MR Assessment of Myocardial Viability
Definition of Viability

• Clinical
  – Metabolism: Presence of glucose uptake
  – Perfusion / Perfusion reserve
  – Morphology: Wall thickness, wall thickening
  – Contractility: Recovery after treatment / Contractile reserve

• Histologic
  – Presence of living myocytes

Kim et al. Herz 2000
Merits of MRI as “one-stop exam”

- Morphology
- Function
- Perfusion
- Viability
- Coronary Artery
- Metabolism
- Flow Measurement
Merits of MRI as “one-stop exam”

- Morphology: T2 WI, T1 WI
- Function: Cine MR, Myocardial Tagging
- Perfusion: MR Perfusion (rest/stress)
- Viability: Delayed Enhancement, Low dose DSMR etc.
- Coronary Artery: Coronary MR angiography
- Metabolism: \(^{31}\text{P}, \, ^{1}\text{H} \text{MR Spectroscopy etc.}
- Flow Measurement: Coronary flow reserve
Merits of MRI as “one-stop exam”

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• Metabolism: $^{31}$P, $^1$H MR Spectroscopy etc
• Flow Measurement: Coronary flow reserve
MRI for Myocardial Viability

1. Detection of Myocyte necrosis or scar: Delayed Enhancement (DE) MRI

2. Assessment of LV function:
   - Myocardial mass: End-diastolic wall thickness
   - LV contractility: End-systolic wall thickening
   - Contractile (inotropic) reserve
I. Delayed Enhancement MRI

ECG Trigger

Non-selective 180° inversion

M_z Infarct

M_z Normal

Trigger delay

TI 200 - 300 msec

Segmented IR Turbo-FLASH

Seg IR-TFL (Null Post)
Reperfused vs Non-reperfused Infarction

Kim et al. Circulation 1999
Delayed Enhancement MRI

Area of delayed enhancement

= nonviable myocardium

(Bright is dead !!!)

Kim RJ et al, Circulation 1999
Fieno et al, JACC 2000
Delayed Enhancement MRI

**CLINICAL IMPLICATIONS**

- Detection of Myocardial Infarction
  - Acute Infarction
  - Chronic (healed) Infarction

- Prediction of Functional Recovery after Revascularization Treatment
DE-MRI vs SPECT

Transmural infarction
DE-MRI vs SPECT

Subendocardial infarction
Detection of MI: DE-MRI vs SPECT

A study in 12 dogs

Wagner et al. Lancet 2003
Detection of MI: DE-MRI vs SPECT

<table>
<thead>
<tr>
<th></th>
<th>*Animal Research</th>
<th>+Human Study</th>
</tr>
</thead>
<tbody>
<tr>
<td>CE-MRI</td>
<td>100/109 (92%)</td>
<td>181/181 (100%)</td>
</tr>
<tr>
<td>SPECT</td>
<td>31/109 (28%)</td>
<td>96/181 (53%)</td>
</tr>
</tbody>
</table>

*Gold standard : TTC, +Gold standard : CE-MRI

Wagner et al. Lancet 2003
DE-MRI vs PET

- In severe ischemic heart failure, hyperenhancement on DE-MRI as a marker of myocardial scar closely agrees with PET data.

Klein et al Circulation 2002
**Detection of MI: DE-MRI vs PET**

Assessment of Myocardial Viability With Contrast-Enhanced Magnetic Resonance Imaging Comparison With Positron Emission Tomography

A study in 31 patients with ischemic heart failure

<table>
<thead>
<tr>
<th></th>
<th>PET(t)</th>
<th>PET(nt)</th>
<th>PET(v)</th>
</tr>
</thead>
<tbody>
<tr>
<td>MRI(t)</td>
<td>126</td>
<td>11</td>
<td>38</td>
</tr>
<tr>
<td>MRI(nt)</td>
<td>7</td>
<td>27</td>
<td>51</td>
</tr>
<tr>
<td>MRI(v)</td>
<td>13</td>
<td>21</td>
<td>695</td>
</tr>
</tbody>
</table>

:t: transmural, nt: non-transmural, v: viable

**Conclusions**—In severe ischemic heart failure, MRI hyperenhancement as a marker of myocardial scar closely agrees with PET data. Although hyperenhancement correlated with areas of decreased flow and metabolism, it seems to identify scar tissue more frequently than PET, reflecting the higher spatial resolution.

*Klein et al Circulation 2002*
Lee SD 76/F

- C.C : Aggravated dyspnea for 1 week
- PMH: Diabetes mellitus for 10 years
- Cardiac Echo: Hypokinesia on anterolateral wall, apical to basal
- Cardiac enzyme:
  - CK(EM)(50-250 U/L) : 265
  - CK-MB(0.0-5.0 ng/ml) : 3682
  - Troponin -I (0-1.5) : 20.2
- EKG : Lateral AMI
Lee SD 76/F
Detection of Multiple Foci of MI: In Addition to the Expected Lesion
47/F chest pain

- Induced ischemia: mid-anterolateral wall
- Normal motion / no enhancement: viable!!
DE-MRI: CLINICAL IMPLICATIONS

• Detection of Myocardial Infarction
  - Acute Infarction
  - Chronic (healed) Infarction

• Prediction of Functional Recovery after Revascularization Treatment
DE-MRI Detection of Healed MI

Large Infarct

Hx: 10 months
CK: 3300 IU/L
MB: 294 µg/L

5 months
CK: 5912 IU/L
MB: 792 µg/L

4 months
CK: 3352 IU/L
MB: 389 µg/L

Wu et al. Lancet 2001
DE-MRI Detection of Healed MI

Small Infarct

Hx: 13 months  2 months  3 months
CK: 513 IU/L  219 IU/L  508 IU/L
MB: 62 µg/L  12 µg/L  35 µg/L

Wu et al. Lancet 2001
Limitations of DE-MRI:  
*Differentiation between Acute and Chronic MI?*

*Lim TH et al. JMRI 1997*
DE-MRI vs T2 MRI

T2 WI

DE-MRI

Acute MI (5 days ago)
DE-MRI vs T2 MRI

T2 WI

DE-MRI

Chronic MI (9 years Hx)
DE-MRI: CLINICAL IMPLICATIONS

• Detection of Myocardial Infarction
  - Acute Infarction
  - Chronic (healed) Infarction

• Prediction of Functional Recovery after Revascularization Treatment
DE-MRI: Functional Recovery

3 VD, F/U 3 months after CABG
EF: 47.4% → 63.4%
DE-MRI: Functional Recovery

3 VD, F/U 3 months after CABG
EF: 17.8% ➔ 19.4%
Assessment of Myocardial Salvage
An animal study in 15 dogs

Hillenbrand et al. Circulation 2000
Assessment of Myocardial Salvage

An animal study in 15 dogs

Mean Transmural Extent of Hyperenhancement on Day 3 in Each Group

% Segment with Improved Wall Motion

% Hyperenhancement on Day 3

Hillenbrand et al. Circulation 2000
Assessment of Myocardial Salvage

A prospective study in 61 patients

MRI for Myocardial Viability

1. Detection of Myocyte necrosis or scar: DE-MRI

2. Assessment of LV function:
   - Myocardial mass: End-diastolic wall thickness
   - LV contractility: End-systolic wall thickening
   - Contractile (inotropic) reserve
2. LV Function: *Cine MRI*
Analysis of LV Function

- Visual qualitative assessment
- Quantitative assessment

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Value</th>
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<tbody>
<tr>
<td>Ejection fraction</td>
<td>15.0 %</td>
</tr>
<tr>
<td>Stroke volume</td>
<td>55.2 ml</td>
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<tr>
<td>Cardiac output</td>
<td>2.9 l/min</td>
</tr>
<tr>
<td>ED phase</td>
<td>0.0 ms (phase 1)</td>
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<tr>
<td>ED volume</td>
<td>368.1 ml</td>
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<tr>
<td>ES phase</td>
<td>461.0 ms (phase 9)</td>
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<tr>
<td>ES volume</td>
<td>312.8 ml</td>
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<tr>
<td>ED wall mass</td>
<td>35.9 g</td>
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<tr>
<td>ED wall + papillary mass</td>
<td>n/a</td>
</tr>
<tr>
<td>ED wall - correct. mass</td>
<td>n/a</td>
</tr>
<tr>
<td>ED wall + papillary - correct. mass</td>
<td>n/a</td>
</tr>
<tr>
<td>Heart rate</td>
<td>52.0 bpm</td>
</tr>
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</table>

Thickening (ED)  Thickening  Motion
Quantitative Assessment

• More accurate than visual assessment

<table>
<thead>
<tr>
<th>Wall thickening results</th>
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<tbody>
<tr>
<td>slice</td>
</tr>
<tr>
<td>-------</td>
</tr>
<tr>
<td>4</td>
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<tr>
<td>4</td>
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47/M
Acute myocardial infarction
Echo: normal wall motion
Real Time Cine MR

- Retrospective TFE Synchronized Real Time Multi Slice (M2D) B-TFE
- Free breathing, real time, no breathhold
  - Pediatric patients, sick patients

Breathhold retrospective bTFE  Free breathing real time cine
2. LV Function: Low Dose DSMR

“contractile (inotropic) reserve”

- DE-MRI: impaired specificity as a predictor of functional recovery in non-transmural scars (1% to 74%).
- Low dose DSMR: superior to DE-MRI as a predictor of functional recovery and does not depend on the transmurality of scar.
- DE-MRI and DSMR provide complementary information.

Wellnhoffer et al. Circulation 2004
DSE vs DSMR

Rest 10µg 20µg

DSE

DSMR
Low Dose DSMR

Rest MRI

Low dose DSMR
Low Dose DSMR

Pre CABG

Post CABG
Low Dose DSMR

Viability

10µg

Rest

20µg
2. LV Function: Tagged MRI

Wall Motion Abnormality due to ICMP
Tagged MRI

Radial thickening

Circumferential shortening
Conclusion

1. Delayed enhancement MRI
   - Detection of area with myocyte necrosis
   - Acute MI vs. chronic MI (with T2 MRI)
   - Prediction of functional recovery

2. Cine MRI / Tagged cine MRI / DSMR
   - Myocardial mass
   - Contractile function
   - inotropic reserve
Thank You!!

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University of Ulsan College of Medicine
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