<td>&lt;160</td>
</tr>
<tr>
<td>&lt; 2 RF</td>
<td></td>
</tr>
<tr>
<td>Mod (no CVD)</td>
<td>&lt;130</td>
</tr>
<tr>
<td>&gt; 2 RF</td>
<td></td>
</tr>
<tr>
<td>High (+ CVD)</td>
<td>&lt;100</td>
</tr>
<tr>
<td>Or DM</td>
<td></td>
</tr>
<tr>
<td>Very High (+CVD)</td>
<td>&lt;70</td>
</tr>
<tr>
<td>Plus tobac,DM</td>
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Limitations of “Lipids” in CAD

Significant overlap of LDL levels exists between populations with and without CAD

- Framingham Heart Study - 26 year follow-up data

  80% of MI patient population had similar cholesterol levels as those who did not have an MI\(^1\)

Elevated LDL may not be the primary lipid abnormality associated with CAD\(^2\)

- Only 25% of premature CAD is attributable to elevated LDL values

\(^1\)Castelli W, *Atherosclerosis* 1996; 124: S1-S9
Problem

• NCEP risk factor guidelines correctly identify only 50% of subjects with CAD
• (same as a coin flip)
Implications

• All the determinants of plaque formation and progression are not known
• We cannot equate abnormal lipids with disease, and normal lipids with absence of disease
• Other factors are important
• We need better methods for predicting CAD
Clinical ASCVD

N

LDL-C ≥ 190 mg/dL

Yes

No

Diabetes Type 1 or 2

Age 40-75 y

Yes

No

Estimate 10-y ASCVD risk with pooled Cohort equations

≥ 7.5%

estimated 10-y ASCVD risk and age 40-75 y

Yes

No

Age ≤ 75 y

High intensity Statin

Age > 75

Moderate intensity statin

High intensity statin

Moderate intensity statin

Moderate to high intensity statin
Problem

• Based on same risk factors as ATP III
• ASCVD scoring works on a population basis.
• Leads to a significant number of pts taking statins who don’t have disease.
• Still misses CAD in some pts without traditional risk factors.
Are there better tools?
Newer Tools for Risk Assessment

- Homocysteine
- CRP
- Lp(a), Apo B
- HDL/Triglycerides/Non-HDL Chol.
- LDL Particle Size (NMR, VAP)

- **Coronary Calcium Score**
Coronary Calcium Scores

No Calcification

Severe Calcification

PA
Ao

LA
RCA Stenosis
History:
• 48 y/o male, active, but recent atypical chest pain
• TMET done by family physician – negative
• lipids – TC = 200, HDL = 48, LDL = 120, TG = 130
• “high” TC in past, but responded well to diet
• CAD in both paternal grandparents at ages <60
• does not smoke
• normotensive
• mother and father with no known heart disease
Case Presentation

NCEP ATP-III

Estimated Risk Using Framingham Analysis

AGE = 3 points, TC = 5 points, HDL = 1 point

SUM = 9 points or 11.5% over ten years
Case Presentation

Recommendations: [conventional assessment]

- no further cardiac testing
- continue to avoid smoking
- LDL <130 mg/dl as goal

**NO DRUGS FOR YOU!**
ASCVD Risk Estimator 10 yr risk 3.0%
No statin indicated
CCTA
History:

- 55 y/o male, active, but recent atypical chest pain
- TMET done by family physician – negative
- lipids – TC = 220, HDL = 48, LDL = 140, TG = 130
- does not smoke
- hypertension
- father had CAD at age 55
# NCEP ATP III Recommendations

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ASCVD Risk Estimator 10 yr risk
8.8%

Atorvastin started
Coronary CTA
We need better tools
Stary Classification of Atherosclerotic Plaques

I        II                      III         IV      V

Regression Possible

VI_a,b,c

Potentially Vulnerable

Normal  Early  Lipid rich  Internal rupture  Calcified  Calcified plaque  Vulnerable  Rupture Thrombus  Myocardial infarction  Obstructive

Fatty streaks  White blood cells  Red blood cells  Calcium  Scar  White blood cells  Platelets and fibrin

Inflammation and calcification

Scar development with calcification
Apparently healthy population men >45 women > 55 year

Step 1
- Very low risk
- Risk Factors present (ASCVD calc >7.5%)

Coronary Artery Calcium Score

Step 2
- CACS = 0
- Positive test: CACS > 1
  - CACS 1-10
  - CACS > 11 < 100
  - CACS 101-400
  - CACS > 400

Step 3
- Low Risk
- Low-Mod Risk
- Moderate Risk
- High Risk
- Very High Risk

MESA 10 yr risk
- < 7.5%
- 7.5%
- 10-20%
- > 20%
- > 20%

LDL goal
- < 160
- < 130
- < 100
- ≤ 70
- ≤ 70

Statin Rx
- Low dose
- Moderate Intensity
- High Intensity
What’s the outcome data?
"Risk" of MI, SCD or Ischemia: (asymptomatic adults)

Calcium Score

Odds Ratio

0 5 10 15 20 25 30

'1-49 >50 >80 >160 >600

2.7 6.9 14.3 19.7 20.2

Arad: JACC 2000;36:1253
MI or SCD in Patients with Very Abnormal Cardiac Test Results

- Asymptomatic Subjects, No Intervention, 36 m. f/u, n=104

* Wayhs JACC 2002;39:225-30
Annualized Rates of Progression - EBT

- Untreated: 30.2%/year
- Treated: 12%/year

Composite of 9 studies – weighted average

n = 292
Ranged: 5%–20%
Individualized Therapy
(not based on population statistics)

• Pts with high cholesterols but normal Ca score/CCTA may not need any drugs.
• Pts with low LDL (even less 100) but with Calcium or soft plaque on CCTA may need drugs.

• TREAT THE PT, not the population
CCTA or Ca Score?
Indication for Ca Score (low to intermediate risk population)

- A tool to help in Primary Prevention
- Establish a Dx of CAD and better estimate risk.
  - Begin Aggressive risk factor modification (exercise, diet etc.)
  - Begin pharmacologic interventions (statin, aspirin etc.)
  - Perhaps, avoid life long medications if not needed
Indication for CCTA
(low to intermediate risk population)

• Persistent symptoms in pts with low/int clinical suspicion of CAD
• Abnormal stress test in pts with low/int clinical suspicion of CAD
• ED pts with c/p
• As alternative to Pharm stress MPI in symptomatic pts
Other uses for CTA
What is the Cause of chest pain?

- Pulmonary Embolism
- Aortic Dissection
- Hiatal Hernia
- Pulmonary Pathology
- Anomalous Coronary Artery Anatomy
- Cardiac Cysts, Masses, Clots
The Triple R/O”

10 million to ED with chest pain a year
10-15% have MI
1M admitted with no disease
Over $5 billion in US.
Leading cause for litigation in ED…average settlement ~ 1 to 2 million
**Triple Rule Out** (in Single acquisition)

- **Aortic Dissection**
- **Pulmonary Embolism**
- **Coronary Artery**

70 year old male with chest pain
Heart Rate: 65-71 bpm
292 mm coverage in 12.6 sec
SnapShot Burst at 0.35sec
Dissecting Aortic Aneurysm
Congenital Coronary Anomalies
Anomalous LMA

RCA  LAD  LCX
Anomalous Coronary Artery

Curved MPR View of the Left Main from RCA

3DVRT View of the RCA from the Left Main
Coronary Artery Anomalies

RCA arising from LM and coursing Anterior between Aorta (Ao) and Pulmonary trunk (PT)

Ropers et al
Am J. Cardiol
2001;87:193-197
Conventional Aortogram:
Congenital Coarctation of the Aorta
Evaluation of Post CABG Patients

- Location of SVG prior to cath
- Patency of SVG
  - Sensitivity (Sen) 96%
  - Specificity (Spec) 95%
  - Positive Predictive Value (PPV) 81%
  - Negative Predictive Value (NPV) 99%

Schlosser, JACC 2004;44:1244
VENOUS BY-PASS GRAFT

SVG to LAD
Bypass Graft
Bypass Graft

LIMA to Diagonals

RIMA to PDA
Indication for CTA

Stents
LMA/LCX Stent Follow-Up

Curved reformat patent stent in LMA/LAD/LCx

Curved reformat patent stent in LMA/LAD

MPR of LCX showing patent stent
Follow-Up Stent
Occluded RCA Stents

RCA with occluded vessel between stent

Occluded RCA

MPR view demonstrating stents and occlusion
Stent Patency

Courtesy of Toyohashi Heart Center, Japan
Cardiac structures and Function
Atrial Appendage Clots
Valve Disease
LV Function

ED Volume (90%) : 201 cm³
ES Volume (30%) : 98 cm³
Stroke Volume : 103 cm³
Ejection Fraction : 51%
• Pre Atrial Fibrillation ablation
• Pre Bi-Ventricular Pacemaker
Pulmonary Veins and Atrial Fibrillation Ablation
Carotid Artery Stenosis

90% stenosis with soft plaque & calcified plaque
Limitations of Cardiac CTA

- High Calcium Scores
- Fast Heart Rates---goal < 60
  - option give 50-100 mg Metoprolol
- Afib, frequent PVC’s
- Renal Insufficiency
- Stents---can’t always see patency
“No Free Lunches”

- Radiation Dose
- Dye Load
- Insurance coverage
# Radiation Dose

<table>
<thead>
<tr>
<th>Procedures</th>
<th>Modality</th>
<th>Effective Dose (mSv)</th>
</tr>
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<tbody>
<tr>
<td>Ca Scoring</td>
<td>MDCT</td>
<td>1</td>
</tr>
<tr>
<td>CTA</td>
<td>MDCT (with prospective gating)</td>
<td>3</td>
</tr>
<tr>
<td>PET</td>
<td></td>
<td>4.9</td>
</tr>
<tr>
<td>Cardiac Cath</td>
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<td>2-15</td>
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<tr>
<td>Stress Test</td>
<td>MIBI</td>
<td>5-12</td>
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<tr>
<td>Stress Test</td>
<td>Thallium</td>
<td>25</td>
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<tr>
<td>Chest X-Ray</td>
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<td>0.1</td>
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Summary...Cardiac CTA Today

• To help with risk stratification and to help guide medical therapy targets
• Evaluate persistent atypical c/p with normal ETT
• F/U of abnl ETT in assx patient with low to mod risk
• Graft location and patency
• Congenital coronary anomalies
Summary...Cardiac CTA in the Future

- E.R. evaluation of c/p..."triple r/o"
- Primary evaluation of patients with c/p
- Stent patency
- EPS planning
- Cardiac structures
- Cardiac perfusion and viability
What is the question?

- 1) Is there CAD?
  - Ca score/CTA

- 2) What is causing the pain?
  - CTA

- 3) Is there ischemia?
  - Nuclear

- 2) Given CAD, what is the near term prognosis?
  - Nuclear
IS THAT THE VIDEO GAME WHERE YOU HELP FIGHT HEART DISEASE?

DOCTOR'S LOUNGE

YEAH... IT'S CALLED "PLAQUE-MAN"
Thank you