Magnetic Resonance Imaging
A Brief History

- Magnetic Resonance phenomenon first described by Felix Bloch and Edward Purcell in 1946. In 1952 they were awarded the Nobel Prize.

- 1971 - Raymond Damadian showed that the nuclear magnetic relaxation times of tissues and tumors differed, sparking interest in medical uses (Science 03/19/71).

- In 1975 Richard Ernst proposed magnetic resonance imaging using phase and frequency encoding, and the Fourier Transform, which forms the basis of current MRI techniques.

- 1987 – MR angiography developed by Charles Dumoulin.

- 1993 functional MRI (fMRI) was developed.
Physical Principles

- Based on the absorption and emission of radiofrequency energy – so there is NO ionizing radiation.

- Uses magnets ranging in strength from 0.3 to 1.5 Tesla to create a magnetic field around the patient.

- Magnetic field causes protons in the body to align and then pulsed radiowaves are directed at the patient causing a disturbance of the proton alignment.

- Atoms then realign and in doing so, emit the absorbed radiofrequency.
The time it takes the protons to regain their equilibrium state = RELAXATION TIME.

2 types of relaxation time: T1 – Longitudinal (parallel to the magnetic field) and T2 – transverse (perpendicular to the magnetic field).

Relaxation Time and Proton Density are the main determinants of signal strength.

The main determinants of contrast or the weighting are:
- 1) Repetition Time (TR) – the time between successive RF pulses
- 2) Echo Time (TE) – time between the arrival of the RF pulse that excites and the arrival of the return signal at the detector.
Short TR + Short TE = T1 weighted
T1 Characteristics

• Dark
  • CSF
  • Increased Water – edema, tumor, infarct, inflammation, infection, hemorrhage (hyperacute or chronic)
  • Low proton density, calcification
  • Flow Void

• Bright
  • Fat
  • Subacute hemorrhage
  • Melanin
  • Protein-rich Fluid
  • Slowly flowing blood
  • Gadolinium
  • Laminar necrosis of an infarct

• White matter brighter than Gray
Long TR + Long TE = T2 weighted

T2 Characteristics

• Dark
  • Low Proton Density, calcification, fibrous tissue
  • Paramagnetic substances - deoxyhemoglobin, methemoglobin (intracellular), iron, hemosiderin, melanin
  • Protein-rich fluid
  • Flow Void

• Bright
  • Increased Water – edema, tumor, infarct, inflammation, infection, subdural collection
  • Methemoglobin (extracellular) in subacute hemorrhage

• Gray matter brighter than white
Our patient K.G.:

43 yo male with h/o of Osler-Weber-Rendu, Hepatitis B Virus, and remote IV Drug Abuse presenting with headache, and episode of aphasia, and seizures.

Differential
- Traumatic bleed
- Stroke – Hemorrhagic vs. Ischemic
- TIA
- Neoplasm
- Infection
Menu of Tests for imaging suspected brain pathology

- Plain films of the skull
- Head CT
- Head MRI
- Radionuclide brain scan/PET
- Cerebral angiography
A bleed was high on the differential because of the AVMs associated with Osler-Weber-Rendu and a history that the patient had fallen during a seizure.

**Head CT**

- Imaging study of choice in acute situations. CT scanning is quicker than MR and acute blood is readily obvious without IV contrast. Further, bone windows will show skull fractures. Finally, intracranial calcifications are more apparent.

- Get the head CT first when considering a hemorrhagic event, possible skull fracture, or any cerebral event in an unstable patient.
A head CT was therefore first obtained on our patient, K.G.;

No Intracranial bleed or fracture evident. No definite abnormality identified. (Subtle low attenuation adjacent to left occipital horn)
In view of our patient’s significant CNS presentation, an MRI was obtained;

**Menu of Head MRI Scans**

1. Routine $T_1$, $T_2$ head MRI
2. Functional MRI
3. Diffusion Weighted Imaging (DWI)
4. Fluid Attenuated Inversion Recovery (FLAIR)
5. MR angiography (MRA)
Our patient’s MRI

There is an abnormal area in the left temporo occipital region.
Gadolinium enhanced MRI

Ddx Ring Enhancing Lesion

- Lymphoma
- Toxoplasmosis
- Cystercercosis
- Bartonella
- TB
- Listeria
- Whipple’s
- Histoplasmosis
- Blastomycosis

- Nocardia
- Actinomycosis
- Coccidiomycosis
- Bacterial Abcess
- Necrotic Met
- Glioblastoma
- Inflammation
- Demyelination (active)
- Radiation Necrosis
Our patient proved to have a bacterial brain abscess. In view of the past history of drug abuse, HIV was considered.
In a patient with HIV one must consider Lymphoma vs. Toxoplasmosis. Their classic presentation on MRI is:

**Toxo**
- T1 axial
- Multiple Lesions
- Basal ganglia
- Brainstem

**Lymphoma**
- T1 axial
- Solitary lesion
- Periventricular or in the corpus callosum
- Rapid progression
- +/- Mass effect

A Functional MRI aids their differentiation further:

**Advanced Imaging fMRI**

**Lymphoma**

**Toxoplasmosis**

**Advantages**

- Detect hyperacute stroke - stroke in evolution, and within first 30min. Standard CT hypodense btw 12-36hrs.

- Differentiate acute ischemia from chronic infarct

Diffusion Weighted Imaging (DWI)/Apparent Diffusion Coefficient (ADC)

- A high signed area on DWI is abnormal.
- Pitfall: The DWI is a manipulated T2 image and therefore high signal areas can be caused by “T2 shine through”. i.e. causing a false positive scan.
- An ADC scan is therefore mapped out with the DWI.
- Rule:
  - High signal DWI + low signal ADC = True abnormality.
  - High signal DWI + high signal ADC = False positive.
DWI continued

CT – 1.5hrs

DWI – 3.5hrs

DWI – 36.5

Fluid-Attenuated Inversion Recovery
FLAIR

- Basically T2 without CSF brightness
- TE>80 and TR>10,000
- Edema and Gliosis are hyperintense
MR Angiography

• Computer-assisted generation of images that result from the difference in signal between flowing blood next to stationary tissue.

• Type of Images:
  • Time-of-flight – utilizes inflow enhancement and highlights vessel morphology.
  • Phase-contrast - utilizes velocity-induced phase shifts and provides information on velocity and direction of blood flow.
  • Contrast Enhanced – utilizes T1 sequence with contrast to further delineate vasculature
### MRI Summary

<table>
<thead>
<tr>
<th>Normal tissue</th>
<th>T1</th>
<th>T2</th>
<th>CT</th>
</tr>
</thead>
<tbody>
<tr>
<td>dense bone</td>
<td>dark</td>
<td>dark</td>
<td>bright</td>
</tr>
<tr>
<td>Air</td>
<td>dark</td>
<td>dark</td>
<td>dark</td>
</tr>
<tr>
<td>Fat</td>
<td>bright</td>
<td>less bright</td>
<td>dark</td>
</tr>
<tr>
<td>Water</td>
<td>dark</td>
<td>bright</td>
<td>dark</td>
</tr>
<tr>
<td>Brain</td>
<td>anatomic</td>
<td>intermediate</td>
<td>intermediate</td>
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</tbody>
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### MRI Summary cont.

<table>
<thead>
<tr>
<th>tissue</th>
<th>MR-T1</th>
<th>MR-T2</th>
<th>CT</th>
<th>enhancement</th>
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</thead>
<tbody>
<tr>
<td>Infarct</td>
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<td>bright</td>
<td>dark</td>
<td>subacute</td>
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<tr>
<td>Bleed</td>
<td>bright2</td>
<td>bright2</td>
<td>bright</td>
<td>no</td>
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<tr>
<td>Tumor</td>
<td>dark</td>
<td>bright</td>
<td>dark3</td>
<td>yes</td>
</tr>
<tr>
<td>MS plaque</td>
<td>dark</td>
<td>bright</td>
<td>dark4</td>
<td>acute</td>
</tr>
</tbody>
</table>

1. Blood brain barrier leak. For MR, gadolinium; for CT, iodinated contrast material.
2. Unless very fresh or very old.
3. Unless calcified.
4. Often isodense.
References

http://www.med.harvard.edu/AANLIB/home.html

http://www.cis.rit.edu/htbooks/mri/bmri.htm

http://mednav.com/zone/Naids/zones/Articles/1999/08/Imaging/#Other


Antonio Culebras, MD, Chair; Carlos S. Kase, MD; Joseph C. Masdeu, MD; Allan J. Fox, MD; R. Nick Bryan, MD, PhD; C. Barrie Grossman, MD; Donald H. Lee, MD. Practice Guidelines for the Use of Imaging in Transient Ischemic Attacks and Acute Stroke.

All web pages referenced on 9/15-18/00.
Acknowledgements

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The end.