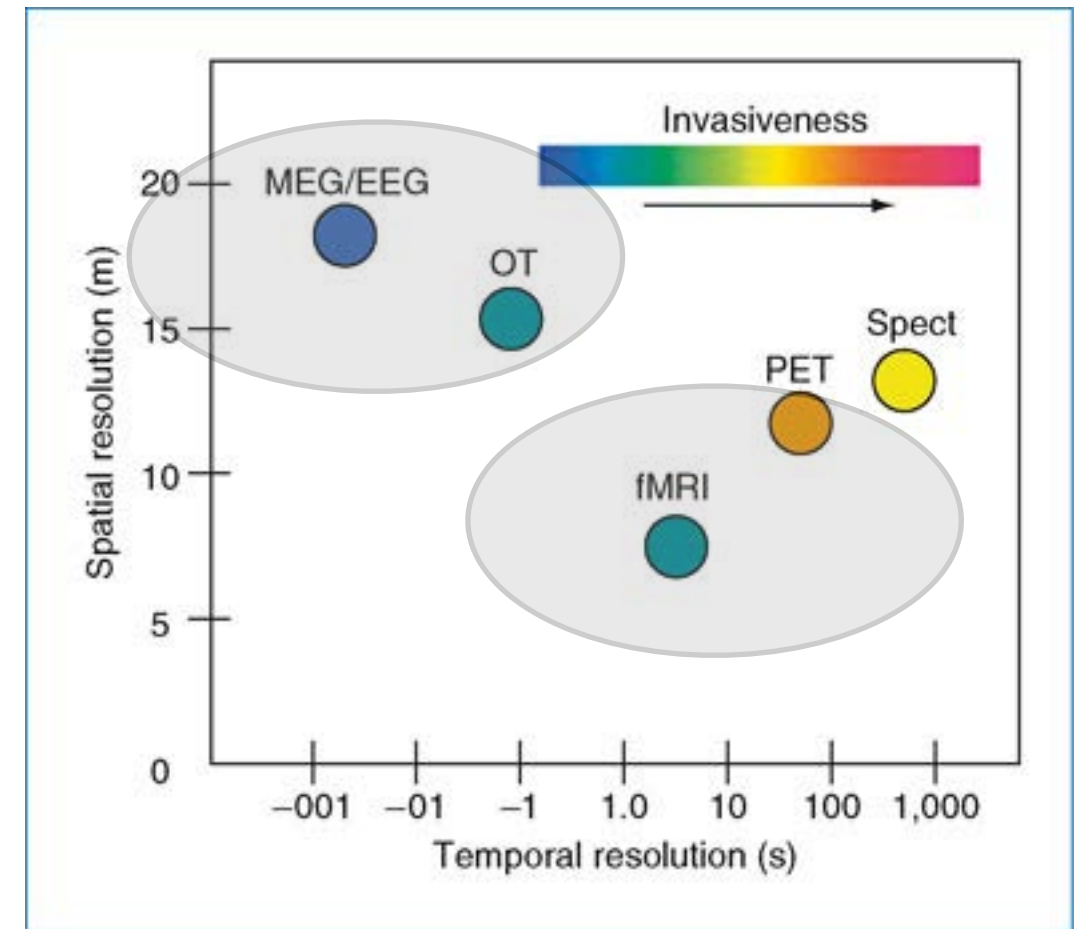
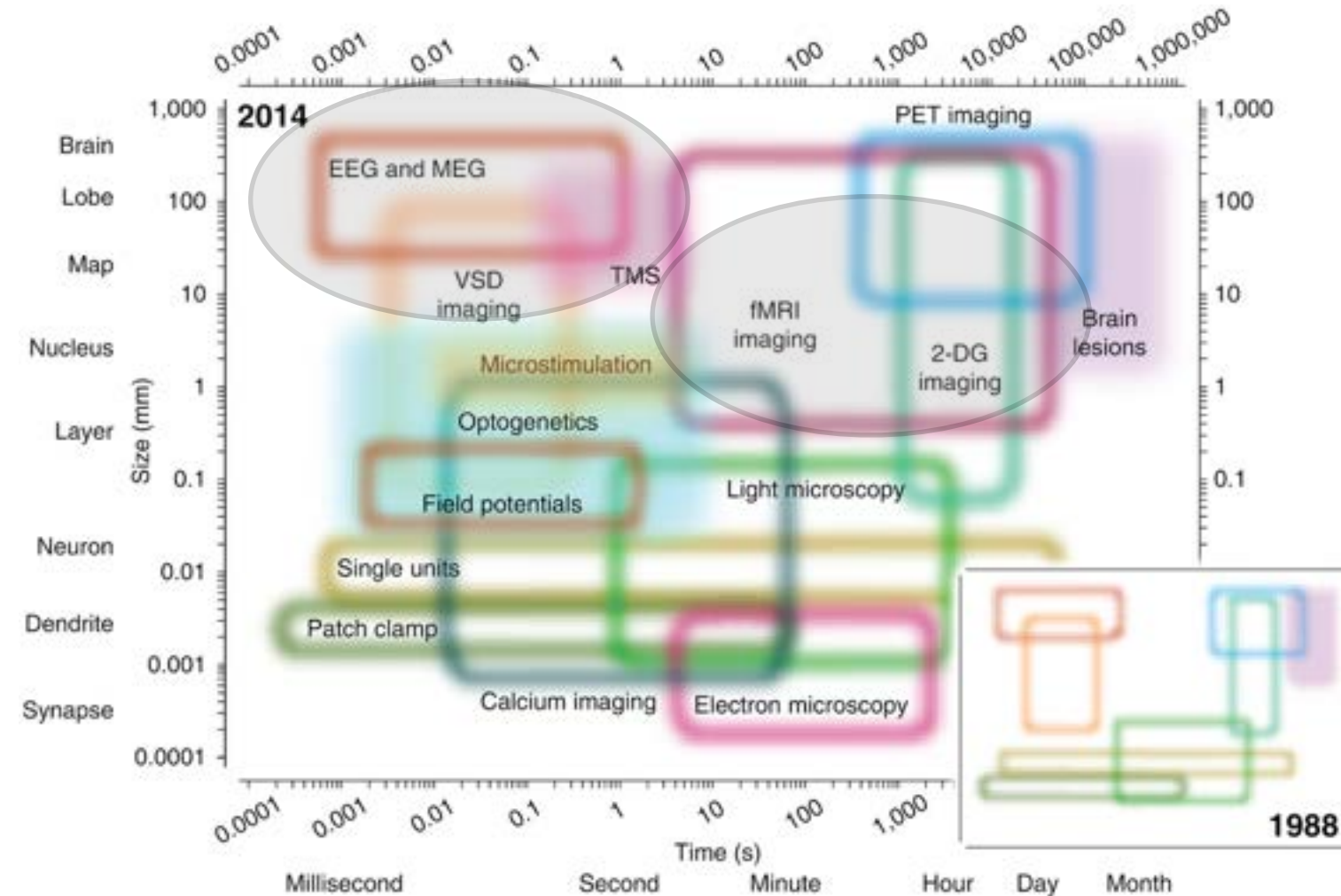


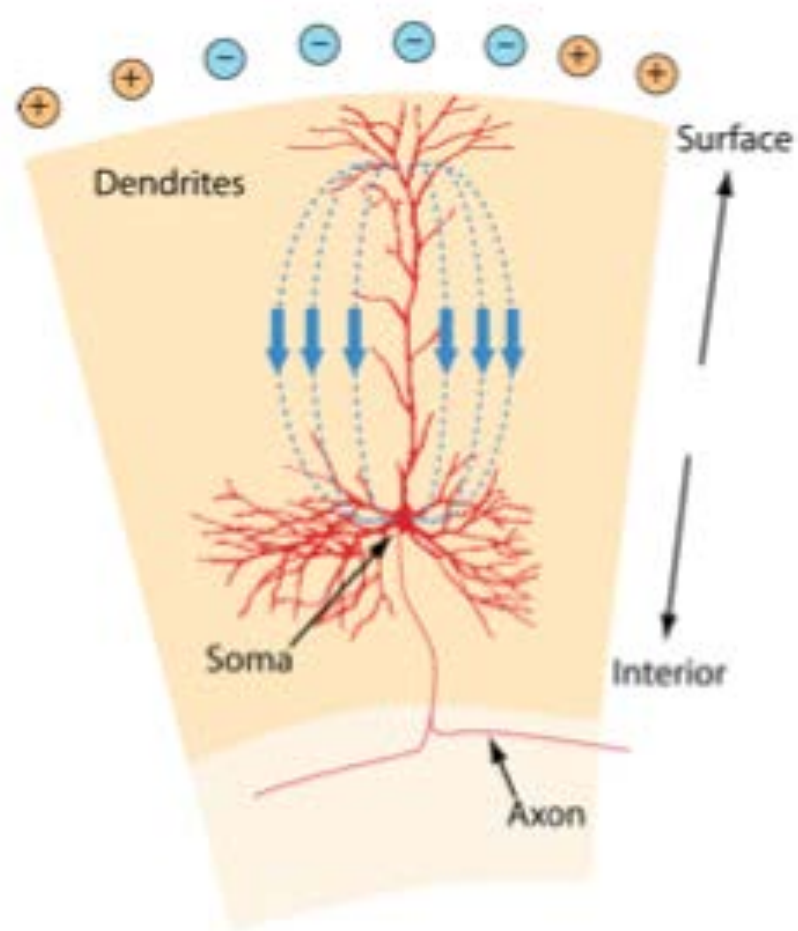
# eeg-fmri concurrent collection

Agatha Lenartowicz  
5/20/15

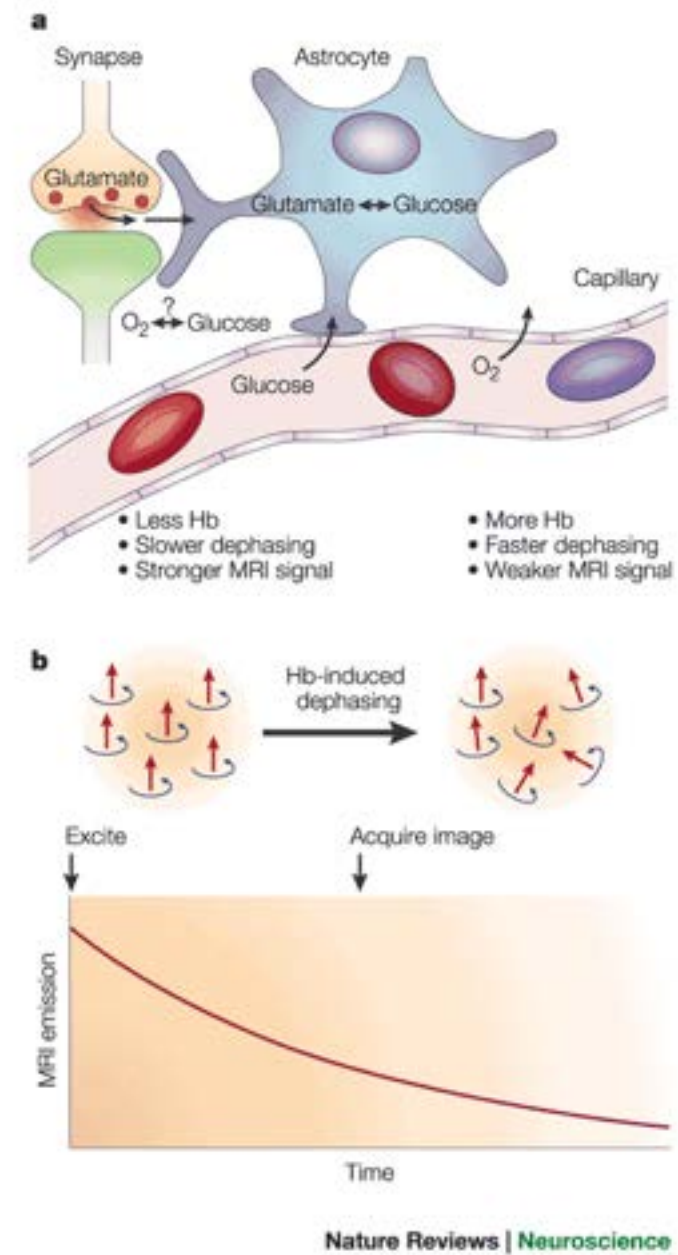
# WHY?



1. whole brain imaging of low invasiveness
2. with temporal resolution of neural events
3. and spatial resolution of neuronal populations\*

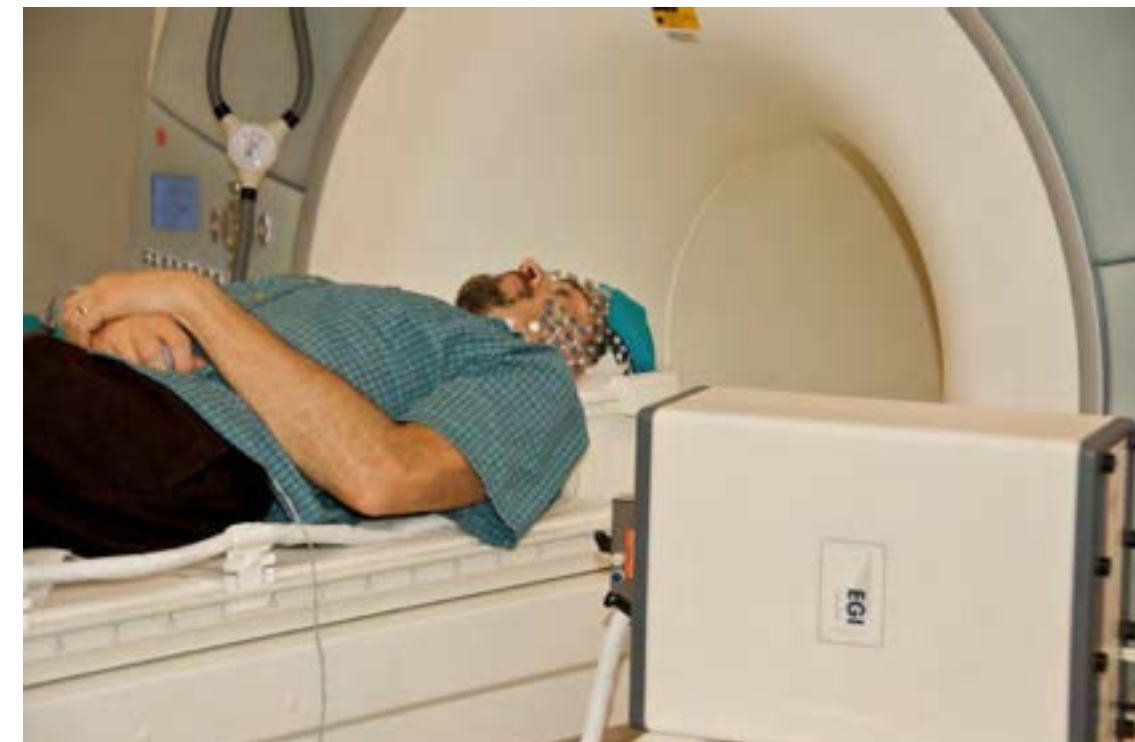
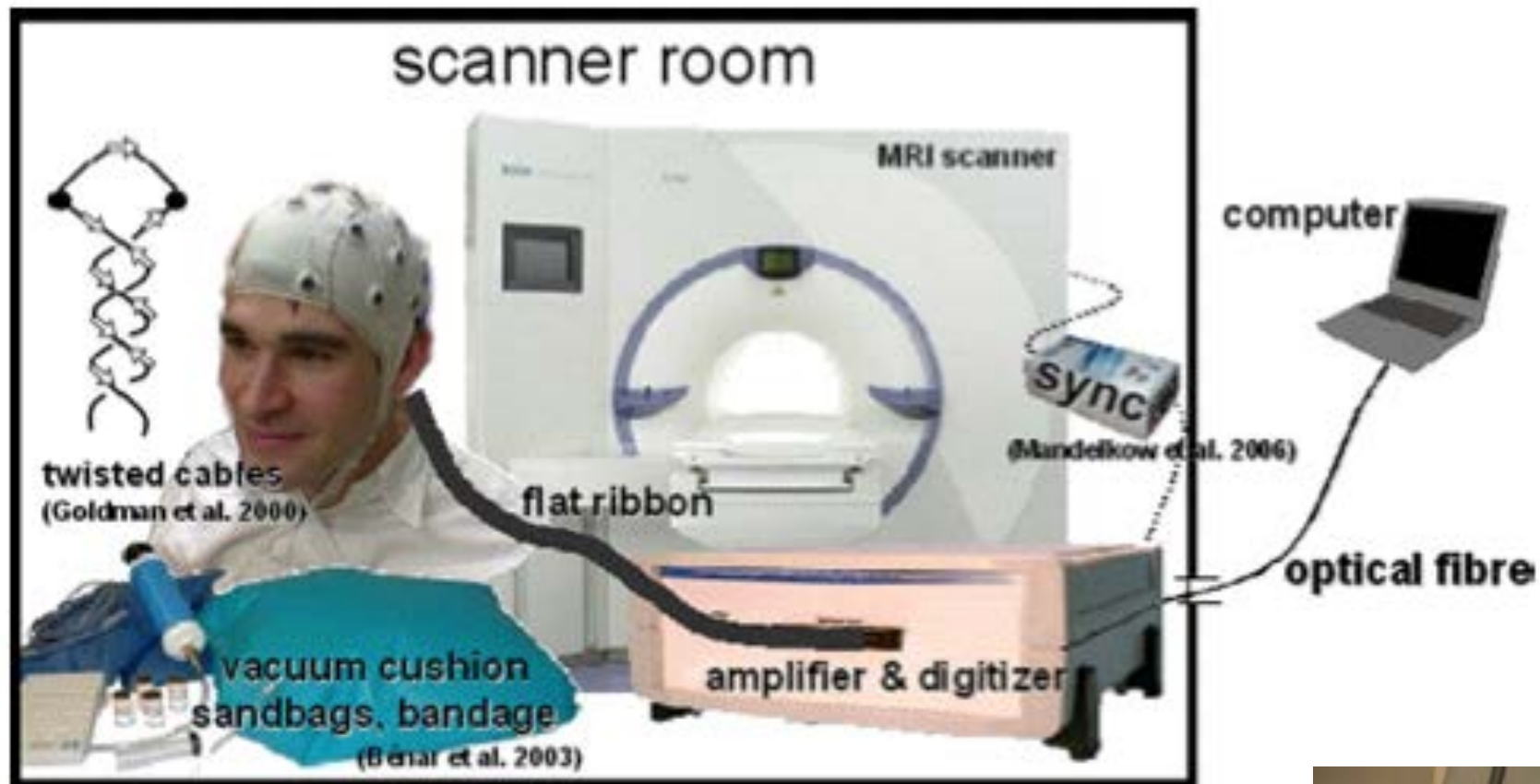


*EEG (electroencephalography)*



*fMRI (functional magnetic resonance imaging)*

# HOW DOES IT LOOK?

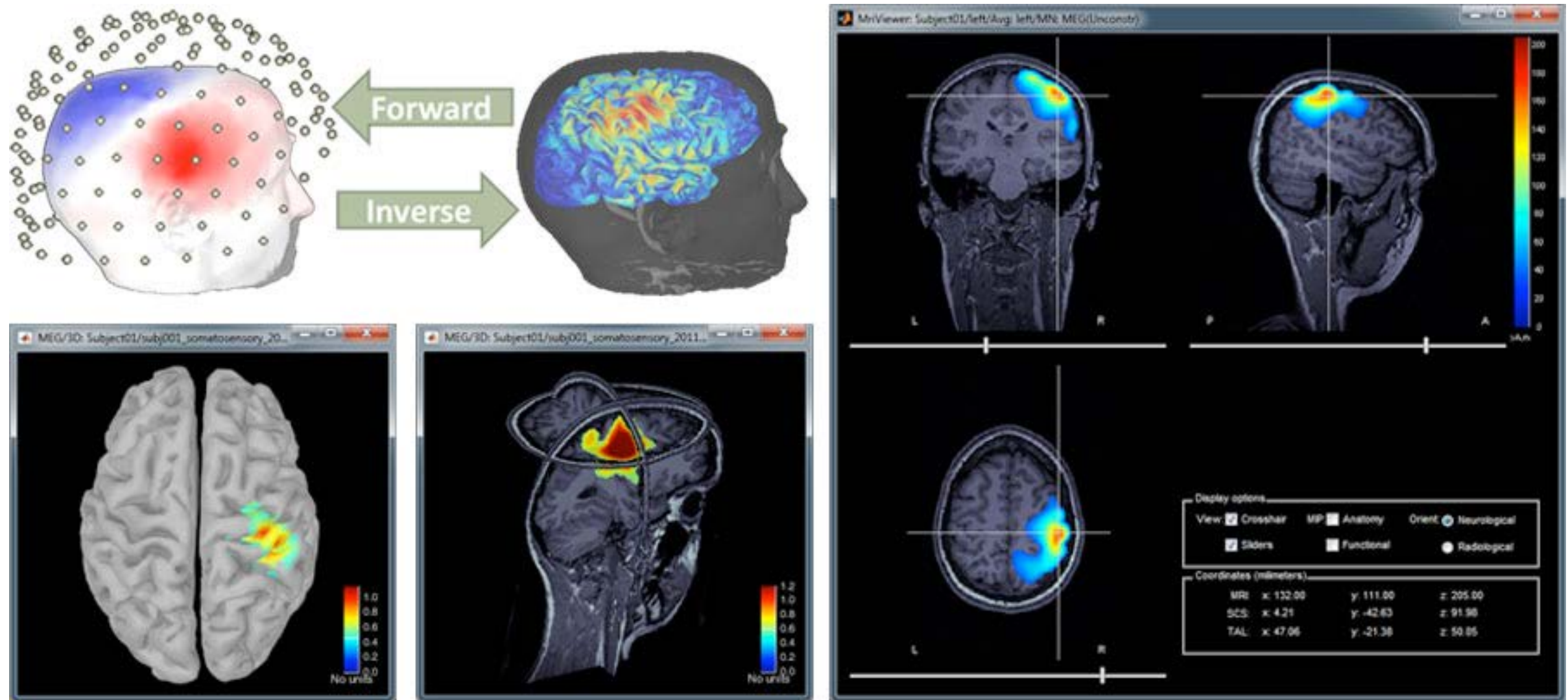


How do we integrate time and space data?

*asymmetric approach (use one to inform the other)*



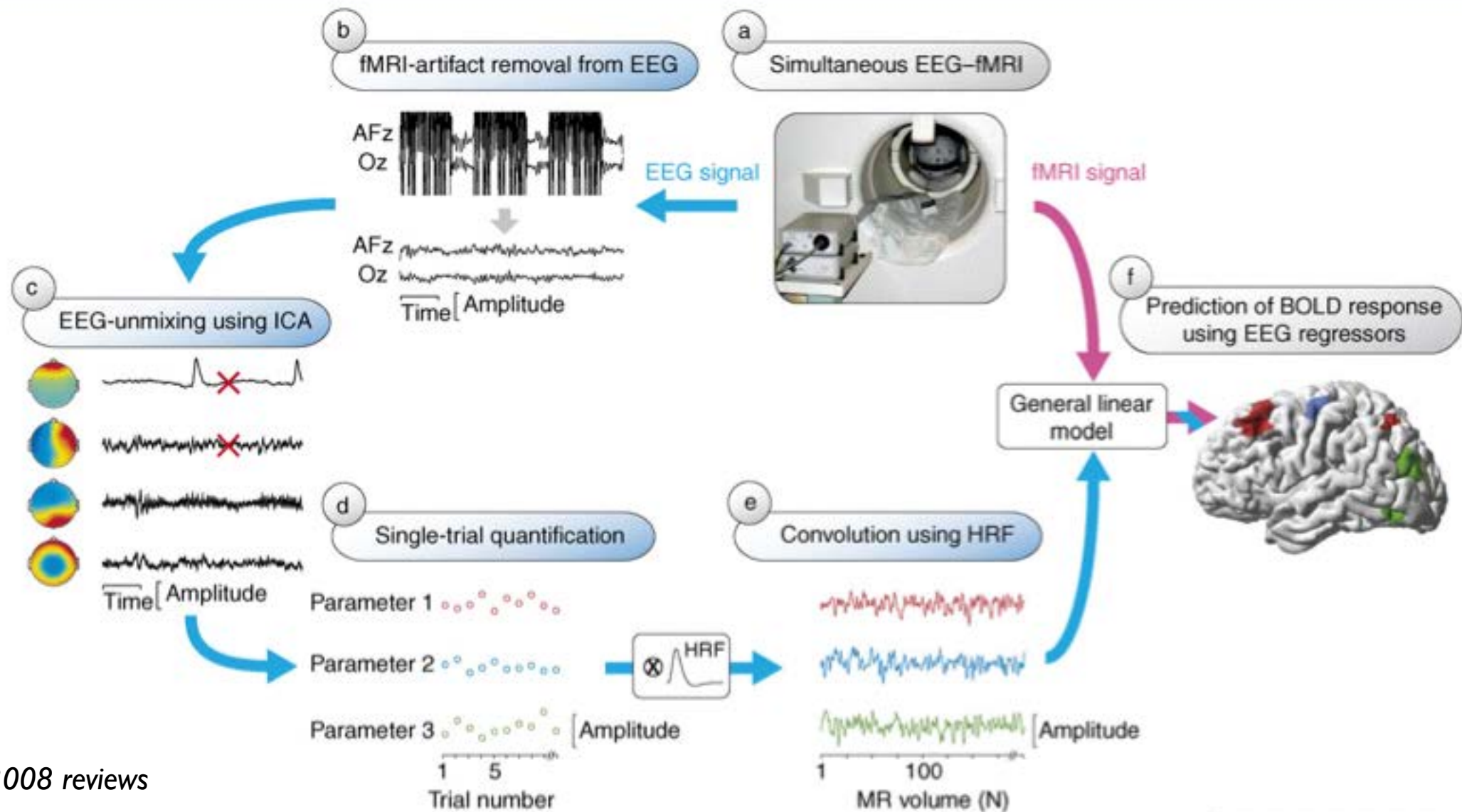
# Improve source modeling accuracy (“resolution?”)



*MRI and fMRI can be used:*

- *improve accuracy of electrode positions on head (registration between cap and skull)*
- *improve accuracy of head model for forward model*
- *constrain cortical search space for solution*
- *latter 2 points not dependent on ‘concurrent’-ness of recording*

# Look for eeg-correlated BOLD activations



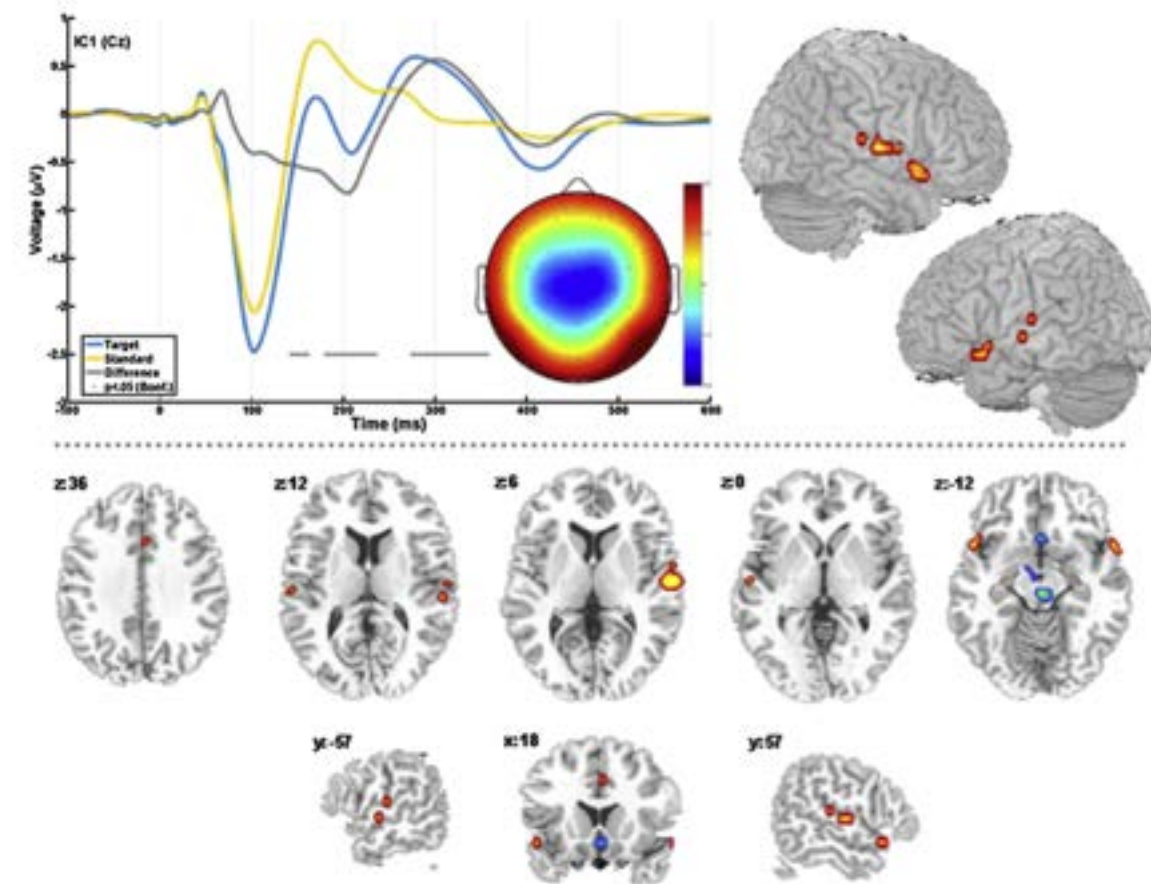
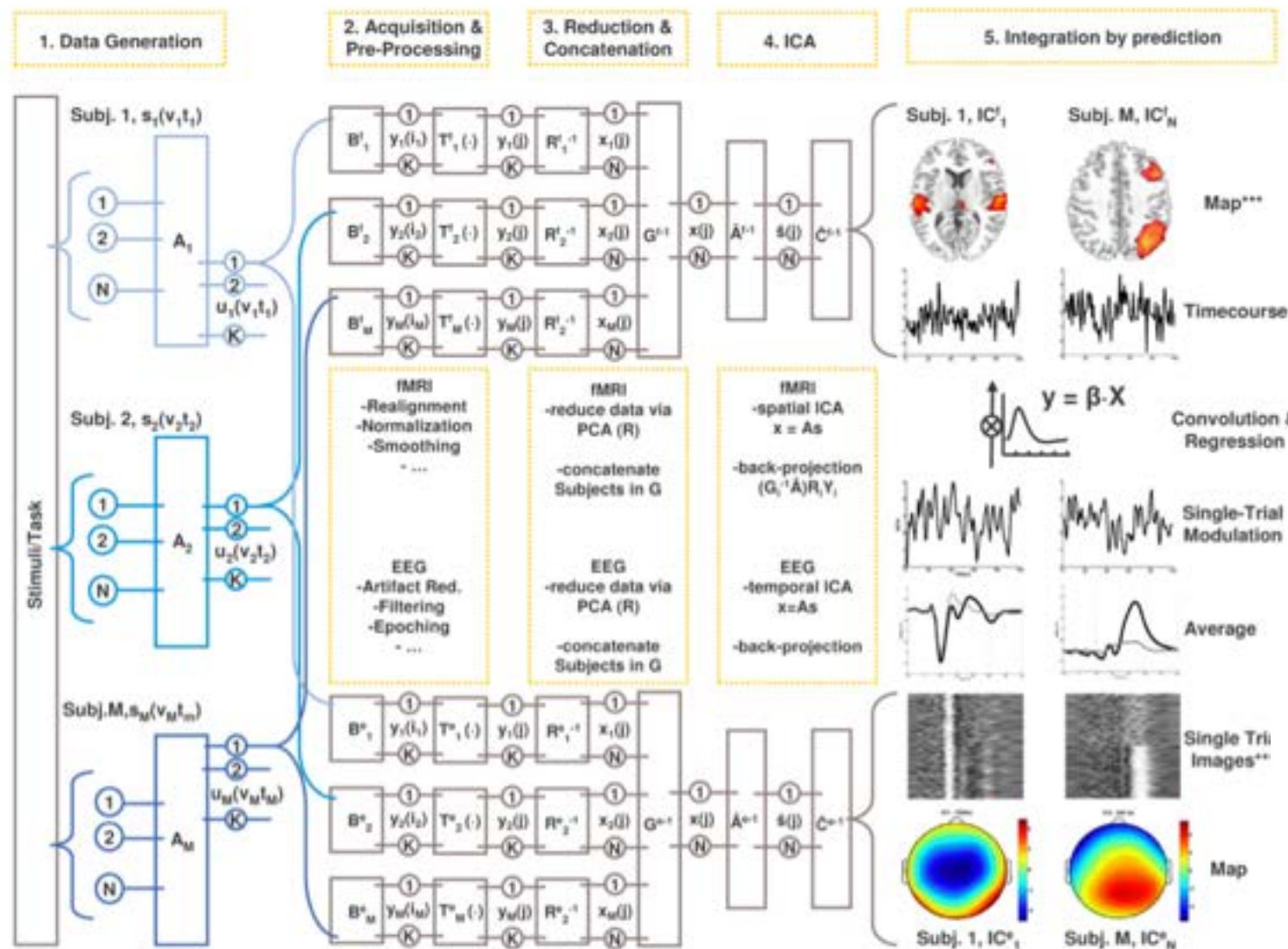
Debener 2006, 2008 reviews

TRENDS in Cognitive Sciences

*EEG can be used to:*

- *model single-trial events, which have to be convolved with HRF, and used as a regressor*
- *ask questions about timing (e.g., what is the BOLD correlate of alpha ERD or P1 amplitude during perception of a visual checkerboard)*





Eichele 2008

Tom Eichele (with Vince Calhoun) has done some of the nicest work extracting single-trial content from EEG data collected in scanner (all based on ICA).



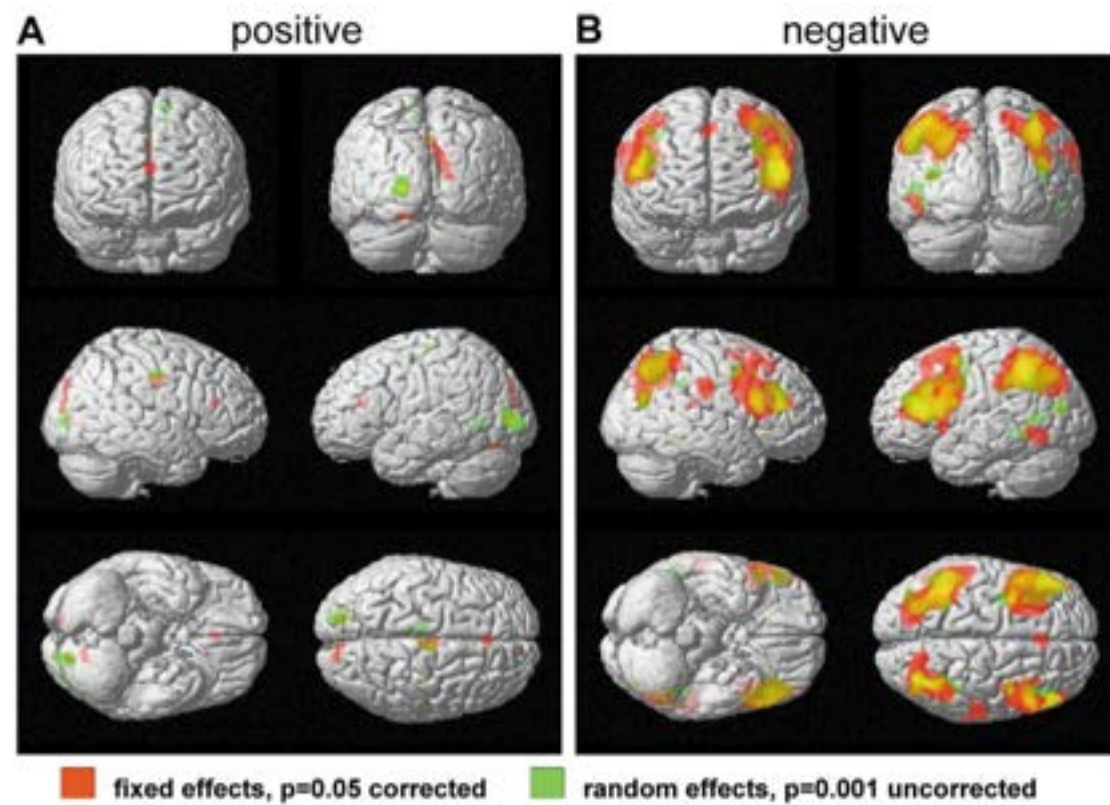
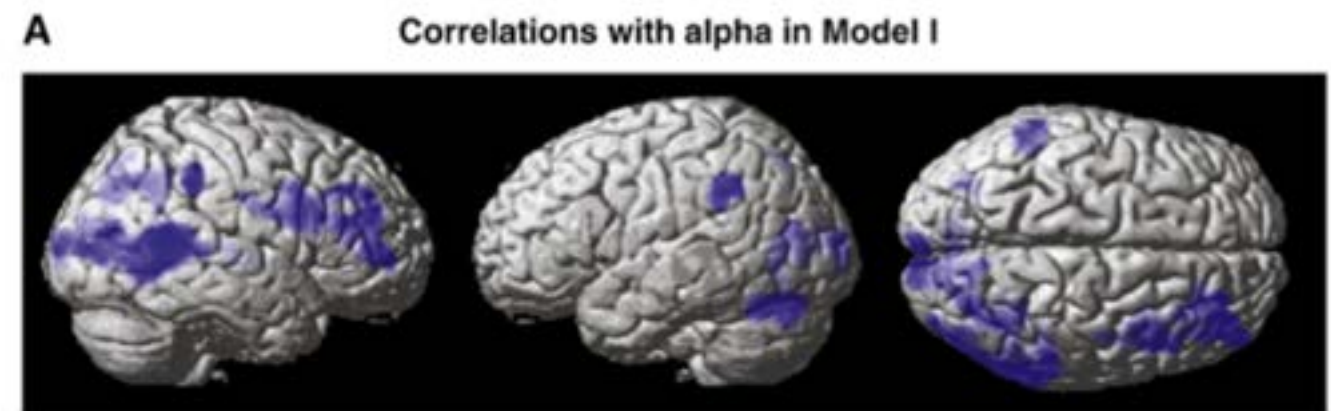
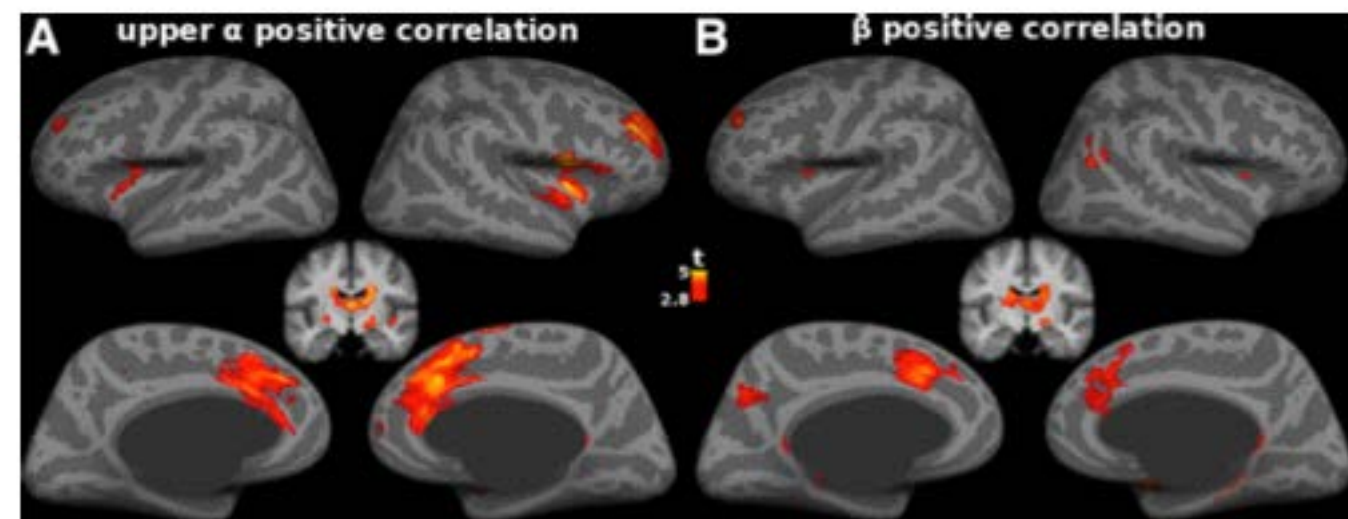


Fig. 3. Activation map. Brain areas activated in correlation with alpha band power. Superimposed on a surface rendered T1-weighted anatomical template brain an overlay of the statistical parametric maps (SPM  $t$ ) for a fixed effects (red;  $P < 0.05$ , corrected) and a random effects group analysis (green;  $P < 0.001$ , uncorrected) of 10 subjects is shown. Brain areas activated when alpha power is high (A, positive correlation with fMRI BOLD signal) are displayed on the left, and areas activated with decreased alpha power (B, negative correlation) are shown on the right.

Laufs 2003



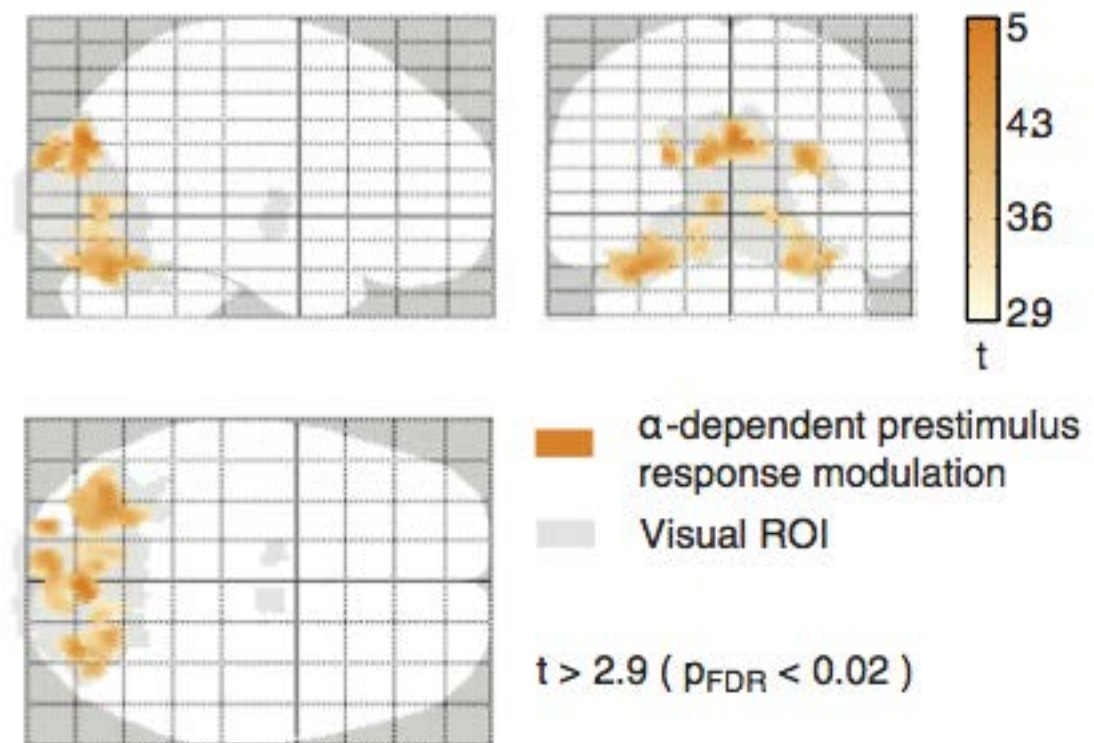
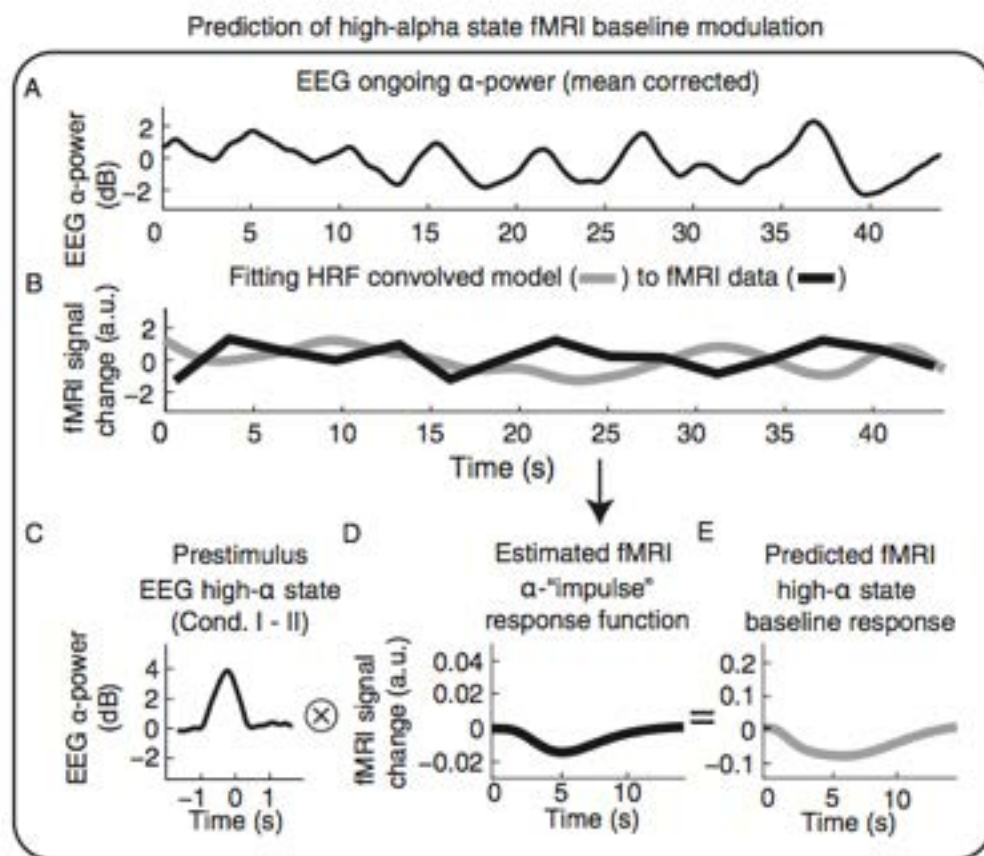
Scheeringa 2009



**Figure 3.** Positive correlation of activity with the global field power of oscillations in the alpha and beta bands. **A**, Positive correlations with upper alpha band power arise selectively within regions of the tonic alertness network including dACC, right anterior insula, right aPFC, thalamus and basal ganglia. **B**, Positive correlations with beta band power occur in some regions of the intrinsic alertness network, notably dACC and subcortical areas.  $p < 0.005$  uncorrected, extent  $> 100$  voxels, mapped on a canonical average inflated cortical surface and a coronal section [ $y = -12$ ].

Scheeringa 2010

Scheeringa has done some work with alpha as a predictor. Villringer has done some even nicer work.

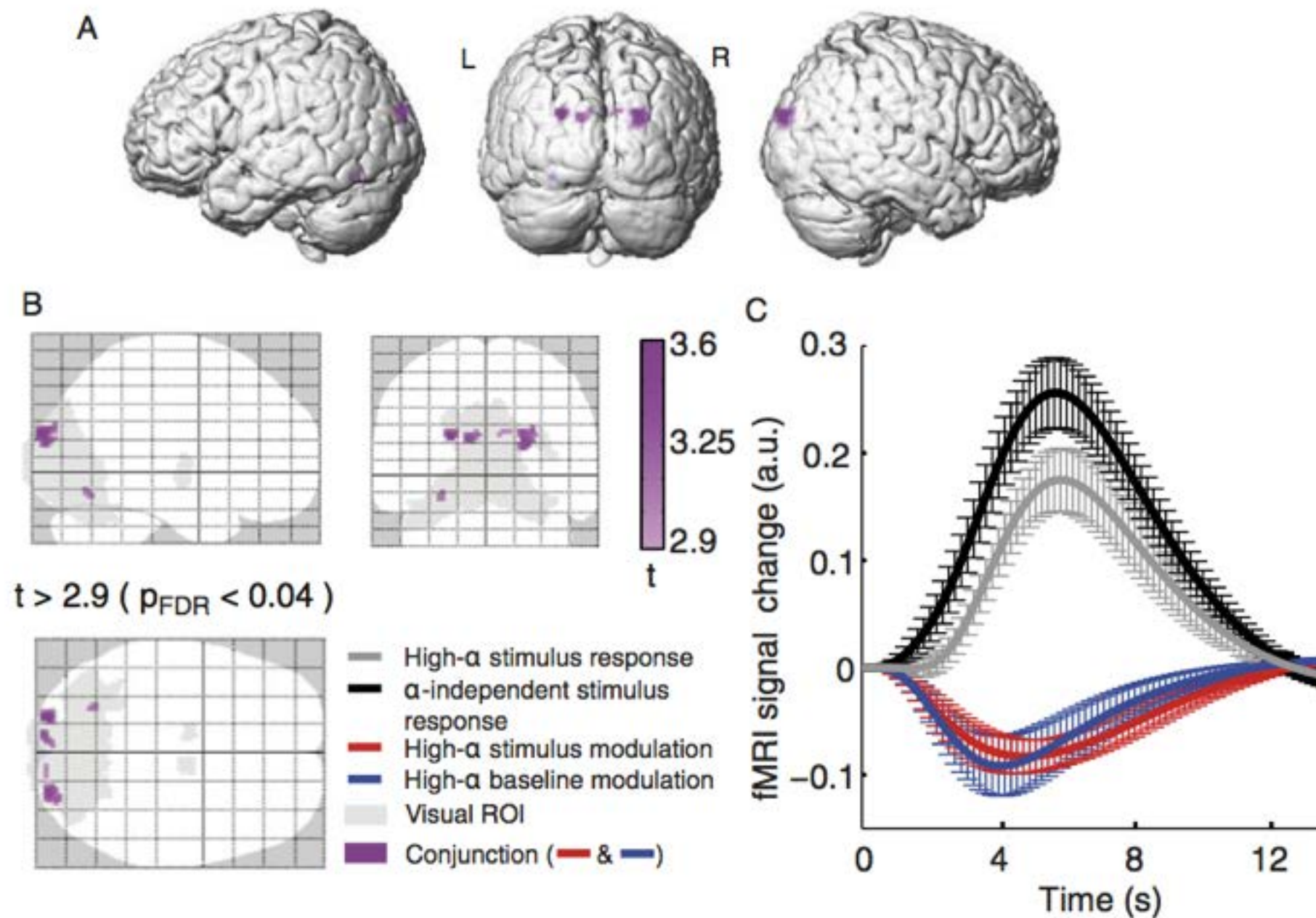


**Figure 8.** fMRI results of single-trial prestimulus alpha correlation analysis, testing the effect of fluctuating prestimulus alpha power on the single-trial evoked fMRI response [ $t > 2.9$ ;  $p < 0.02$ , FDR corrected ( $p_{FDR}$ ); extent threshold 10 voxels]. Found deactivations are depicted on a glass brain (orange; visual ROI, gray). For corresponding z values and MNI coordinates with anatomical labeling, see Table 1.

Becker 2010

Scheeringa has done some work with alpha as a predictor. Ritter/Villringer have done some even nicer work.

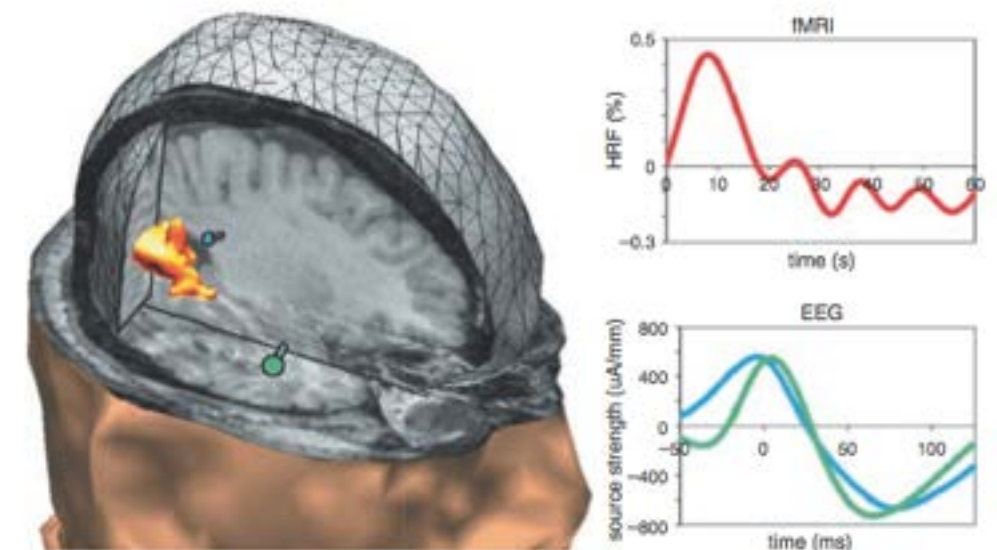
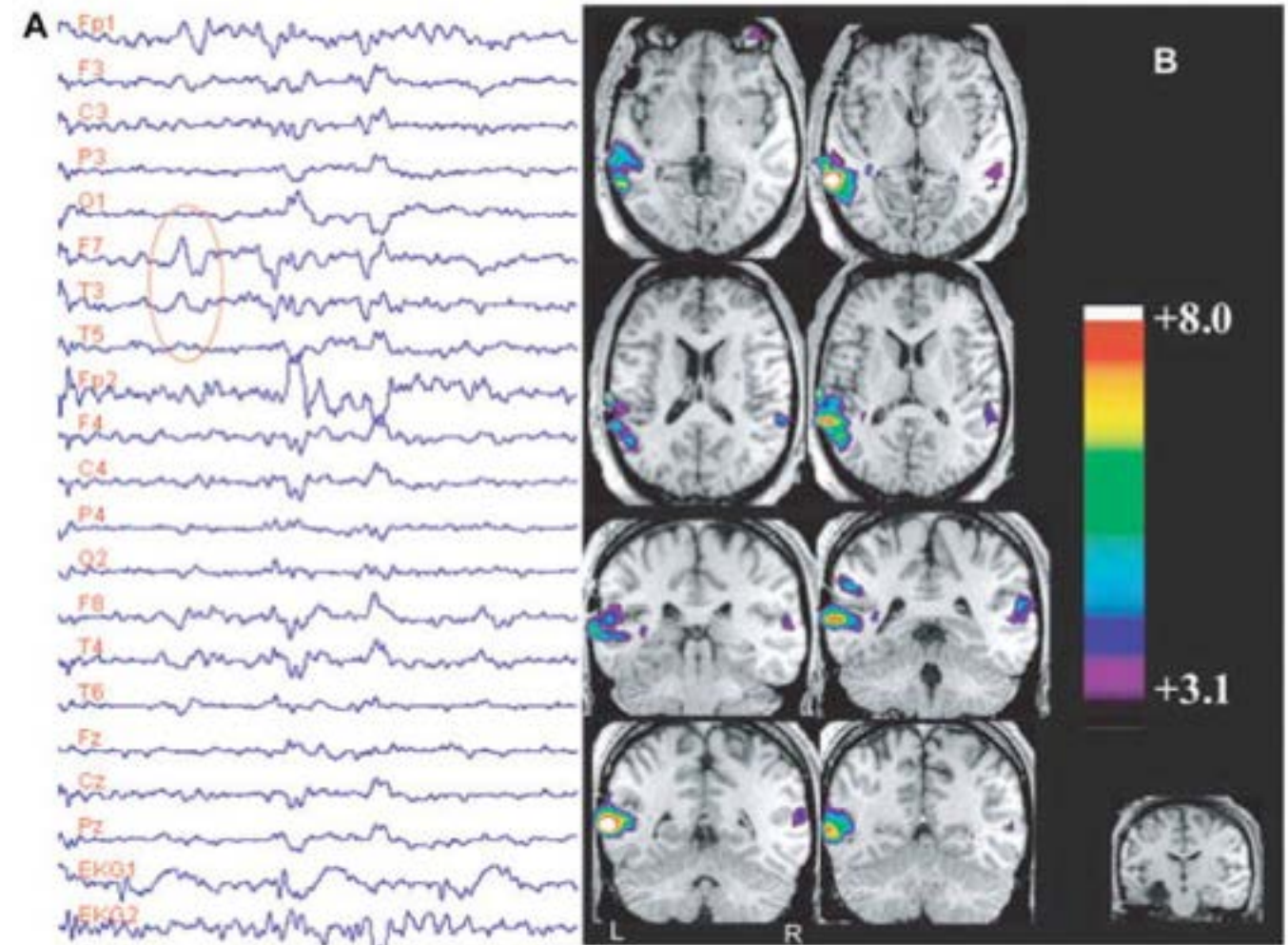
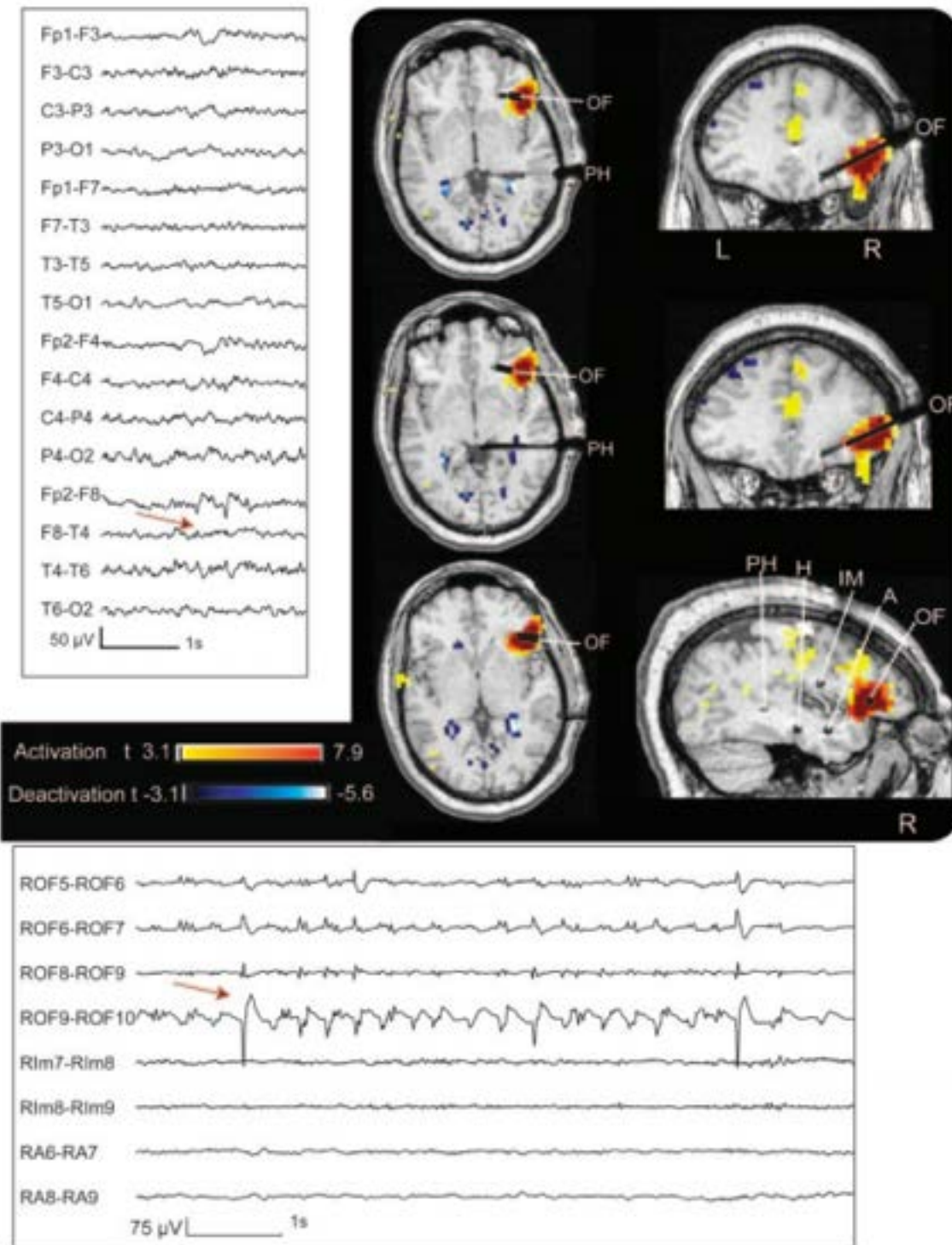




**Figure 6.** fMRI results of conjunction analysis. **A, B**, Occipital and occipitoparietal clusters deactivating during high-alpha activity during both stimulation and nonstimulation periods ( $t > 2.9$ ;  $p < 0.04$ , FDR corrected ( $p_{\text{FDR}}$ ); purple) depicted on a typical brain (Colin single-subject MNI brain template) and on a glass brain, together with the visual ROI (gray). **C**, In these areas, the observed difference in evoked fMRI responses due to high alpha stimulation (gray line) compared with state-independent stimulation (black line; difference depicted by red line) can be explained by the modulation of the fMRI baseline due to high alpha during nonstimulation periods (blue). Error bars indicate SEM across subjects. For corresponding  $z$  values and MNI coordinates with anatomical labeling, see Table 1.



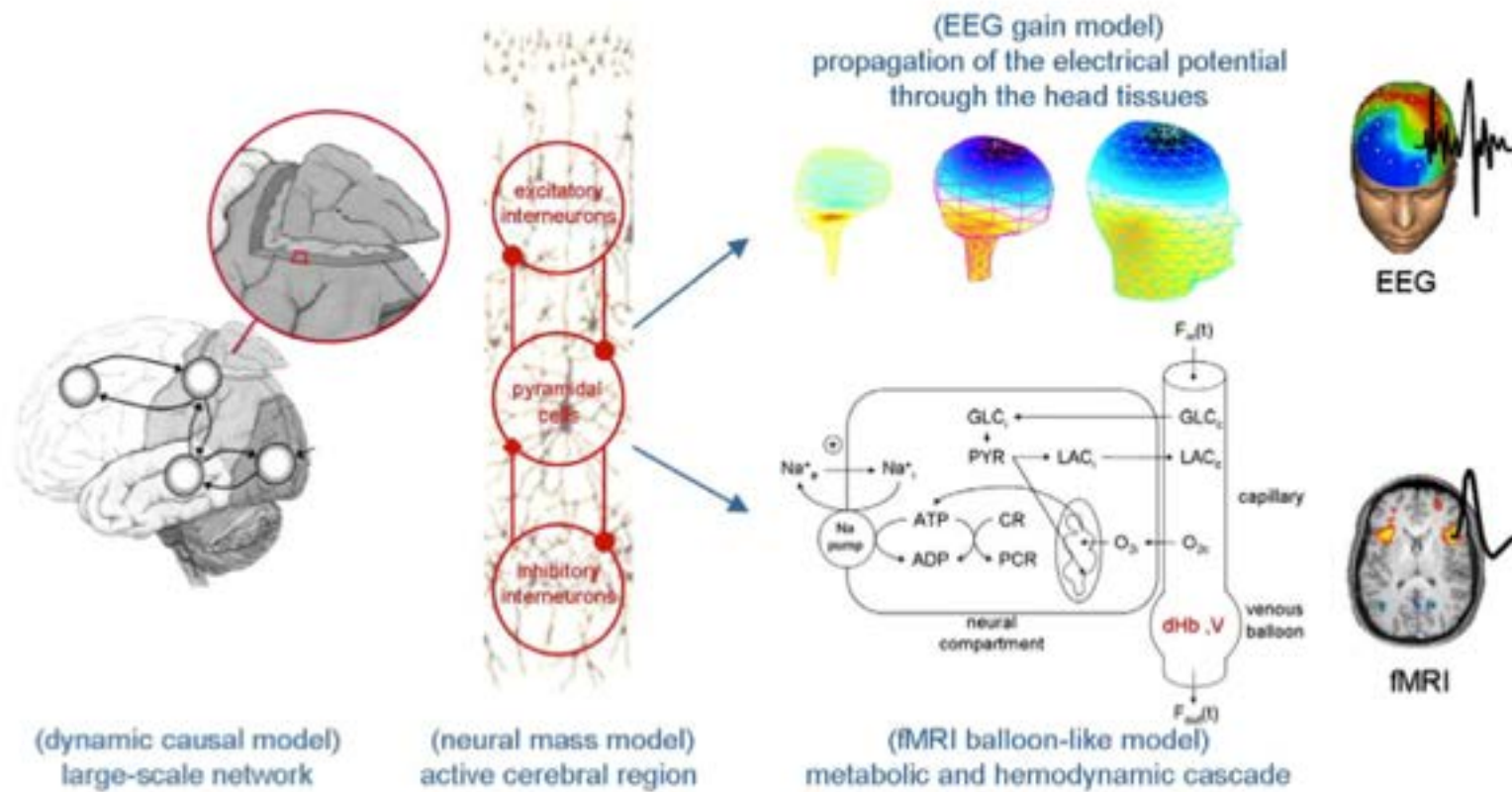
**Figure 1** A 25-year-old patient (patient 17) with right frontal epilepsy



**Fig. 6** Comparison of epileptic spike EEG dipole source localisation and fMRI activation (subject 1 of Bénar et al. 2002). Here, only one of the two dipoles (blue) matches the fMRI cluster. The evolution of the dipole strength at the concordant dipole location may provide additional time resolution

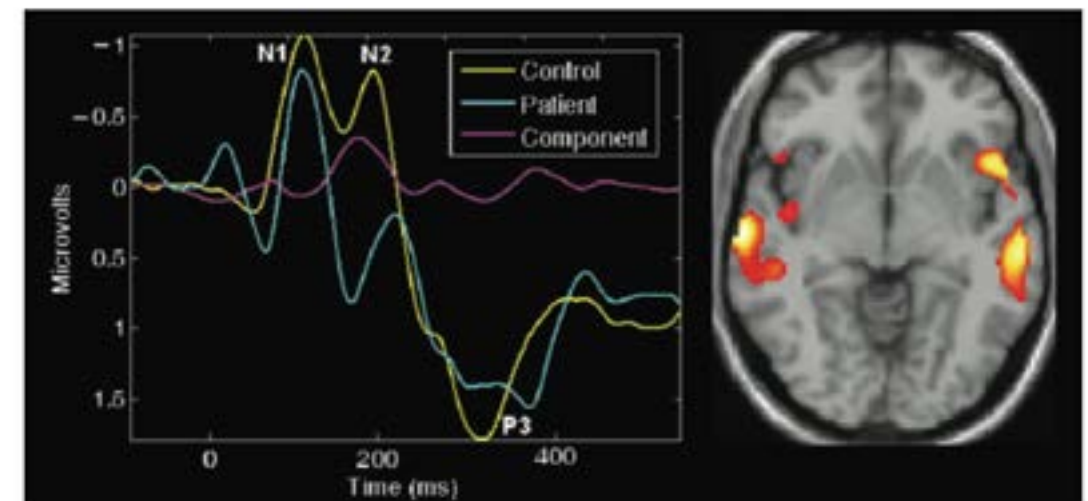


# Symmetric approaches and data “fusion”



Co-Analysis e.g., Joint ICA (Calhoun)

Modeling of shared variance - forward model for generation of both signals.



**Fig. 12.5.** ERP/fMRI jICA: Joint component which showed significantly different loading parameters ( $p < 0.0001$ ) for patients versus controls: (left) control (yellow; highest curve) and patient (blue; lowest curve) average ERP plots along with the ERP part of the identified joint component (pink; center curve) (see online version for color figures). (right) Thresholded fMRI part of the joint component showing bilateral temporal and frontal lobe regions

# Why would you **not** want to use EEG-fMRI?

- *experimentally the tradeoffs don't disappear*

*EEG => need many trials coming fast*

*fMRI => typically <30 trials coming slow or slow-ish  
you are always under-powered*

BLOCKED:



SPACED MIXED TRIAL:



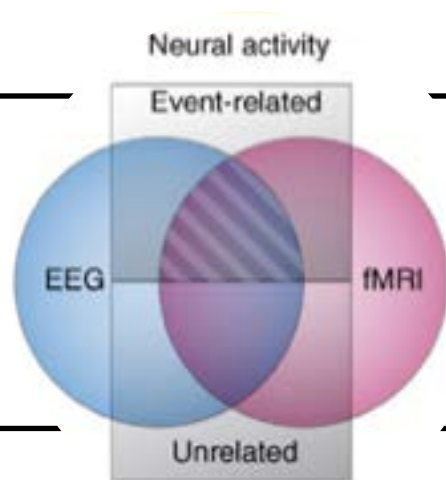
RAPID MIXED TRIAL:



From R. Buckner, HBM2001



# EEG and fMRI measure different things



	YES	NO
EEG	population extracellular potential; pyramidal cells aligned; surface	small population; randomly oriented cells; deep; sulci
fMRI	metabolic response that produces a BOLD response; LFP-related	anything that fails to occurs without changes in oxygenation of blood hemoglobin; brief, small localized effects

# EEG and fMRI measure different things

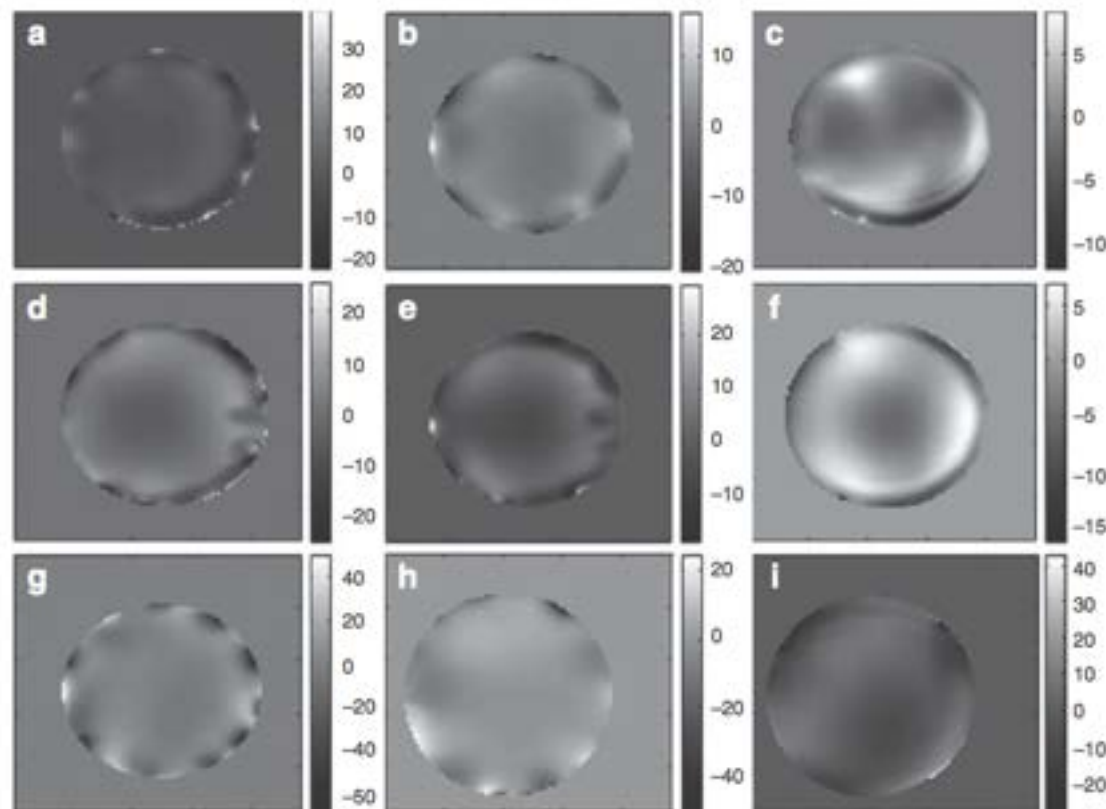
	EEG Yes	EEG No
fMRI yes	broad cortical responses	small population; randomly oriented cells; deep; sulci
fMRI no	very brief but strong responses, fast changes	neurotransmitters, small localized effects, non-pyramidal activity (?)

*Striatum (spiny neuron)? Stellate cells? Inhibitory cells? Sulcus activity in M1? Tapping right fingers vs toes  
1 Hz? Gamma neural activity?*

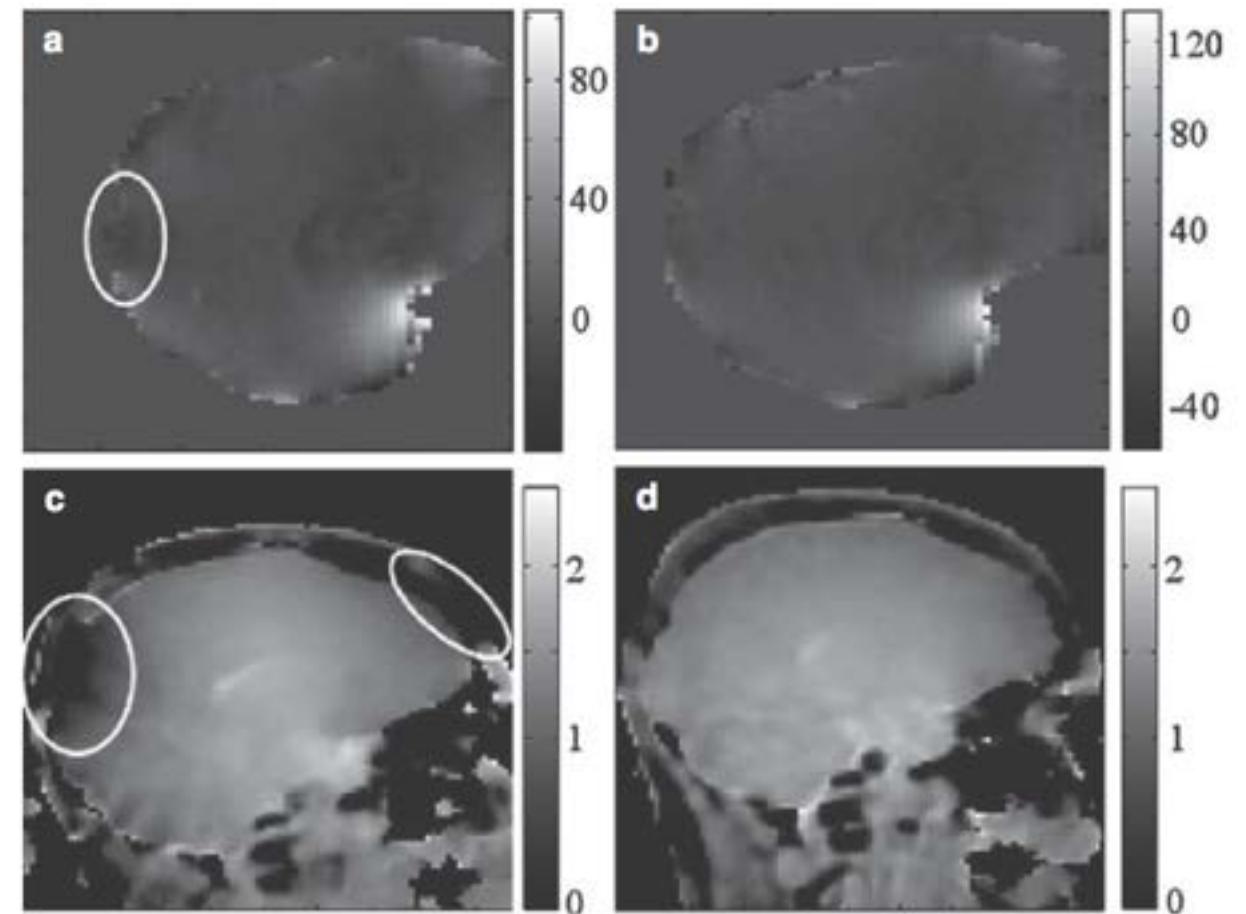
# Why would you not want to use EEG-fMRI?

*because it compromises data quality*

*fMRI ok*



**Fig. 5a-i**  $B_0$  field maps (in Hz) acquired from the phantom. Maps are shown after removal of large-scale field variations (due to the global shim) to view primarily the effect of the EEG cap at 1.5 T (a-c), 3 T (d-f) and 7 T (g-i) with the 64-electrode cap (left), 32-electrode cap (centre) and no cap (right) on. Reproduced with permission from Mullinger et al. (2007)



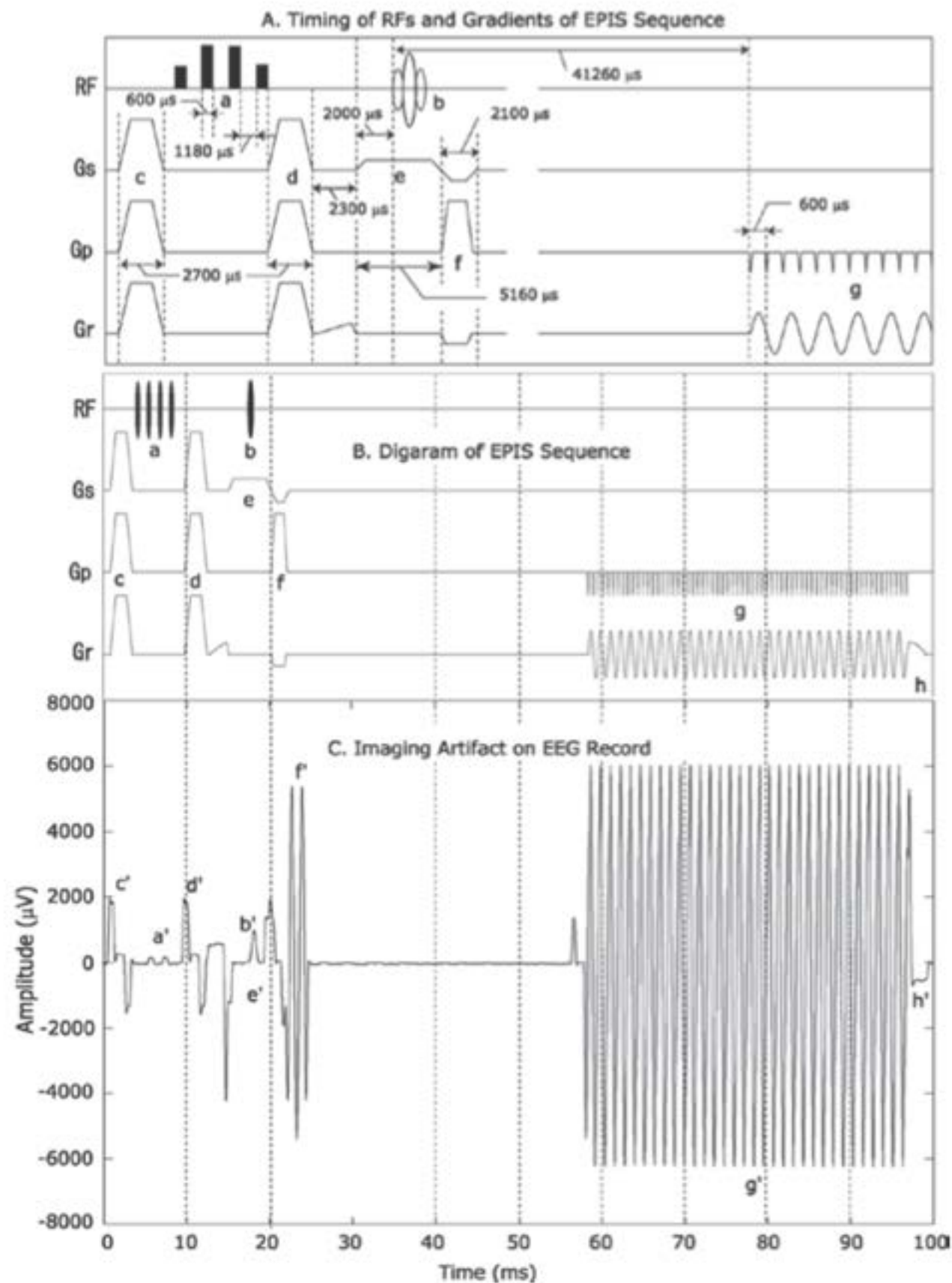
**Fig. 6a-d**  $B_0$  and  $B_1$  maps obtained in the human head. Effects of 32-electrode cap at 3 T on  $B_0$  maps (in Hz) (a, b) and flip angle maps (normalised to average flip angle) (c, d). a, c Acquired with the cap on (regions affected are highlighted); b, d with no cap. Reproduced with permission from Mullinger et al (2007)

*seldom discussed*

*can get susceptibility artefact around electrodes & RF coupling in wires disturbs B1 excitation efficacy (bigger for longer wires)*



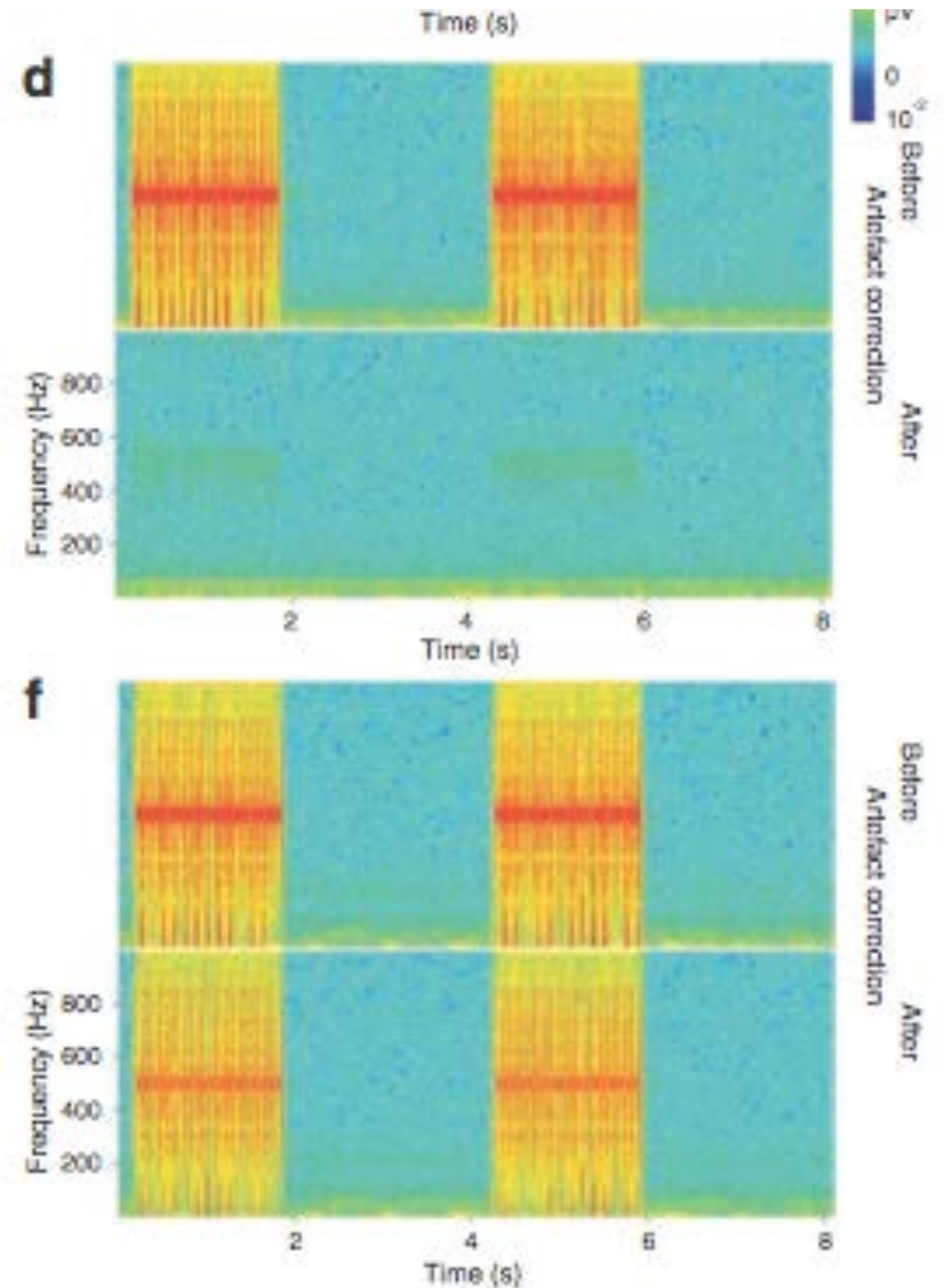
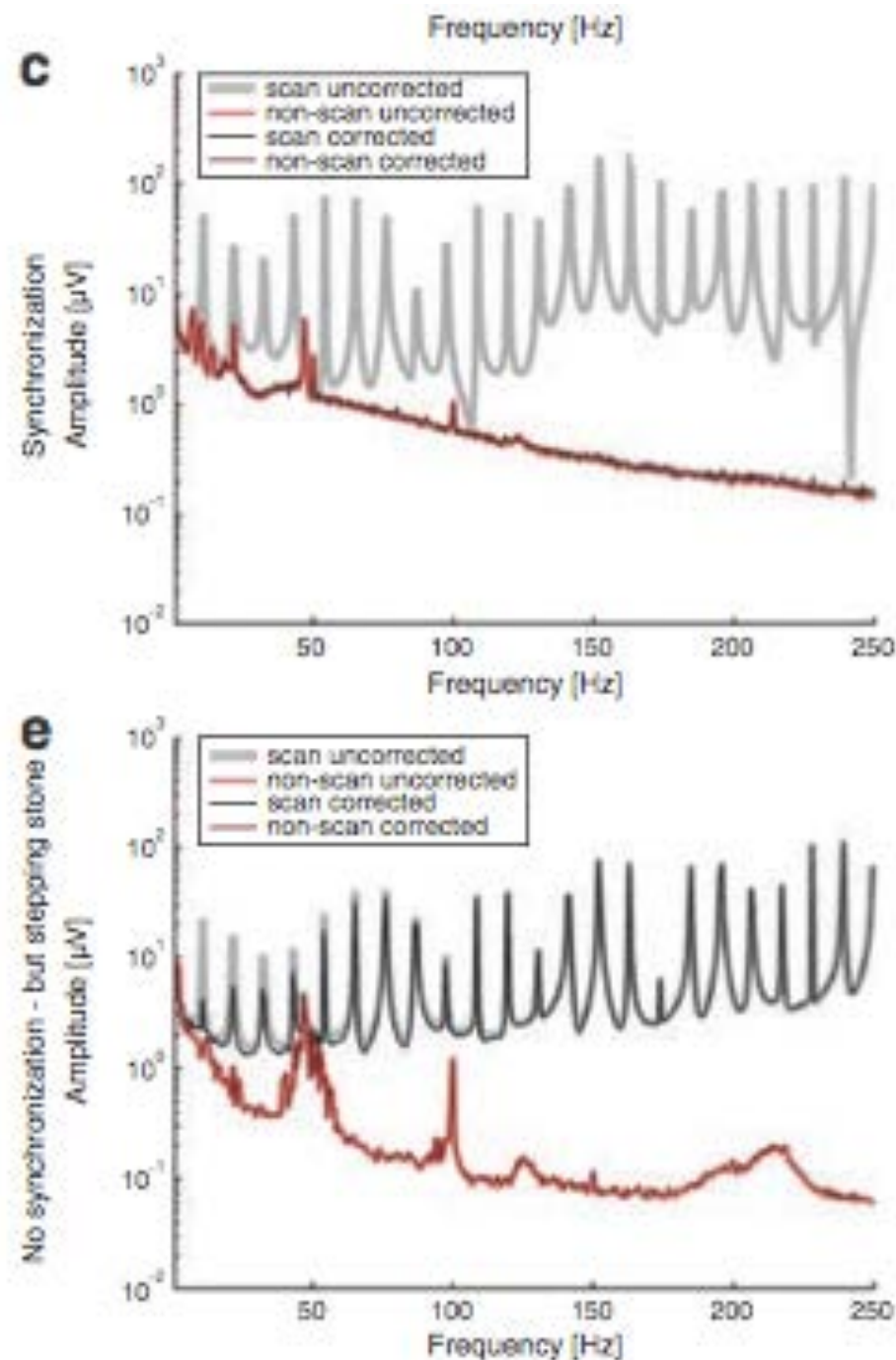
# EEG severely impacted by MR-related artifacts



least severe (but largest amplitude) is MR-gradient artifact caused by magnetic field gradients used for signal localization (electromagnetic induction in circuit, by varying magnetic field)

# EEG severely impacted by MR-related artifacts

assuming synchronization between clocks of EEG and MR pulses, the gradient artifact can be removed using template subtraction  
because of its deterministic nature (reality a bit more complicated)  
\* dominant frequency in EPIs varies with TR and slice timing

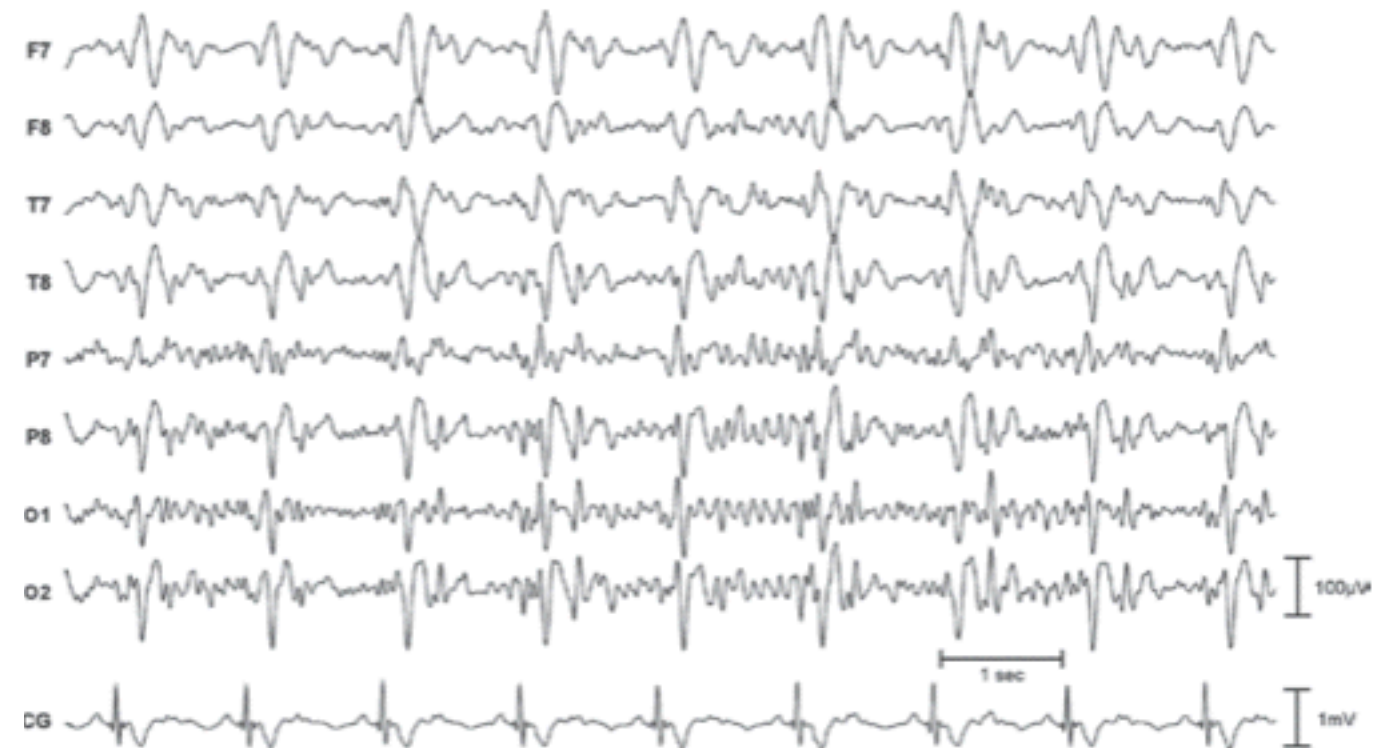
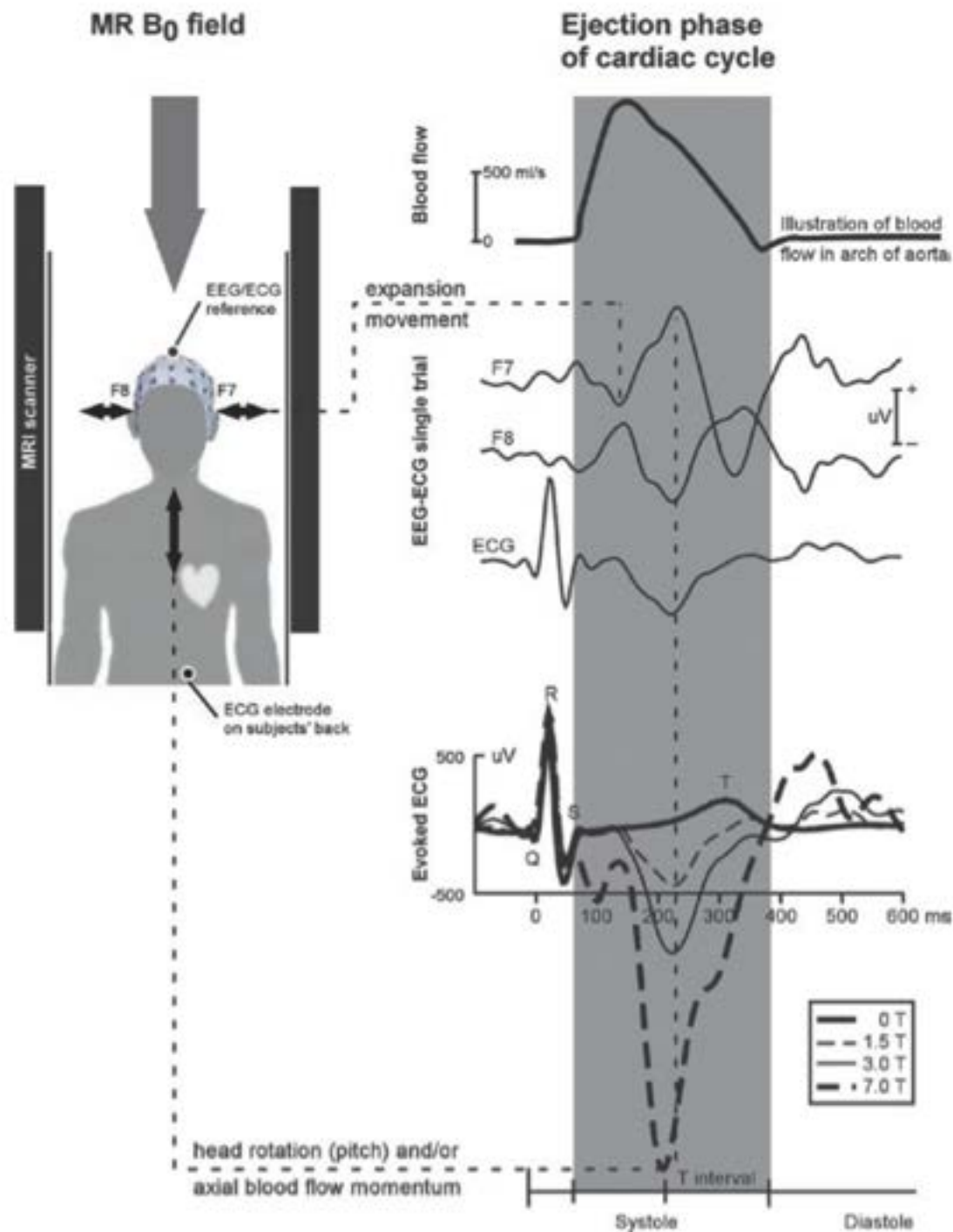


most severe is BCG (ballistocardiogram)

notice magnitude of alpha versus BCG

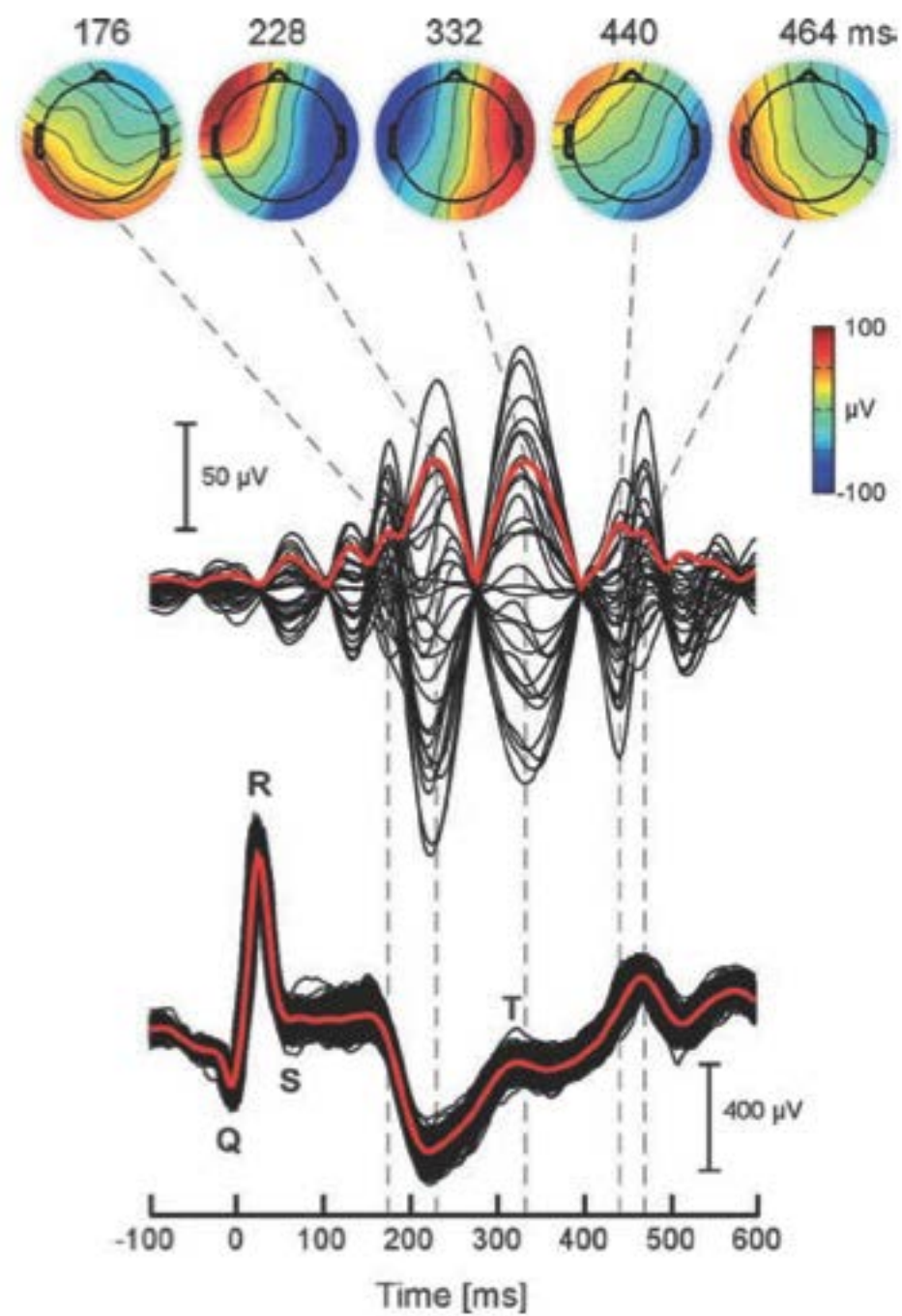
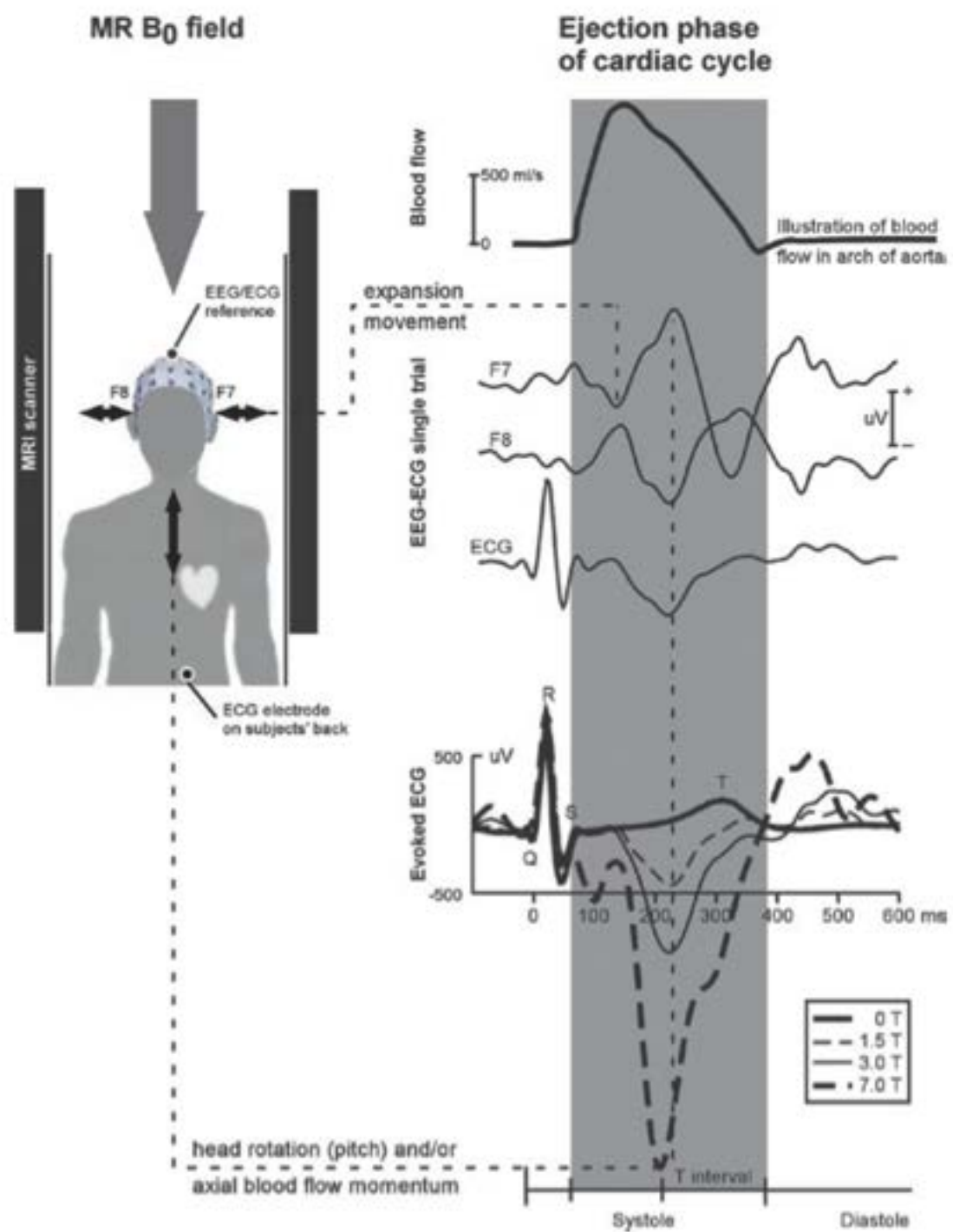
origin is somewhat debated but it's related to electromotive forces (current) created by a moving electroconductive material (leads):

- axial head rotation
- pulsatile movement of major blood vessels
- Hall effect (abrupt changes in blood velocity)

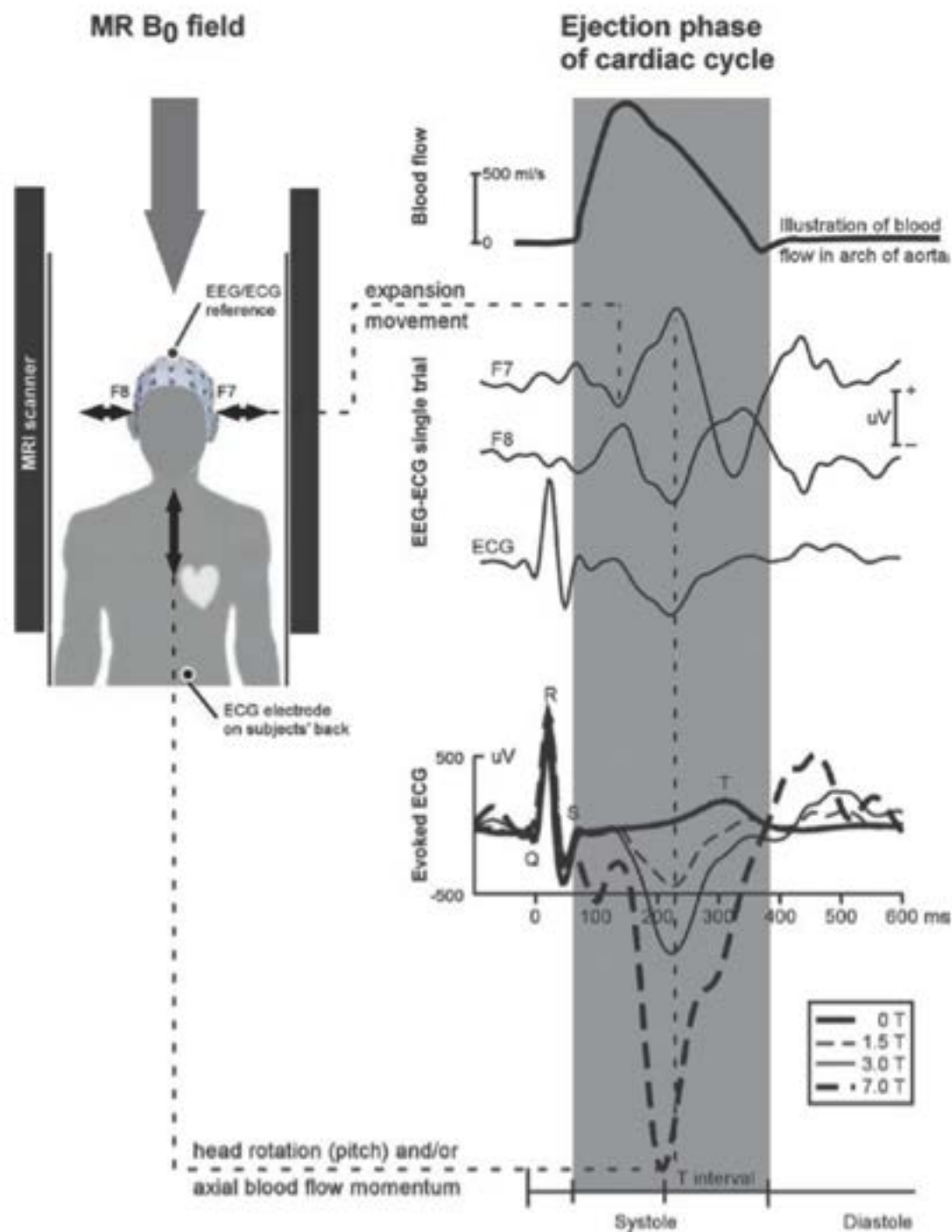




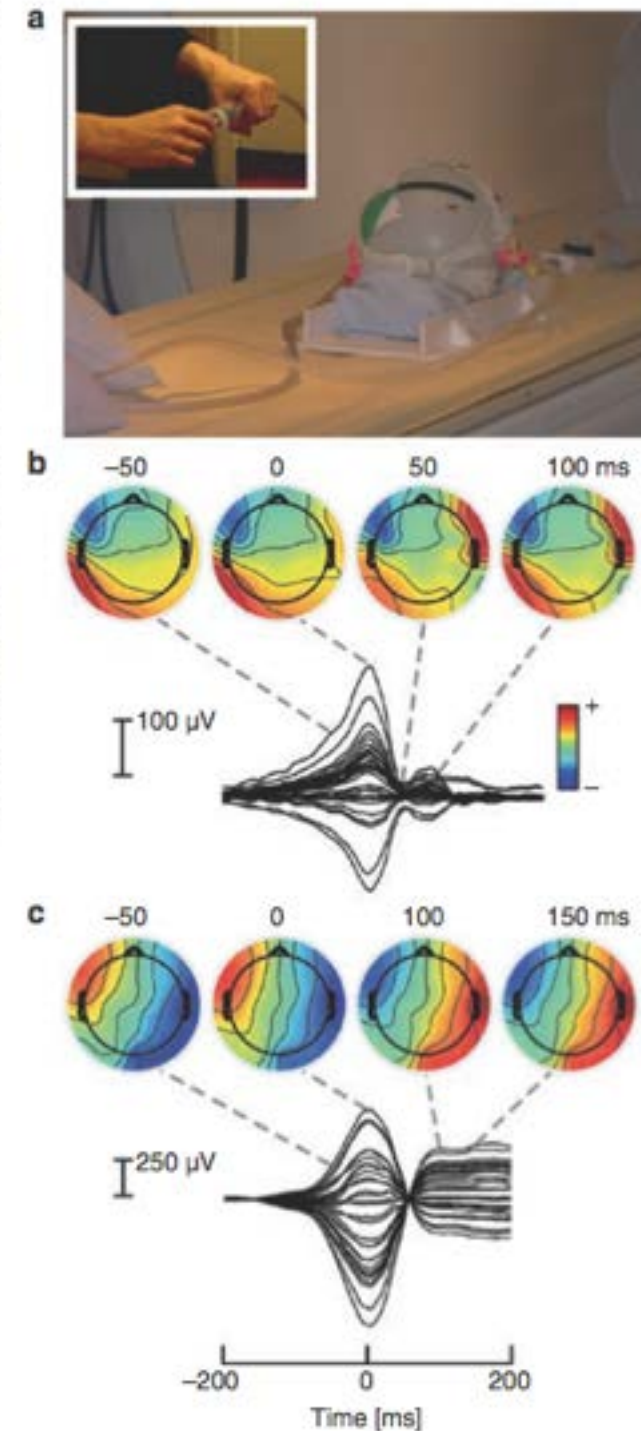
most severe is BCG (ballistocardiogram)



most severe is BCG (ballistocardiogram)

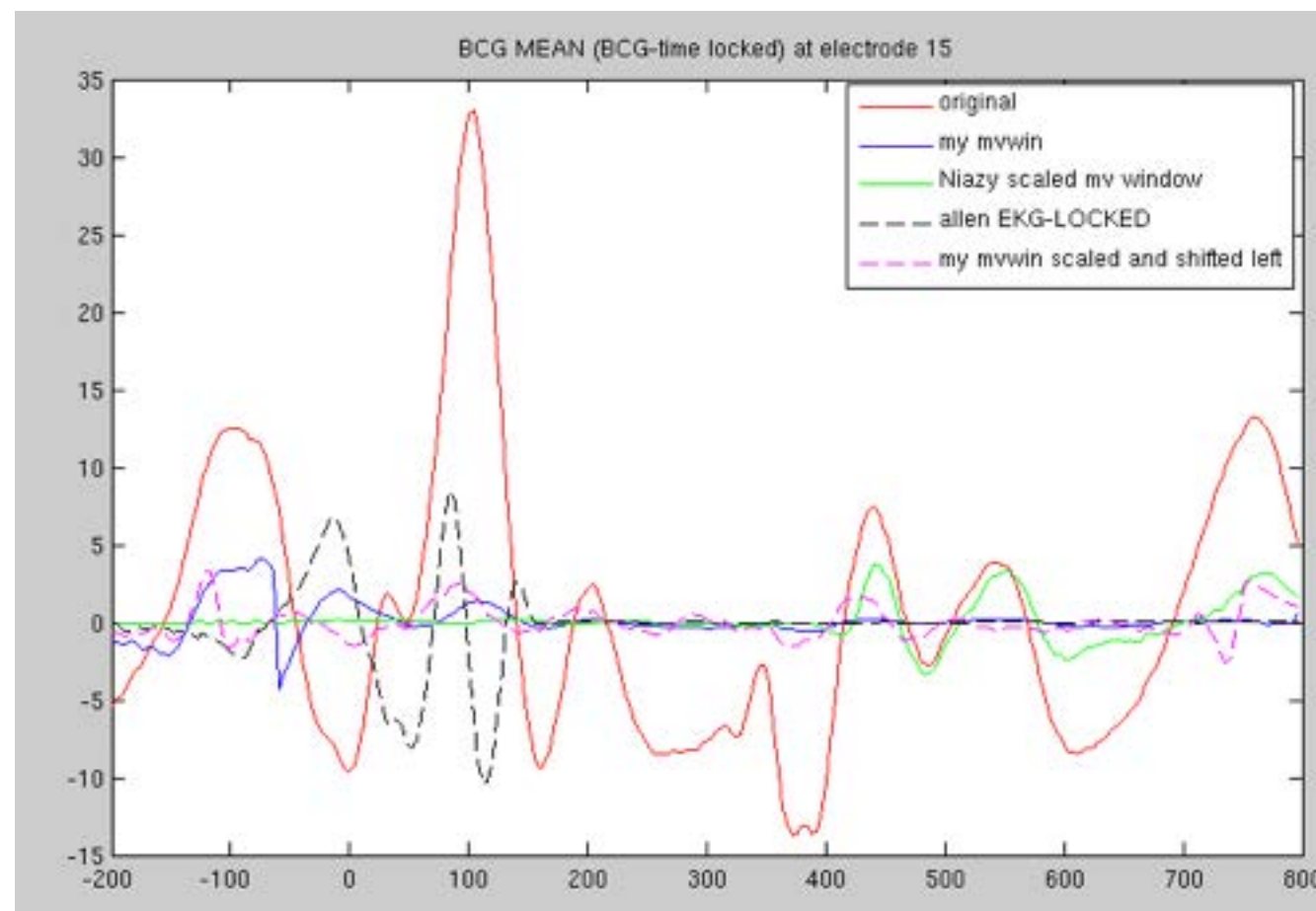


**Fig. 4** Results of a motion simulation study. A spherical phantom was covered with a layer of electrode gel, fitted with an EEG cap, and positioned in the centre of an MRI scanner (a). Different types of motion were induced while EEG was recorded, among them bilateral expansion motion (b), as caused by the inflation of balloons positioned underneath temporal electrodes, and axial, nodding head motion (c). Recordings and the respective voltage topographies shown in (b) and (c) are based on averages over a few repetitions. Lateral expansion motion caused rather locally circumscribed voltages, which may resemble tangential (*left*) or radial (*right*) features. Axial nodding head rotation, on the other hand, contributed a low spatial frequency map, which was characterized by a polarity change over time, and by different polarities between left and right hemisphere electrodes

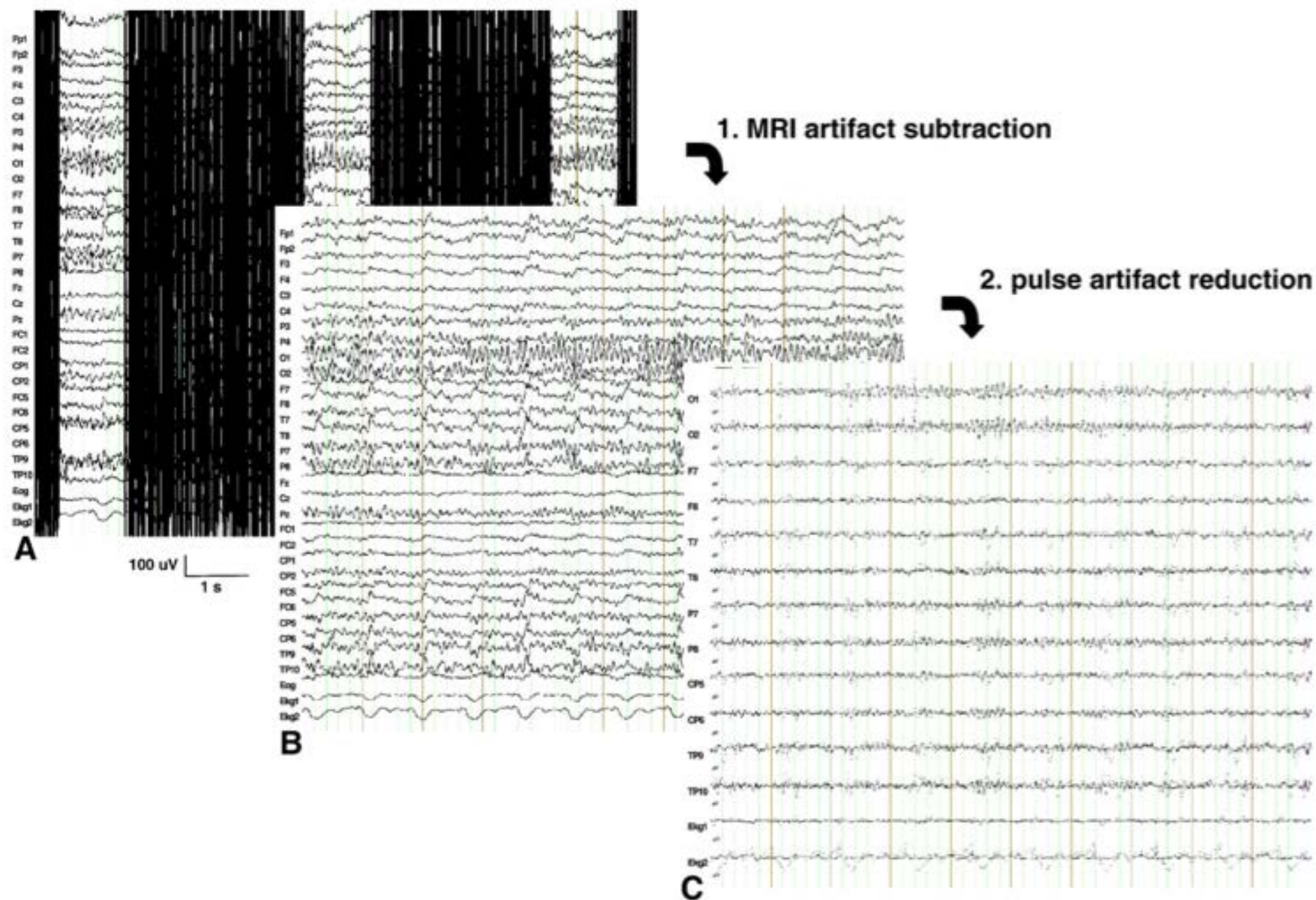


# approaches to remove BCG?

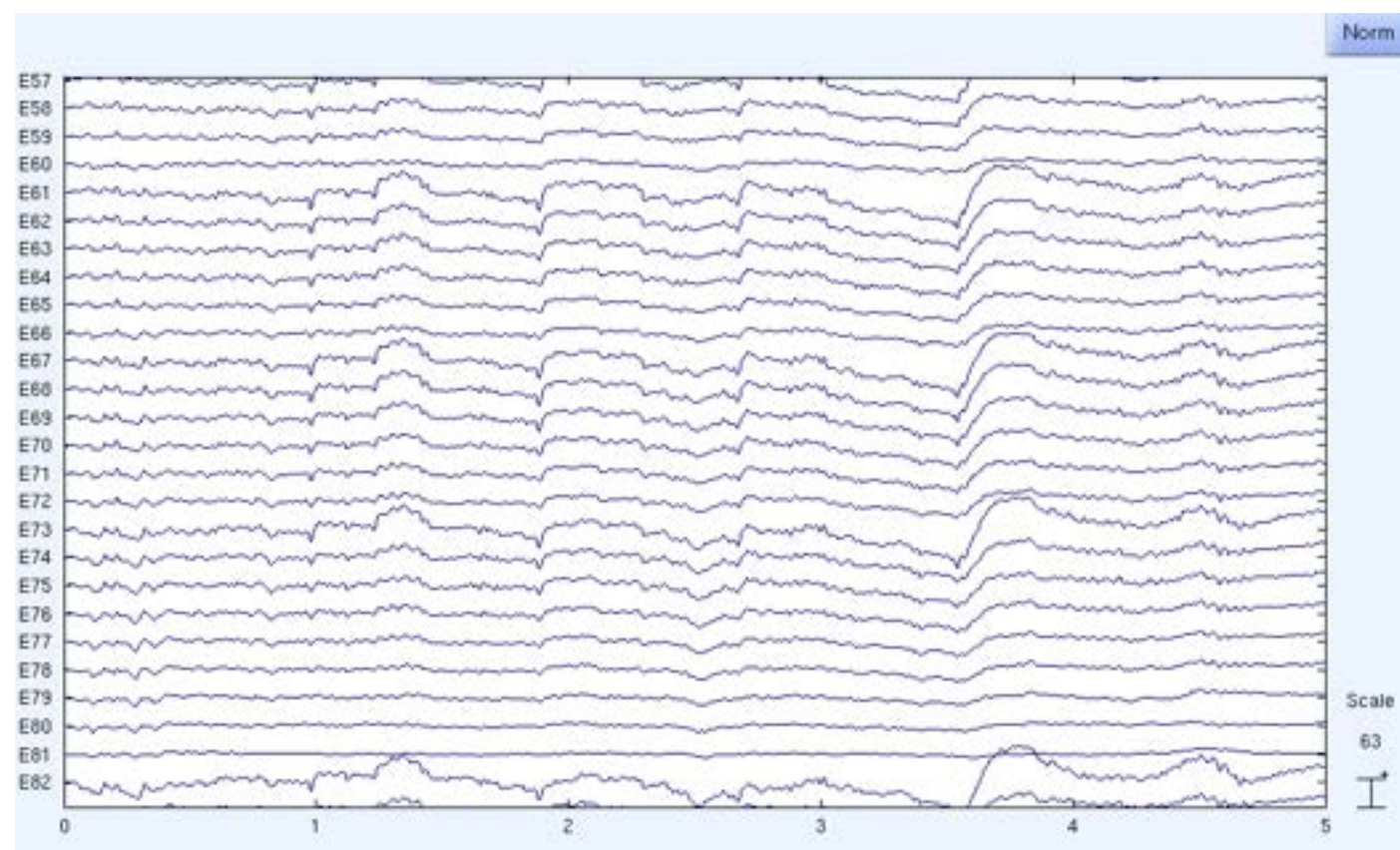
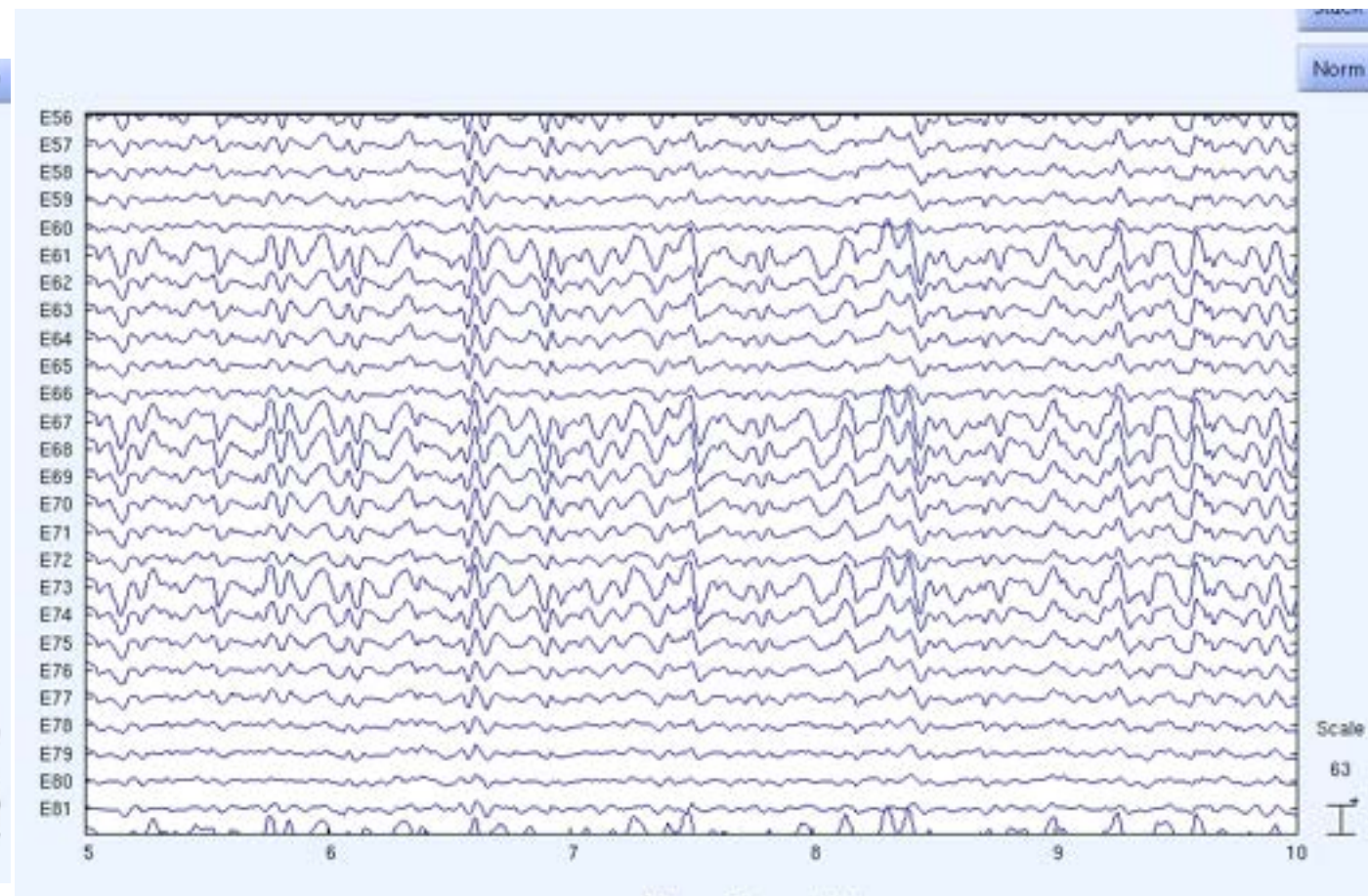
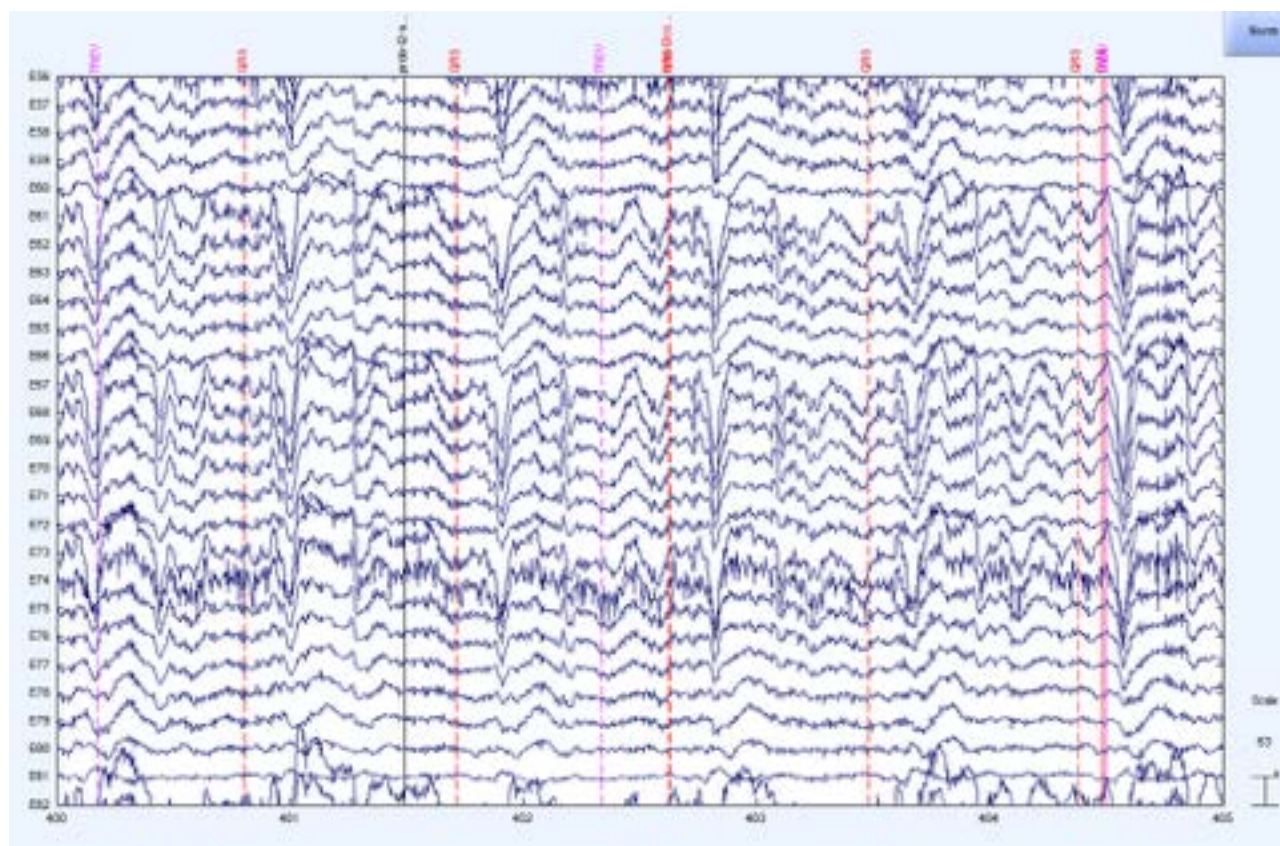
- preventative
  - immobilize the head
  - twist leads to minimize induced current
  - record artifact at electrode and subtract out (\*\*)
  - interleaved acquisition of MR/EEG
  - pulse-triggered acquisition
- post-hoc
  - template subtraction (just like for MR artifact - complicated b/c not stationary)
  - ICA approaches (ok 1.5T fails >1.5T)





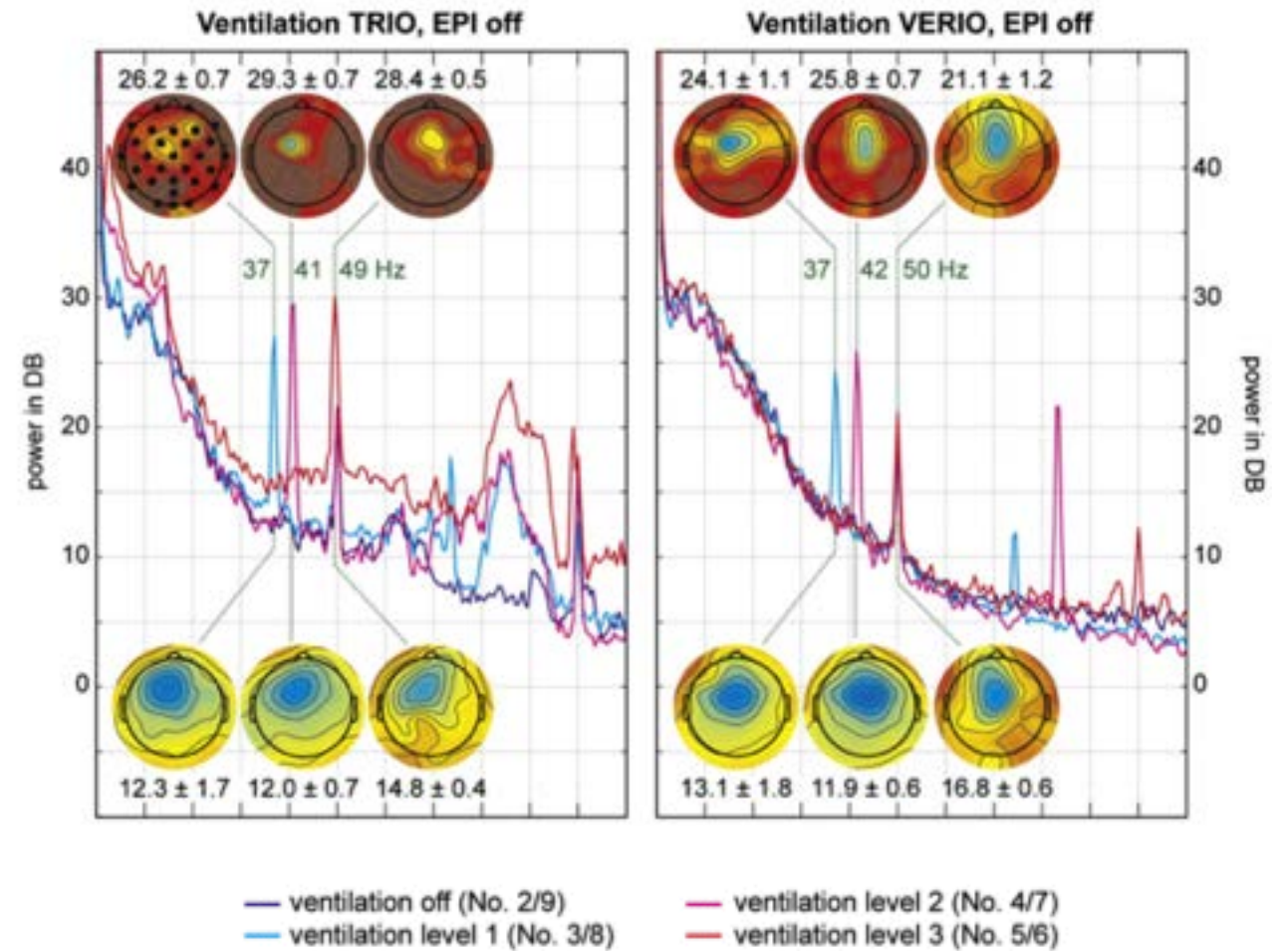
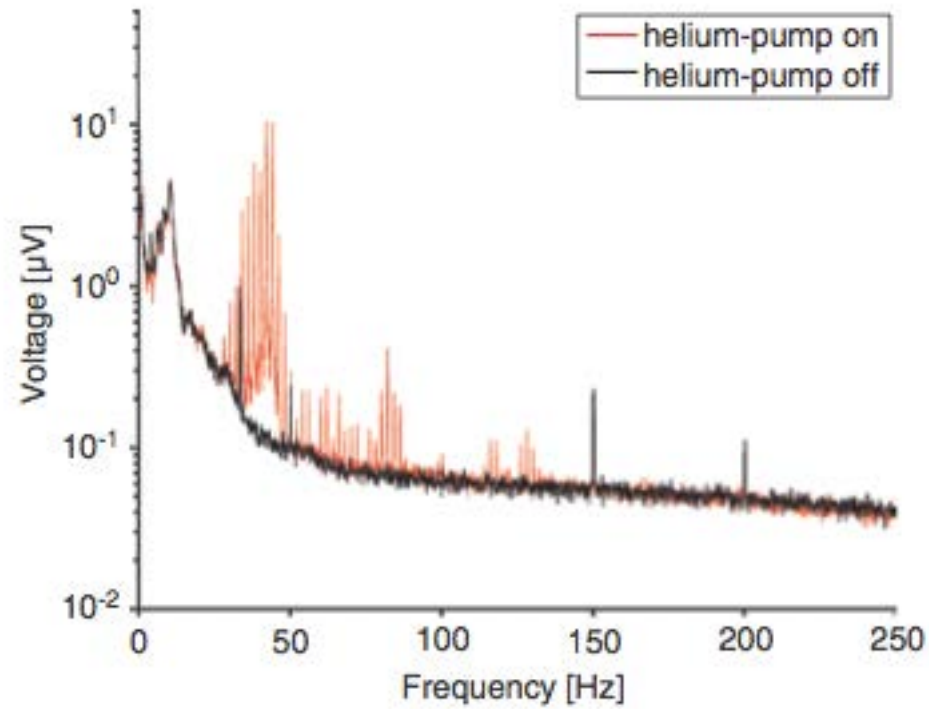






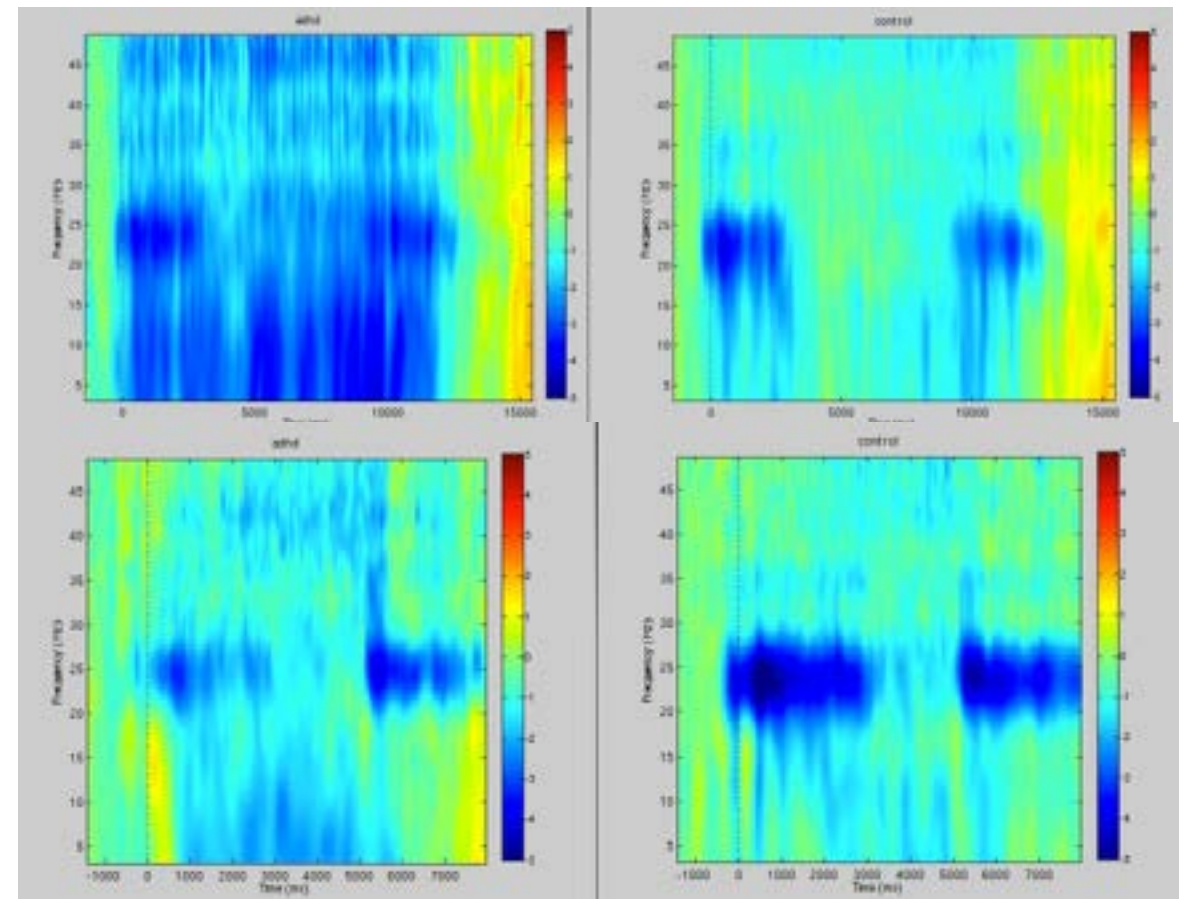
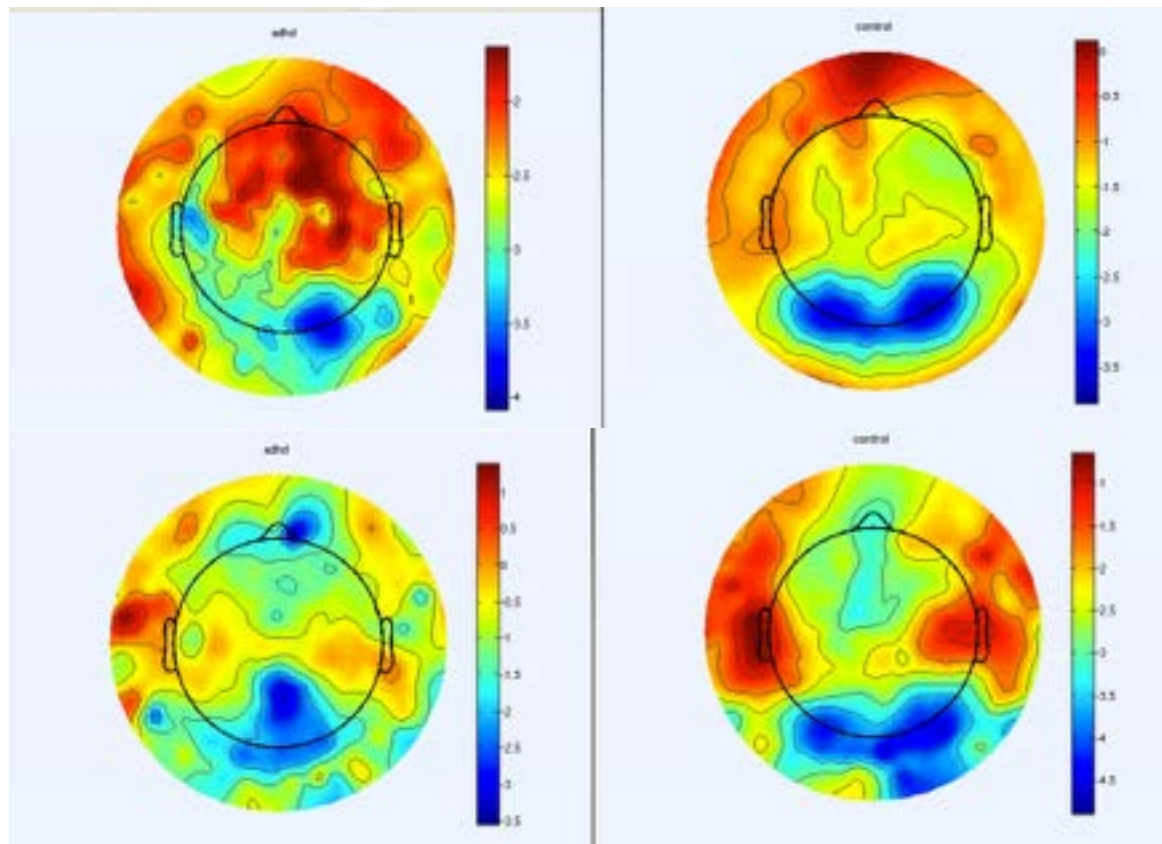


# Other issues: any motion will cause artifact





Is it the end of the world? No.  
Is it extremely infuriating and potentially compromising to getting SNR  
sufficient to ask your questions? Yes.



*after removing outlier subjects, in/out correlations in alpha  
group total,  $\rho=.65$ ,  $p<.001$  versus  $\rho=.45$  ( $p<.02$ ) before*

# WHY?

*Debener 2006*

**Table 1. Comparison of separate and simultaneous EEG–fMRI recording protocols**

Protocol feature	Separate	Simultaneous
Optimal signal quality	Yes	No
Possibility to optimize design	Yes	No

# WHY?

Debener 2006

**Table 1. Comparison of separate and simultaneous EEG–fMRI recording protocols**

Protocol feature	Separate	Simultaneous
Optimal signal quality	Yes	No
Possibility to optimize design	Yes	No
Avoidance of order effects	No	Yes
Identical sensory stimulation	No	Yes
Identical subjective experience	No	Yes
Identical behavior	No	Yes
Direct temporal correlation of EEG and fMRI signals	No	Yes

*the only reason here that is justifiable (to me) is temporal correlation of EEG and fMRI signals*

e.g., epileptic spikes, sleep events, event-related (robust) markers (like alpha)

*the only other reason that is justifiable (to me) is having two complementary but different things that you want to measure (my personal strategy)*

e.g., timescales (our current project)



# Conclusion

- \* combining data modalities is hard - high-risk and sometimes high-reward (consider that the technique has been around for 20 years, relatively little output)
- \* in reviewing papers consider the value of the scientific question, quality assurance protocol, reference conditions to validate interpretation

Questions?