Disclosures

- Off-label: gadolinium MR of the heart and vessels, adenosine MRI
- Research support: Epix Medical
- Consultant: Bracco, Berlex
- Speaker: Toshiba, GE Healthcare

Essentials of Cardiac MRI

- MRI cardiac pulse sequences
- Evaluation of myocardial masses
- Evaluation of coronary heart disease
- Evaluation of the right ventricle

<table>
<thead>
<tr>
<th>Purpose</th>
<th>Type</th>
<th>Sequence</th>
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<tr>
<td>morphology</td>
<td>Black blood</td>
<td>double IR</td>
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<tr>
<td></td>
<td>T1, T2</td>
<td>FSE/TSE</td>
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<tr>
<td>function</td>
<td>cine</td>
<td>steady state free precession (SSFP)</td>
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<td>tissue</td>
<td>gadolinium</td>
<td>IR prepared gradient echo</td>
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<tr>
<td>characterization</td>
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"Double IR" black blood FSE

Breath-hold high resolution, intracardiac detail

- "T1" weighted, where TR = 1 R-R interval
- PD (TR 1000, TE 20), T2 weighted (TR 2000, TE 80)

"Double IR" FSE: 1st inversion pulse

nonselective 180° inversion pulse
“Double IR” FSE: 2nd inversion pulse

2nd selective 180° inversion pulse

“Double IR” FSE: wait for the TI time

• The inversion time for blood varies based on heart rate, from 400-600 msec
• If gadolinium present, use TI 200 msec

“Double IR”:

TI = 600 msec, inflowing blood now nulled (dark)
Perform a gated FSE acquisition

Cine: Steady state free precession (SSFP)

- balanced FFE®
- TruFISP®
- Fiesta®

ALL THE SAME

SSFP Cine

- fast gradient echo pulse sequence, balanced gradients in x,y,z
- Contrast $\sqrt{\frac{T2^*}{TI}}$

CINE images

Retrospective/Retriggering: continuous imaging through cardiac cycle

T. Foo
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Metastatic disease 20x more common than primary cardiac tumors

- Leiomyosarcoma metastasis

Primary benign tumors:

1. Myxoma 41%
2. Lipoma 14%
3. Papillary fibroelastoma 13%
4. Rhabdomyoma 11%

Myxoma

- Interatrial septal attachment
- 4:1 left vs. right sided

- High T2 signal
**Lipomatous atrial septal hypertrophy**
- echogenic mass on echocardiogram
- low association with arrhythmia, obesity

**Right atrial lipoma**
- T1 fat sat
- High T1 signal mass, well defined
- DDx: clot, proteinaceous fluid, melanoma

**Crista terminalis**
- T1 fat sat is diagnostic
- Associations: obesity, steroid use

**Crista terminalis**
- Smooth ridge within the right atrium
- Related to embryonic development of the right atrium

**Primary malignant tumors:**
1. Angiosarcoma 31%
2. Rhabdomyosarcoma 20%
3. Other sarcoma 16%
4. Mesothelioma 15%
5. Primary Lymphoma 6%

**62 yo female, CHF and abnormal CT**
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Non viable Myocardium (scar)

1. Lack of contraction, and
2. Lack of recovery of normal function after revascularization (CABG).

Chronic MI

- Images every 1 minute after gadolinium injection
Use an inversion pulse to suppress normal myocardium

- Optimal TI time depends on clearance of gadolinium from the normal myocardium
- Typical range: 175-250 msec
- Lower TI time when more gad is present:
  - decreased renal function
  - CHF

"TI Scout"

Single breath-hold, 50 phases, 20 msec temporal resolution

"TI Scout"

Images every 20 msec

Phase Sensitive Inversion Recovery

Magnitude Reconstruction

Phase Sensitive Reconstruction

Key factor: transmurality of the gadolinium enhancement

Improved function

% Transmurality

MRI is higher resolution than SPECT

TTC  MRI  SPECT

Canine model

Wagner A. et al., Lancet. 2003 Feb 1;361(9355):374-9

Kim et al. NEJM 2001: 345

MRI is higher resolution than SPECT

Canine model

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**Coronary Heart Disease**

1. No delayed enhancement = no myocardial scar/ fibrosis.
2. *Transmural* delayed enhancement = no functional recovery even after treatment by bypass or stent.

**Coronary Heart Disease: Acute coronary blockage (infarct)**

- Initially, there is myocardial necrosis (cell death) than enhances with gadolinium.
- If the blockage is not rapidly opened and the patient survives, the capillary blood supply may also be damaged.
**Q-Wave Acute MI**

- Images every 1 minute after gadolinium injection
- Capillary blockage is termed “microvascular obstruction”

**Acute infarct with microvascular obstruction (at the infarct core)**

1st pass image  
Filling in  
Infarct

25 sec  
40 sec  
10 min

**Acute LAD infarction**

1st pass  
delay

**Microvascular Obstruction (MO)**

MO predicts significantly increased rate of cardiovascular complications after MI (unstable angina, reinfarction, CHF, embolic stroke, death).

**Examples:**

**Coronary Heart Disease**
New onset CHF
pseudoaneurysm (rupture) vs. true aneurysm

True LV aneurysm + clot due to old infarct

LV aneurysm/infarct and clot
9% EF
LV volume 820 ml

16% EF

Hibernating Myocardium

Delayed Enhancement is nonspecific
- Fibrosis (MI, hypertrophy)
- Tumor
- Inflammation – myocarditis
- Amyloid
- Chagas disease (fibrosis)
- Sarcoid

Hypertrophic Cardiomyopathy (HOCM)
- Most common cause of sudden cardiac death <30 yrs old
- Obstruction of outflow tract
- Genetic abnormality: sarcometric contractile proteins
- Autosomal dominant
HOCM - pathophysiology

Systolic anterior motion of the mitral valve, resulting in aortic outflow obstruction

HOCM: MRI shows collagen deposition associated with “myocardial disarray”

Cine

Delayed contrast

HOCM: minimally invasive Rx (EtOH)

Pre Treatment

2 mths Post Treatment

Acute Onset Ventricular Tachycardia, Fever, Malaise

Patchy epicardial enhancement, noncoronary distribution

Cardiac Sarcoidosis

Patchy myocardial enhancement, especially septal/ basal/ epicardial regions

Acute Sarcoidosis: Hunold, J. Barkhausen, AJR 2005; 184

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Progressive RV failure
Giant Cell Myocarditis

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Arrhythmogenic right ventricular dysplasia/ cardiomyopathy (ARVD/C)
• MRI is the most important noninvasive imaging test for diagnosis.
• 71% of cases referred for 2nd opinion were overdiagnosed by MRI*
  (?high sensitivity, low specificity)

* Bomma et al. J Cardiovasc Electrophysiol 2004; 15

Arrhythmogenic RV Dysplasia
• Fibrofatty infiltration of RV resulting in ventricular tachycardia
• Palpitations, syncope, sudden death
• Age 33 ± 14 yrs.
• 30-50% cases are familial. MRI used to screen family members.

“McKenna” Criteria:
2 major, 1 major+2 minor, 4 minor*

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<tr>
<th>Criteria</th>
<th>Major</th>
<th>Minor</th>
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<tr>
<td>Abnl structure/ function by echo</td>
<td>Severe dilatation and</td>
<td>Localized RV aneurysms</td>
</tr>
<tr>
<td>ventriculography</td>
<td>reduction of RV EF</td>
<td></td>
</tr>
<tr>
<td>MRI or nuclear</td>
<td>Severe segmental</td>
<td></td>
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<tr>
<td></td>
<td>RV wall remodeling</td>
<td></td>
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<tr>
<td>ECG</td>
<td>QRS prolongation</td>
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<tr>
<td>Repolarization of depolarization</td>
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<tr>
<td>or ischemia</td>
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<tr>
<td></td>
<td>Confirmed at necropsy or</td>
<td></td>
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<tr>
<td>Family history</td>
<td>surgery</td>
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</tbody>
</table>

Br Heart J 1994;71

ARVD MRI Diagnostic Findings
1. Abnormal RV morphology
2. Abnormal RV function
3. Abnormal signal intensity (fat)
4. Enhancement in the RV wall (fibrosis)
Presence of RV chamber enlargement

ARVD: findings on axial images

RV enlargement: differential diagnosis in the setting of suspected ARVD

1. Normal variant (young age)
2. Pulmonary Hypertension
3. PAPVR
4. Intracardiac cardiac shunt or valve dysfunction

RV and Pulmonary outflow tract enlarged, poor function

Right ventricular aneurysm

Typical ARVD

37% of normal volunteers
**Tissue Characterization: RV fat**

**Right ventricle fat**

**Detection of RV fibrosis in ARVD**

- Delayed enhancement due to fibrosis present in 60% of ARVD patients.
- All patients had other RV abnormalities (wall motion, morphology).

**Fat alone is insufficient for ARVD/C diagnosis: not arrhythmogenic**

LV EF 68.5%
RV EF 71%
EDV 120 ml
EDV 124 ml

**RV delayed enhancement**

*ICD, investigational*

*Tandri, JACC 2005; 45*
RV delayed enhancement

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www.heartMRI.com

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