Assessment
Of Myocardial Viability

James K. Min, MD FACC
President, Society of Cardiovascular Computed Tomography
Associate Professor of Medicine, UCLA School of Medicine
Associate Professor of Medicine and Imaging, Cedars-Sinai Medical Center
Co-Director, Cardiac Imaging, Cedars-Sinai Heart Institute
Director, Cardiac Imaging Research, Cedars-Sinai Medical Center

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Typical Viability Scenario

- 55 year-old male with severe chest pain; dragged into hospital by wife after 1 day
- Cath: occluded LAD, low BP → balloon pump
- Bedside echo: EF 20%
Management Dilemma

- Balloon pump gradually weaned, but ongoing low-output symptoms
- Bypass surgery (CABG) is being considered to improve blood flow to hypocontractile myocardium

- CMR ordered to assess viability . . .
Questions We Really Want Answered

• *Will this patient do better with coronary revascularization?*
  – Relieving symptoms?
  – Do better with an ICD?
  – Experience improved long-term survival?

• *Will this patient improve (prognosis and therapeutic benefit)?*
  – Improve LV function? Not have worsening LV function?
  – Respond to medical therapy?
  – Do better with an ICD?
Premise for Viability Imaging

• Sick myocardium is substrate for -
  – Heart failure
  – Arrhythmias
  – Cardiovascular death
• If myocardium can be restored to health (i.e. if viable), then outcomes should improve
What is myocardial viability?

- Absence of scar: LGE-CMR
- Integrity of cell membranes: LGE-CMR, thallium scintigraphy
- Metabolic activity: FDG-PET
- Demonstration of inotropic reserve: dobutamine stress echo or CMR
- Intact generation of high-energy phosphates: P-MRS
- Lack of sodium accumulation: Na-MRS

*Evaluated in STICH Viability study*
(1) CMR is validated against a pathologic reference standard


(2) CMR LGE depicts a logical stepwise pathophysiologic process

Normal myocardium

Acute infarction

Scar

Intact cell membrane

Ruptured cell membrane

Collagen matrix
50 pts with LV dysfunction undergoing coronary revascularization (NEJM 2000;343:1445-53)

Wall Motion Improvement Predicted by MRI?

MRI #1
- Cine MRI for wall motion
- DE-MRI for viability

PCI (32%) or CABG (68%)

18 ± 25D

MRI #2
- Cine MRI for wall motion

12 ± 5W
Prediction of Regional & Global Improvement

Likelihood of Wall Motion Improvement

Transmural Extent of Hyperenhancement

- 0%: 256/32
- 1-25%: 109/18
- 26-50%: 46/110
- 51-75%: 13/12
- 76-100%: 1/58

p < 0.0001 for trend

N Eng J Med 2000;343:1445-1453
(4) Infarct Transmurality Predicts Therapeutic Response


Wall Motion Improvement Predicted by MRI?

MRI #1
Cine MRI for wall motion
DE-MRI for viability

Beta blocker

MRI #2
Cine MRI for wall motion

6 months
Relation Between Transmural Extent of Scar and Contractile Improvement after Beta-Blocker Therapy

![Bar Chart]

- All Dysfunctional Segments
- Improved Contractility (%)

- Transmural Extent of Hyperenhancement (%)
- P<0.0001 for trend
(5) Infarct Surface Area by CMR Better Predicts VT Inducibility than LVEF

Stevenson WG. JCE 1995.  
(6) Scar is Substrate for Adverse Post-MI Remodeling

- acute MI (hours)
- infarct expansion (hrs to days)
- global remodeling (days to months)
(7) CMR Evaluates Effects on Coronary Microcirculation

- Essential for delivery of substrates/O$_2$ & washout of metabolites
MO and Recovery of Function Post-MI

Does Viability Imaging Improve Outcomes?

Significant reduction in cardiac events w/FDG-PET if management adhered to PET recommendations

Who Got a Viability Study in STICH?

• Caucasians (82% vs. 54%) [Fewer Asians (5% vs. 30%)]
• Atrial fib / flutter (15% vs. 10%)
• Better Med Tx (higher rates of beta-blocker, ACEI, statin, ASA use)
• No CMR viability data available
Back to Our Patient…

- 55 y/o male with recent MI; occluded LAD
- LGE-CMR ordered for viability assessment
Our Patient...

No viability in LAD territory
→ Left ventricular assist device → transplantation
Utility of CMR for Viability Imaging

• Direct relation with pathology
• Accurate imaging of infarct size/ viability
• Predicts functional response to revascularization
• Incremental utility vs. PET/ SPECT
• Targets therapeutic approaches to CM
• Identifies at-risk arrhythmogenic patients
• Examines effects of MI on coronary microcirculation
Thank you.
The Reports of Viability Imaging’s Death are Greatly Exaggerated…

- Randomization is key
- Multiple modalities need to be compared
- Management decisions based on viability should be standardized
Clinical Definition of Viability

QUESTION:
Is the anterior wall viable or not viable?
Definition of Viability

MRI: \( \frac{a}{a+c} \)

SPECT: \( \frac{a}{b} \)
Limited Spatial Resolution (Partial volume effects)

- If <50% of counts of remote region → Fixed (Infarct) by SPECT
- If <50% myocardium involved → No Infarct by SPECT

Infarct Morphology is Important

- Transmural extent of infarct predicts functional response
- DE-MRI is uniquely capable of imaging transmural extent of infarct
Microvascular Obstruction by LGE-CMR
You’re worse off without viability
Case: 46 y/o Caucasian Man

- **Chest pain**: Atypical chest pain prompted CT angiogram at OSH
  - Reported to have left main dissection
  - High-grade stenosis in LAD
  - Other coronaries reported as “moderate”
  - Now CP-free

- Self-refers to 2 cardiologists for 2nd and 3rd opinion
  - Coronary CT angiogram re-reviewed
STICH-Viability Trial

• 1,212 patients underwent physician-directed viability testing with SPECT or dobutamine echo
• 601 with usable test results

Bonow RO et al. NEJM 2011.
Relation Between Transmural Extent of Scar and Contractile Improvement after Beta-Blocker Therapy
Prediction of Global Improvement

\[ r = 0.75 \]

\[ p < 0.0001 \]

N Engl J Med
2000;343:1445-1453
Contrast-enhanced MRI and routine single photon emission computed tomography (SPECT) perfusion imaging for detection of subendocardial myocardial infarcts: an imaging study

- 5 dogs with CMR and SPECT prior to sacrifice
- Pathology infarct verification

Dog B

SPECT infarct

DE-CMR infarct

HISTOLOGY infarct

Lancet 2003;361:374-79
<table>
<thead>
<tr>
<th>SPECT</th>
<th>DE-CMR</th>
<th>HISTOLOGY</th>
</tr>
</thead>
<tbody>
<tr>
<td>No infarct</td>
<td>Infarct</td>
<td>infarct</td>
</tr>
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</table>
Myocyte Necrosis = Hyperenhancement

JACC 2000; 36:1985 - 91
Late Gadolinium Enhancement (LGE)

Thank you.
(4) Understanding of Atherosclerotic Adaptive Mechanisms, 1980s to Present

- Plaque localization
- Artery wall adaptive responses
- Shear stress regulation of artery size
- Atherosclerotic plaque evolution
- Autoregulation

**COMPENSATORY ENLARGEMENT OF HUMAN Atherosclerotic CORONARY ARTERIES**

*Seymour Glagov, M.D., Elliot Weisenberg, B.A., Christopher K. Zarins, M.D., Regina Stankevicius, M.P.H., and George J. Koletis, B.A.*

*NEJM 1987*

“*Function Follows Form*” Relationships
Vulnerable Plaque

Prototype high-risk plaque at risk of rupture.
Most Myocardial Infarctions Are Caused by Low-Grade Stenoses

Coronary stenosis severity prior to MI

- >70% Stenosis: 14%
- 50%-70% Stenosis: 18%
- <50% Stenosis: 68%

- In >50% of victims, the first symptom of asymptomatic atherosclerosis is sudden cardiac death or acute MI

Mild Plaques Cause Adverse Events

2,583 patients undergoing CCTA with \( \leq 50\% \) stenosis followed for 3.1 years

- >6-fold higher mortality for patients with 3-vessel mild CAD

Source: Lin et al., J Am Coll Cardiol 2011
Where do we go from here?
Increased hazards for ACM evident for those with low FRS and no medically modifiable risk factors

HR 4.40  
(95% CI 2.68-7.22)  
P < 0.0001

HR 3.20  
(95% CI 1.57-6.50)  
p = 0.001

Low FRS  
56.5%

No Tx CAD RF  
28.6%

Medically Modifiable CAD RF = diabetes, dyslipidemia, hypertension

Source: Min et al. ACC 2011 Scientific Sessions 2011; Chow et al. AHA 2011