

## ISMRM Clinical MRI Course 2006

### From Physics to Protocols

#### Neuro I

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The various contrasts of MRI form an artist's palette of colors which can be drawn upon to compose the clinical protocol. In part I of this presentation, we will introduce the component contrasts, examine their interpretation and explore properties and limits of their implementation. The completed "palette" of contrast possibilities will then be passed on to the clinical neuroradiologist to create disease specific protocols drawing on the available contrast mechanisms (see Neuro II, D. Mikulis).

In particular, we will revisit T1, T2 and T2\* and focus on specific neuroradiological issues associated with each relaxation time constant. We will then proceed to examine diffusion-weighting and MR spectroscopy. Intravenous administration of Gd-based contrast media ("magnetic dye") will be introduced. The associated applied use of T1 and/or T2\* mechanisms will additionally allow us to develop approaches to "perfusion-sensitive" imaging, imaging of microvascular permeability and imaging sensitive to the oxygenation level of blood (Blood Oxygenation Level Dependent, BOLD, imaging). Some introductory applications of these approaches will be introduced to yield maps of white matter fiber paths (based on diffusion weighted imaging), functional brain mapping (based on BOLD) and microvascular permeability (based on kinetic analysis of dynamic contrast enhanced MRI). Finally recent advances in parallel imaging (with multiple receiver coils) will be introduced as the technology pertains to implementation issues of imaging with the above contrast mechanisms.

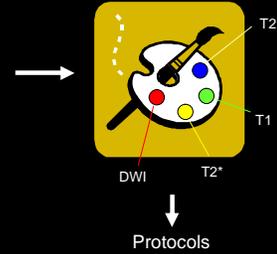
# Principles and Protocols Neuro MRI

Tim Roberts, PhD  
University of Pennsylvania

→ Neuro Protocols  
(D. Mikulis)

## Overview

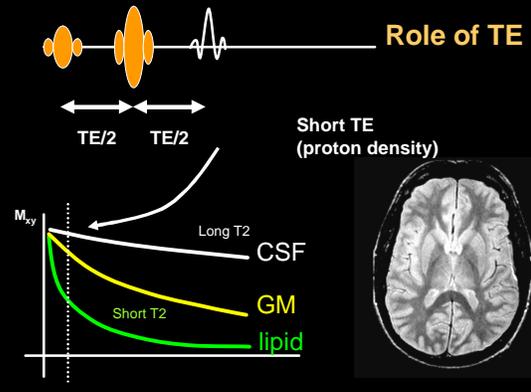
T<sub>2</sub> sequences  
T<sub>1</sub> sequences  
T<sub>2</sub>\* and Susceptibility  
Diffusion



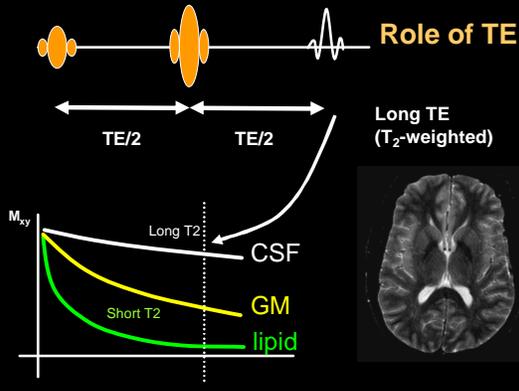
## T<sub>2</sub> Sequences

- SE - spin echo
- FSE - fast spin echo  
(FRFSE)
- ssFSE - single shot fast spin echo
- FLAIR (after T<sub>1</sub> discussion)
- Parallel Imaging – SENSE, iPAT, ASSET

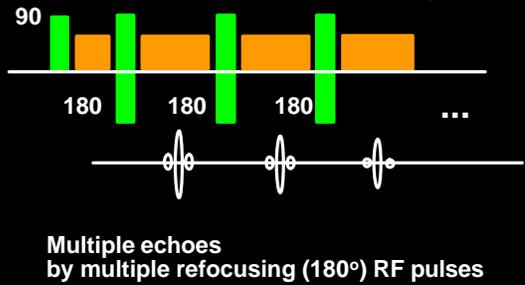
## Role of TE

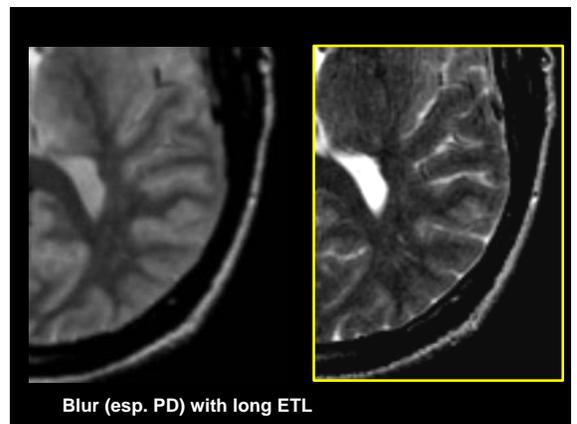
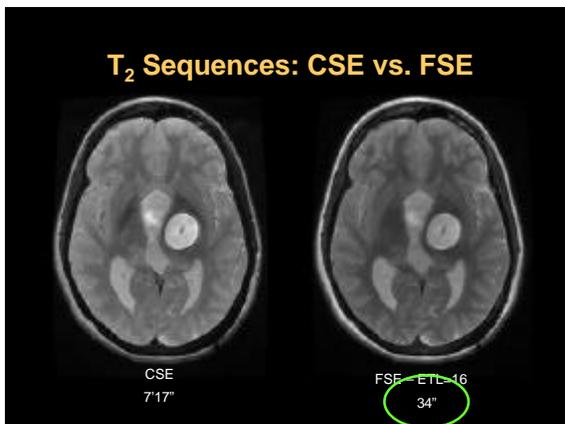
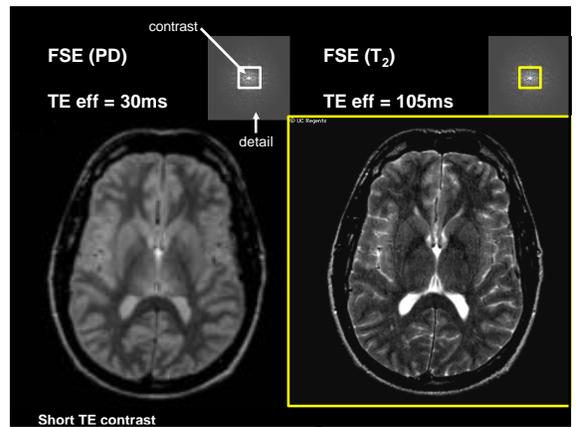
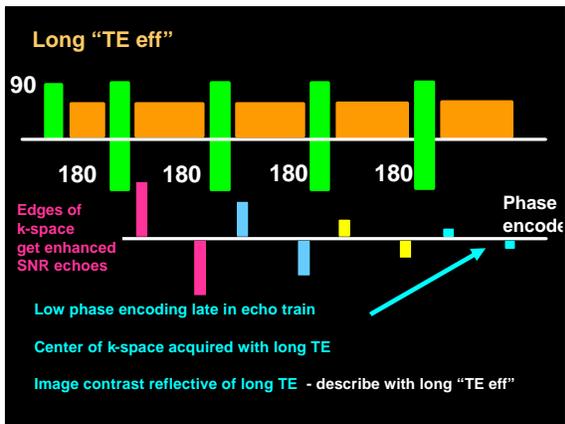
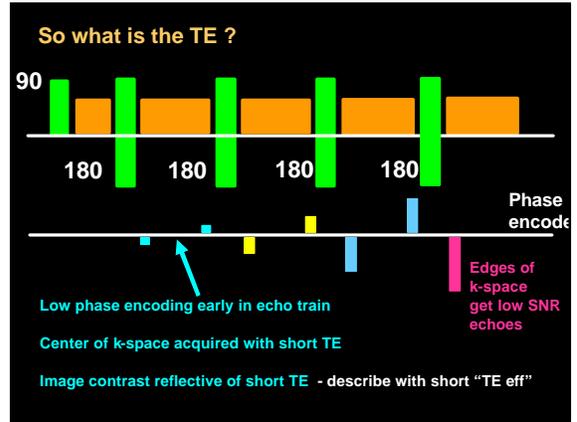
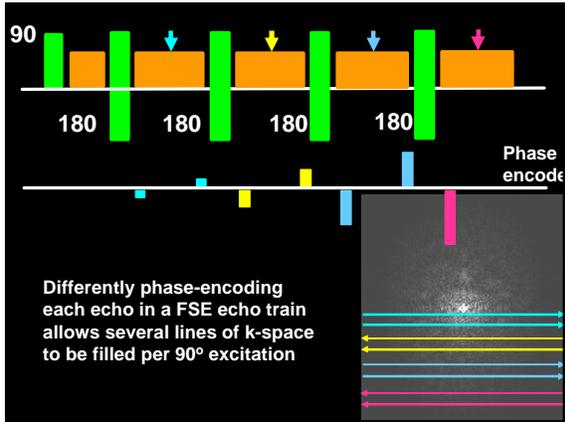


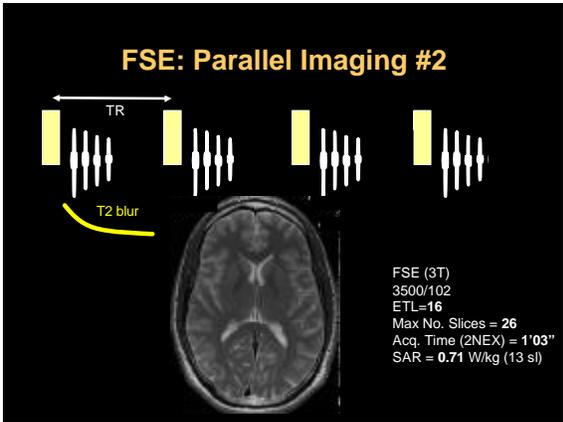
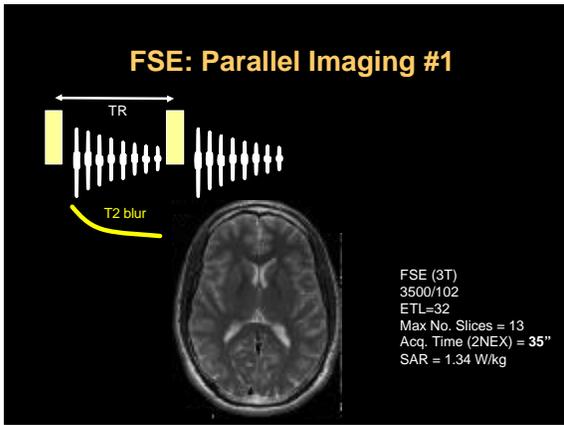
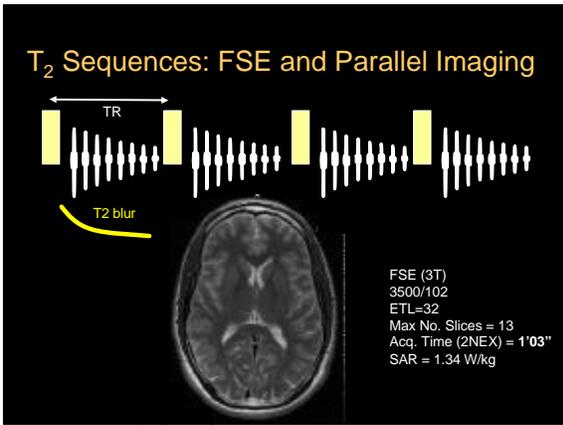
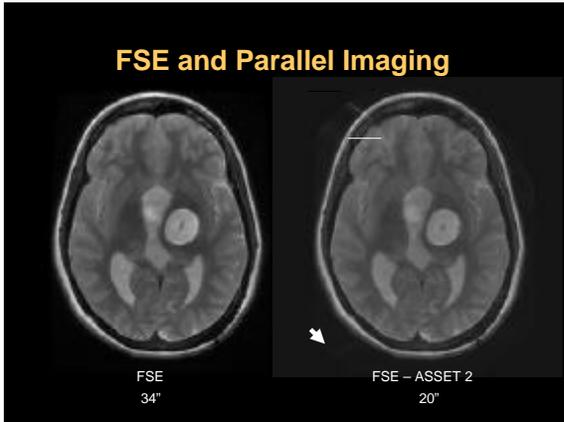
## Role of TE



## Fast Spin Echo (also TSE, RARE)





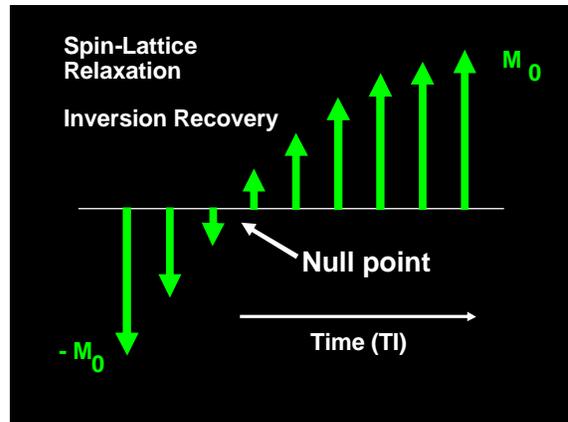
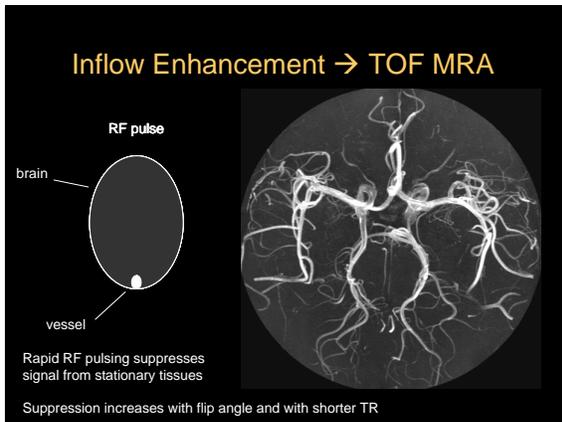
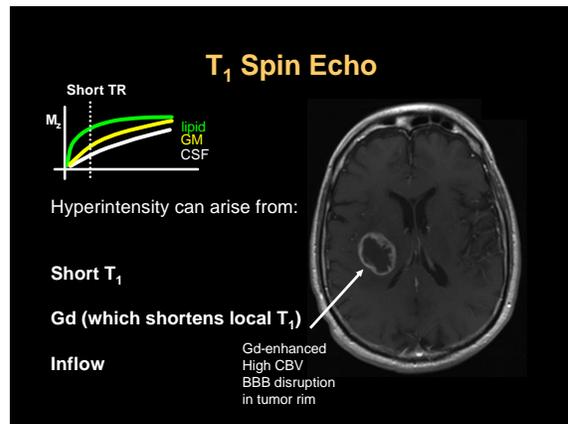
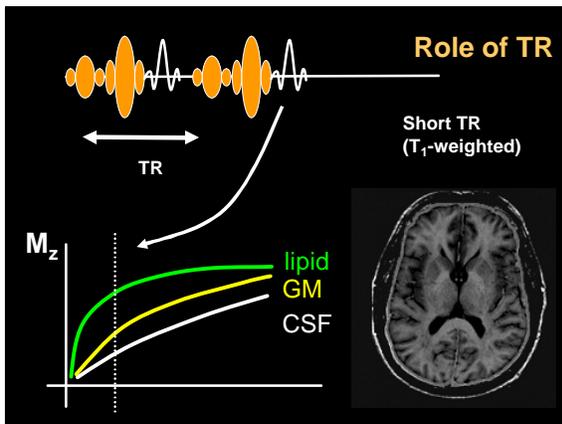
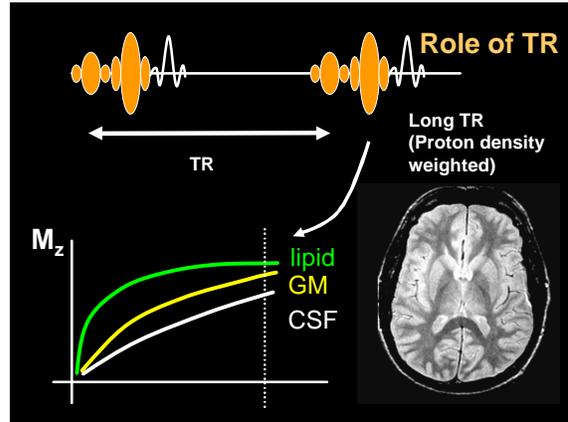


**T<sub>2</sub> Sequences**

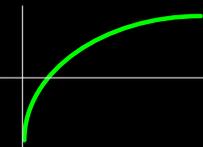
- SE - fluid hyperintensity / good resolution
- FSE - similar to SE, 8x or 16x faster
- (FR)FSE - fast recovery
- ssFSE - single shot
- FLAIR - CSF suppressed
- Parallel Imaging - speeds up acquisition, or shortens ETL  
- compromise SNR, some artifacts

## T<sub>1</sub> Sequences

- SE - workhorse
- (IR-prep - 3D)
- + Gd - BBB disruption
- TOF-MRA - MR angio
- Permeability - dynamic 3D T<sub>1</sub> + Gd



### Inversion Recovery



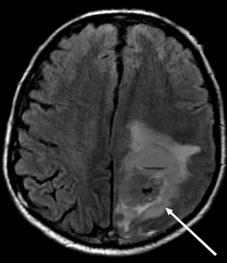
$M_z = 0$  when  $TI = 0.69 \cdot T_1$

Signal from this  $T_1$ -species goes to zero !

STIR – short TI (null fat)  
*don't use Gad*

FLAIR – long TI (null CSF)

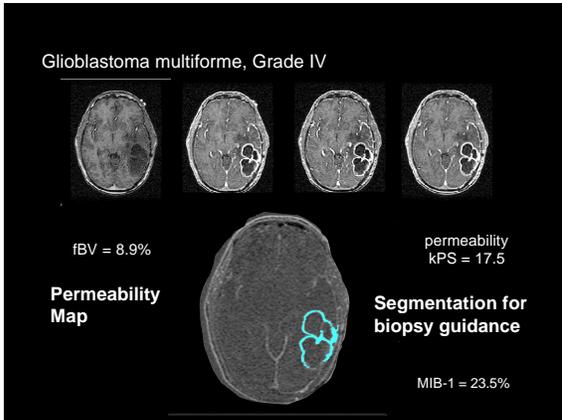
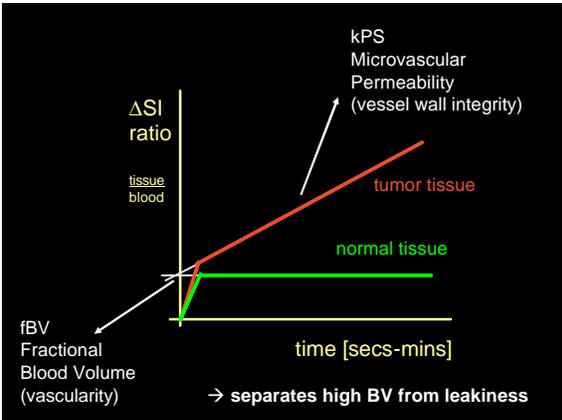
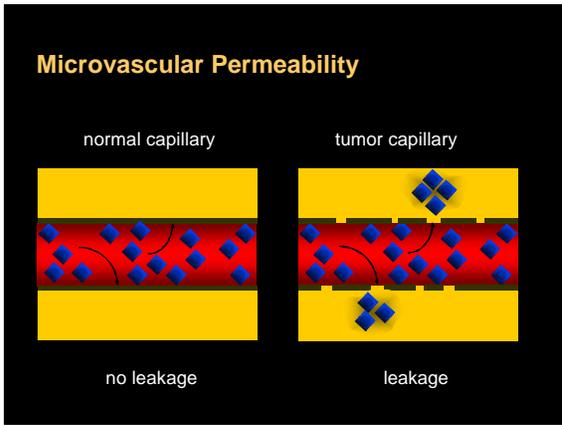
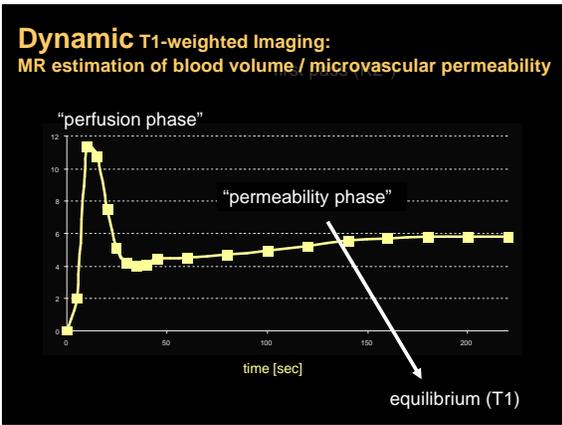
### FLAIR: $T_2$ Contrast without the CSF



Long TI allows most tissues to recover towards equilibrium.

Long  $T_1$  CSF just gets to "null point"

TI ~ 2secs @ 1.5T



## T<sub>1</sub> Sequences

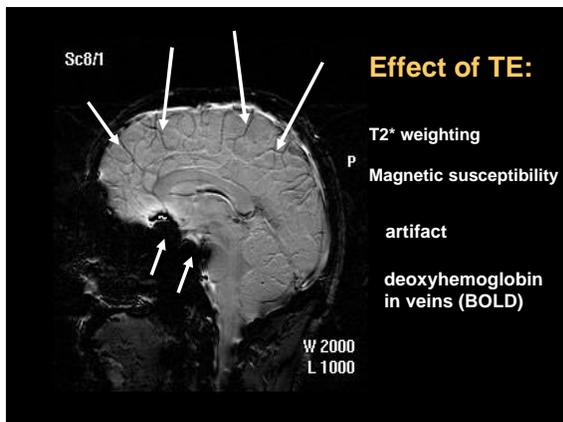
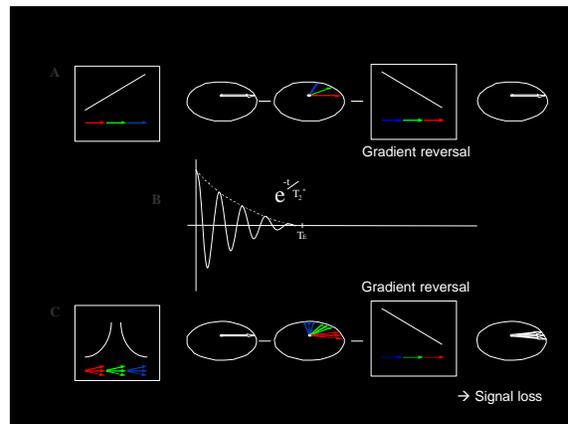
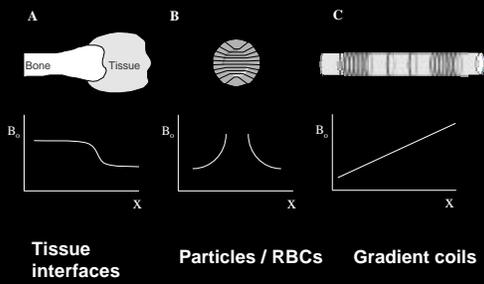
- SE - workhorse
- (IR-prep - 3D)
- + Gd - BBB disruption
- TOF-MRA - MR angio
- Permeability - dynamic 3D T1 + Gd



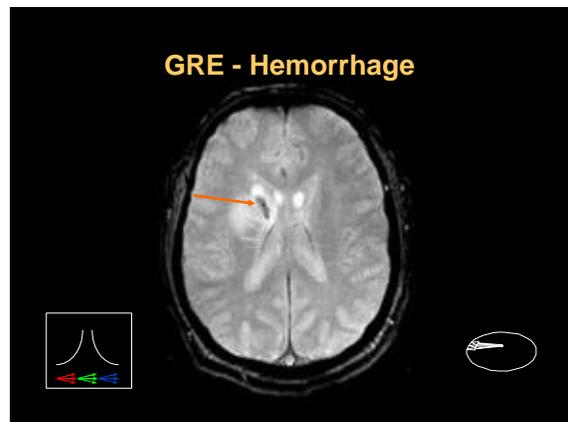
## T<sub>2</sub><sup>\*</sup> and Susceptibility

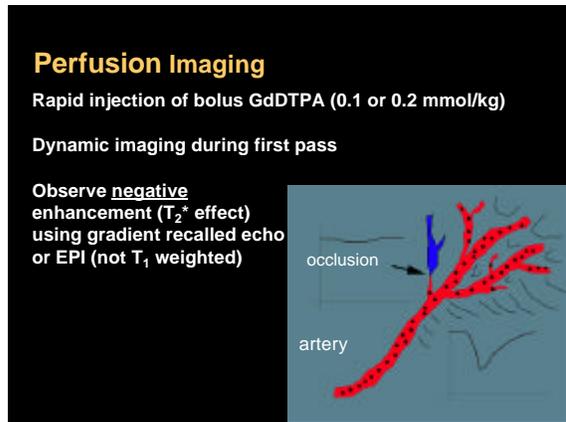
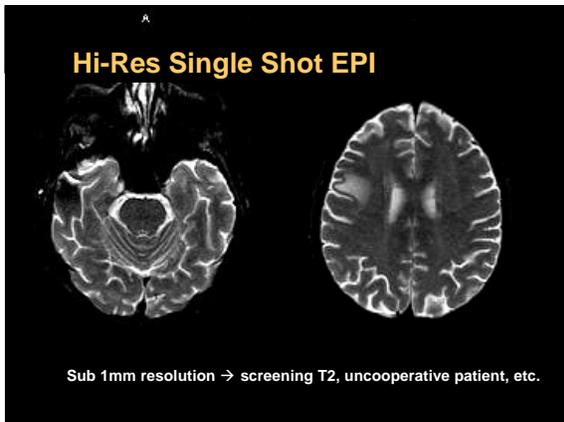
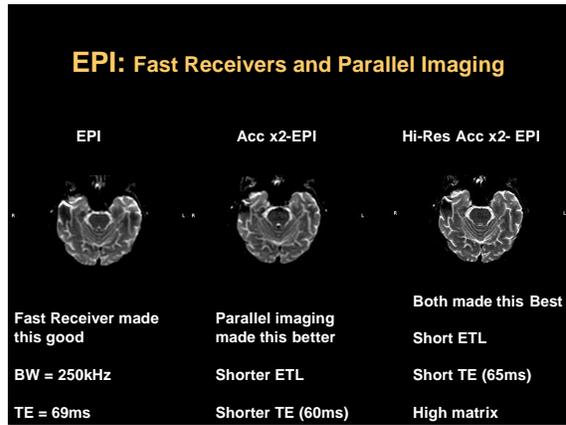
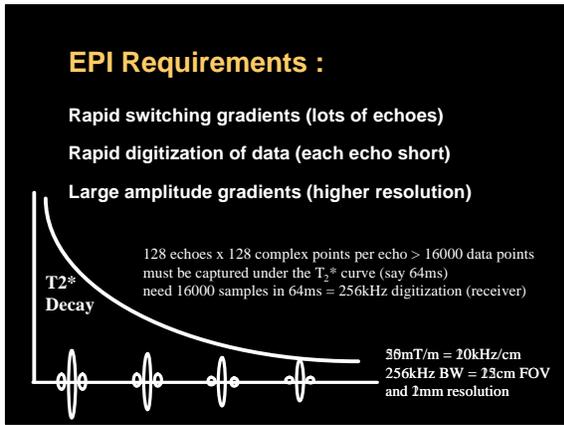
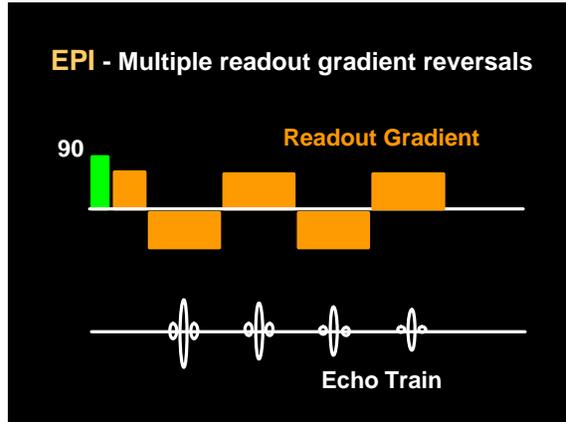
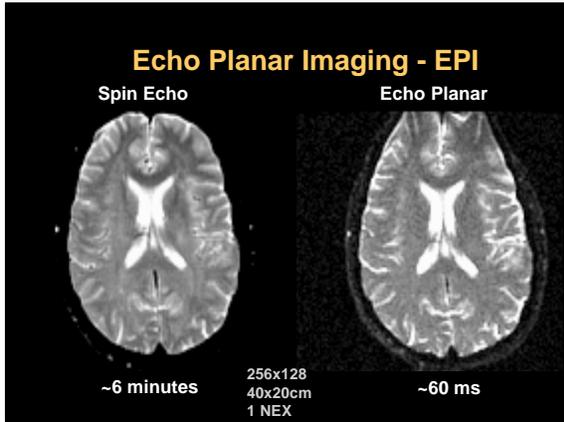
- GRE - hemorrhage
- EPI - artifact, speed
- Perfusion - sensitivity to Gd as a *negative* contrast
- BOLD - fMRI / hypoxia
- (FIESTA, bFFE, True-FISP)

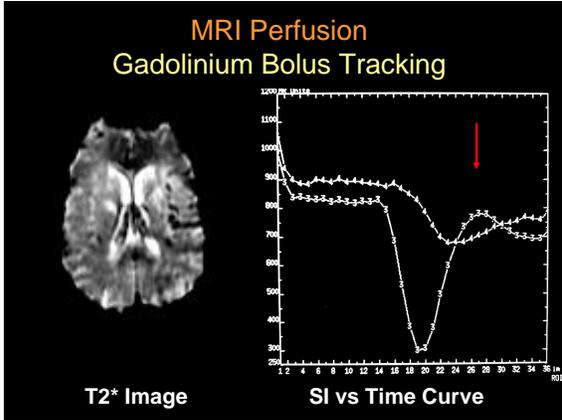
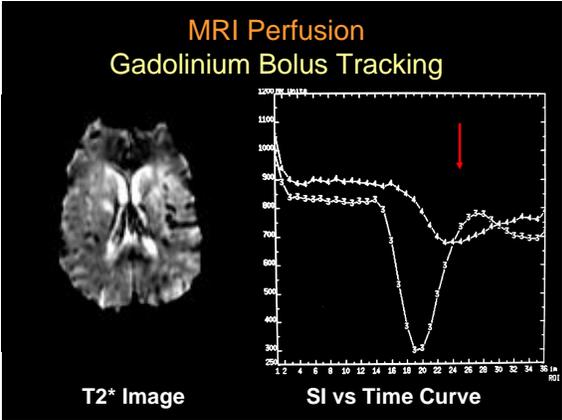
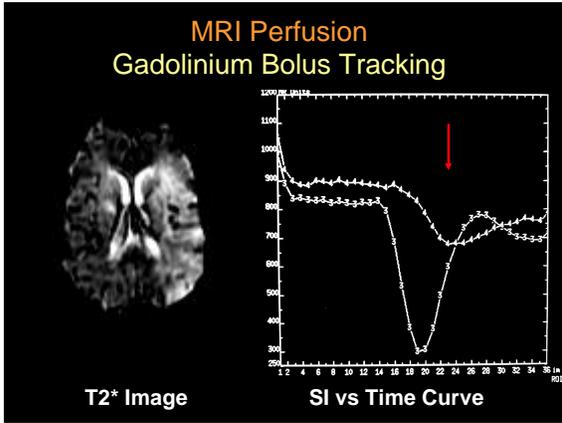
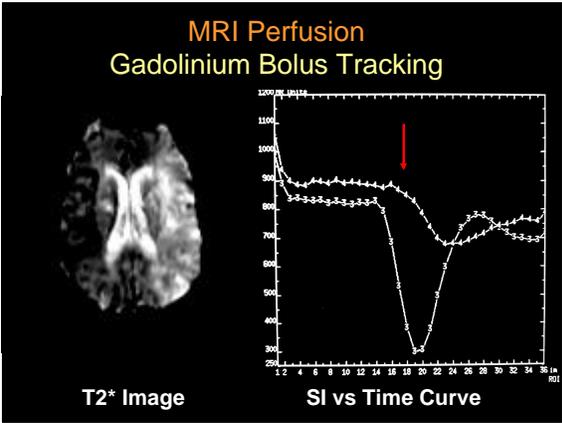
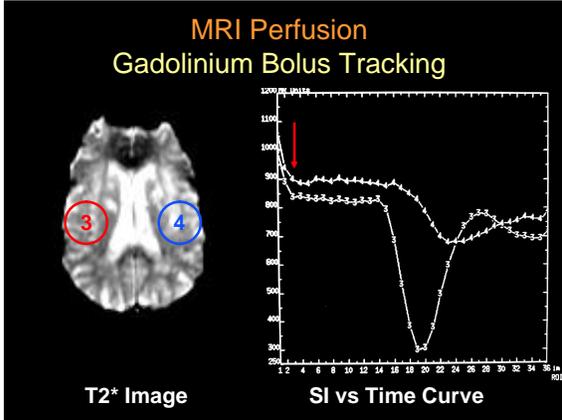
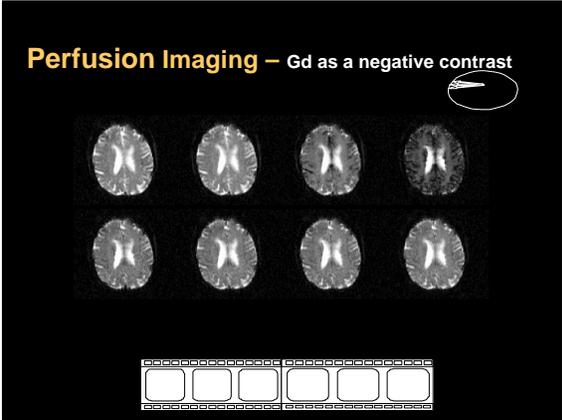
## Magnetic Field Homogeneity

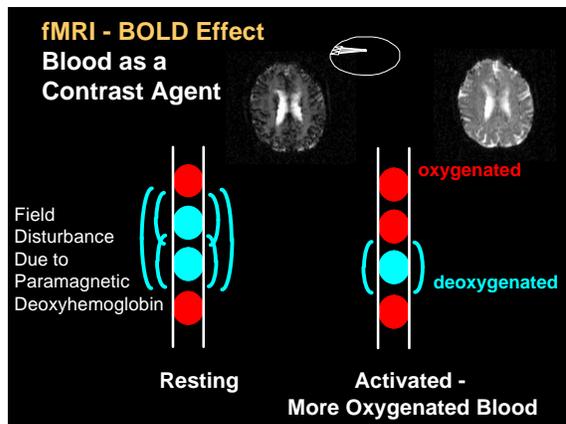
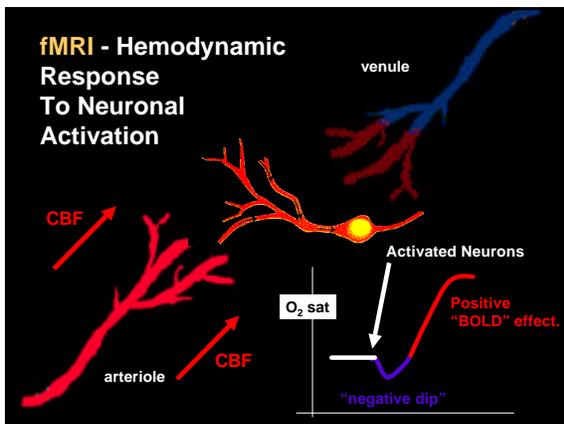
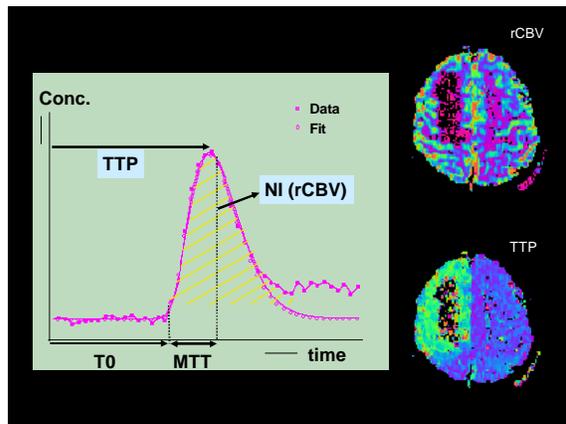
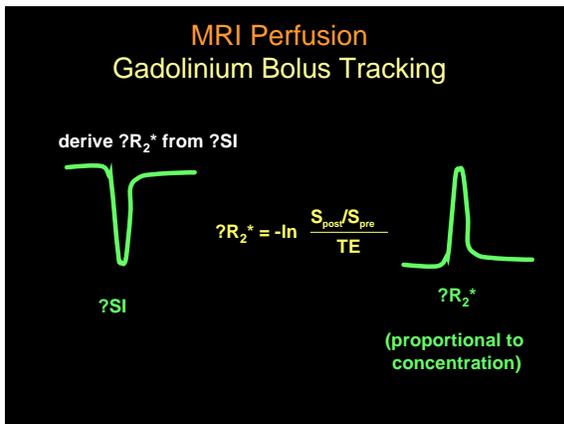
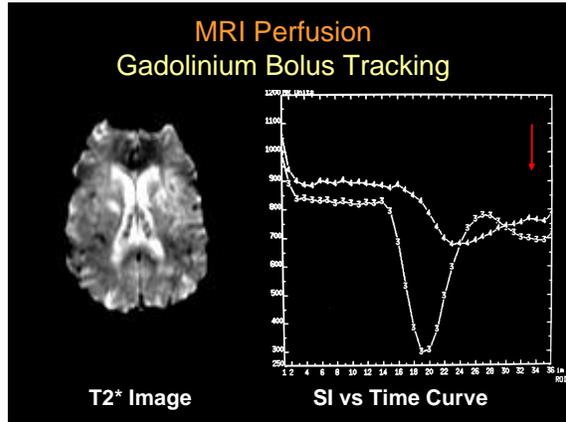
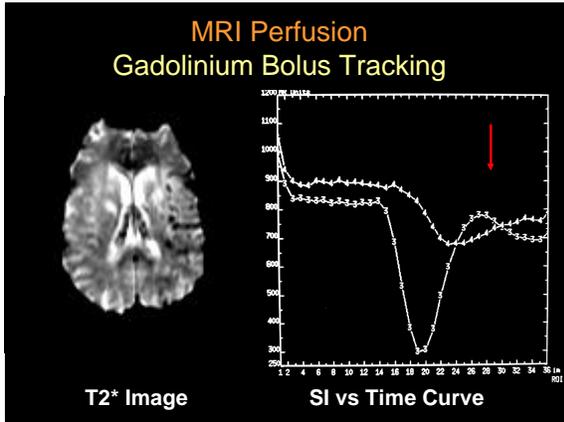


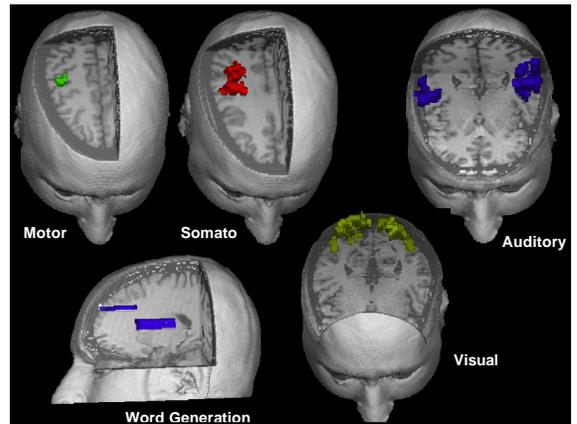
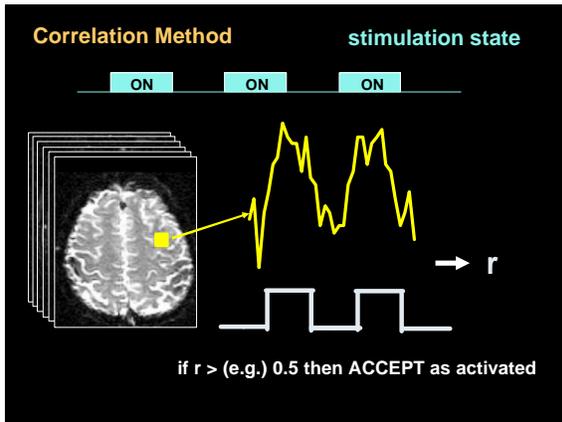
## GRE - Hemorrhage











### T<sub>2</sub>\* and Susceptibility

- GRE - hemorrhage
- EPI - artifact, speed
- Perfusion - sensitivity to Gd
- BOLD - fMRI / hypoxia

(FIESTA, bFFE, TrueFISP)

### Diffusion

- DWI - principles
- DWI - stroke
- DWI - T2 shine through
- DTI - anisotropy
- DTI - fiber tracking

### Relevance of Diffusion

free diffusion high ADC

hindered diffusion low ADC

membrane etc.

"apparent diffusion coefficient"

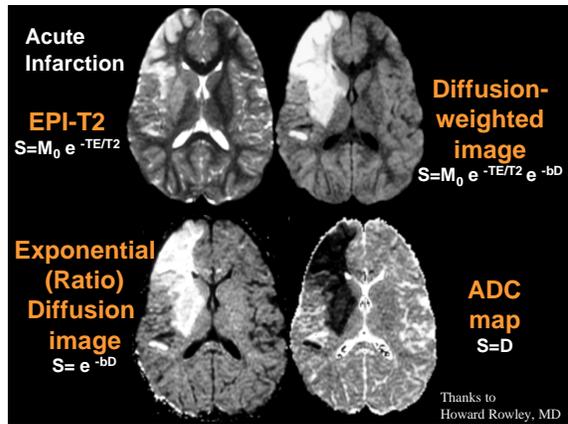
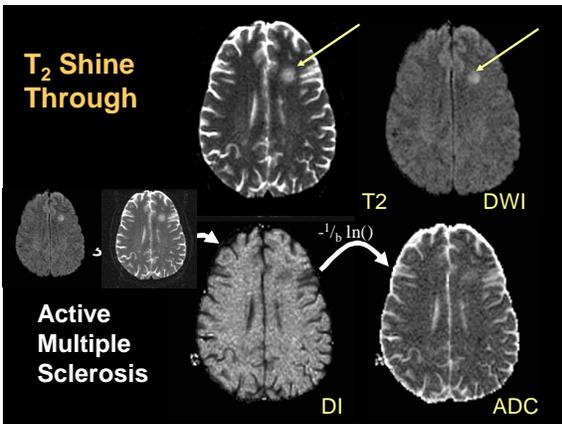
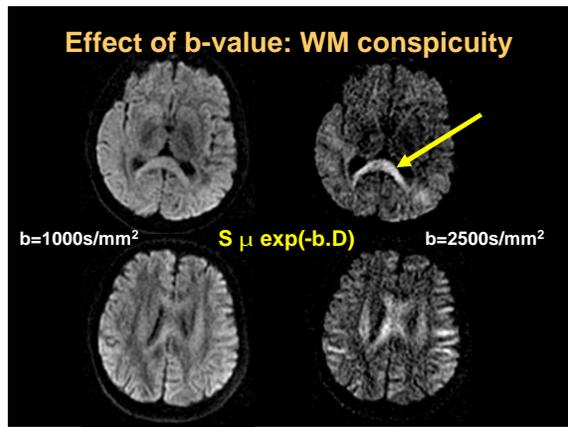
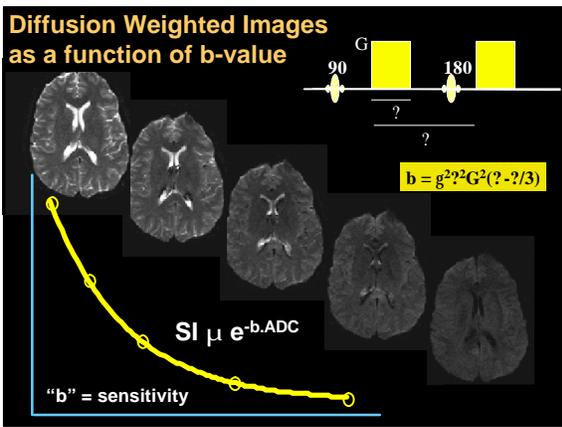
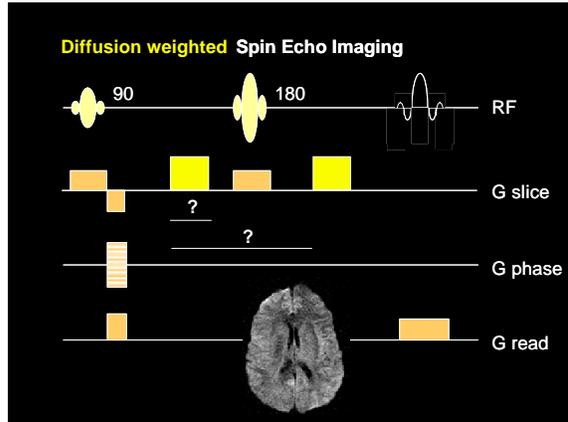
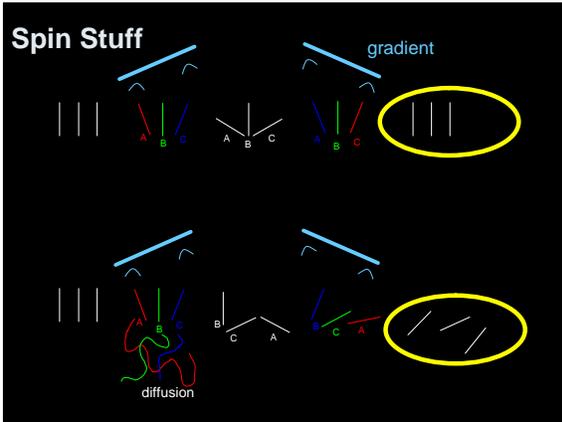
### Diffusion Weighted Imaging

T<sub>2</sub>W image with two equal and opposite dephasing gradient pulses separated in time

dephase incompletely rephase dephased signal

Moving Molecule

Stationary Molecule



**Old Infarction**

**EPI-T2**  
 $S = M_0 e^{-TE/T2}$

**Diffusion-weighted image**  
 $S = M_0 e^{-TE/T2} e^{-bD}$

**Exponential (Ratio) Diffusion image**  
 $S = e^{-bD}$

**ADC map**  
 $S = D$

Thanks to Howard Rowley, MD

**Diffusion – Embolic Lesion**

128x128 DWI-EPI

128x128 DWI-EPI with Acc x2

256x256 DWI-EPI with Acc x2

**Hi-res Single Shot Diffusion EPI**

256 x 256

→ Small lesion conspicuity

**Diffusion weighted Spin Echo Imaging read**

90 180 RF

G slice

G phase

G read

**Anisotropic Diffusion**

Can evaluate the components of the ADC in 3 directions (slice, phase, read: e.g. x, y, z)

If  $ADC_x = ADC_y = ADC_z$ , we call the tissue **isotropic**

free fluids are isotropic, also gray matter

If one direction is preferred, we call the tissue **anisotropic** (e.g. white matter)

Slow diffusion

Medium diffusion

Rapid diffusion along axons

Slow diffusion across axons

**Anisotropic Diffusion in Celery**

