Application of neural fields to EEG dynamics during general anesthesia

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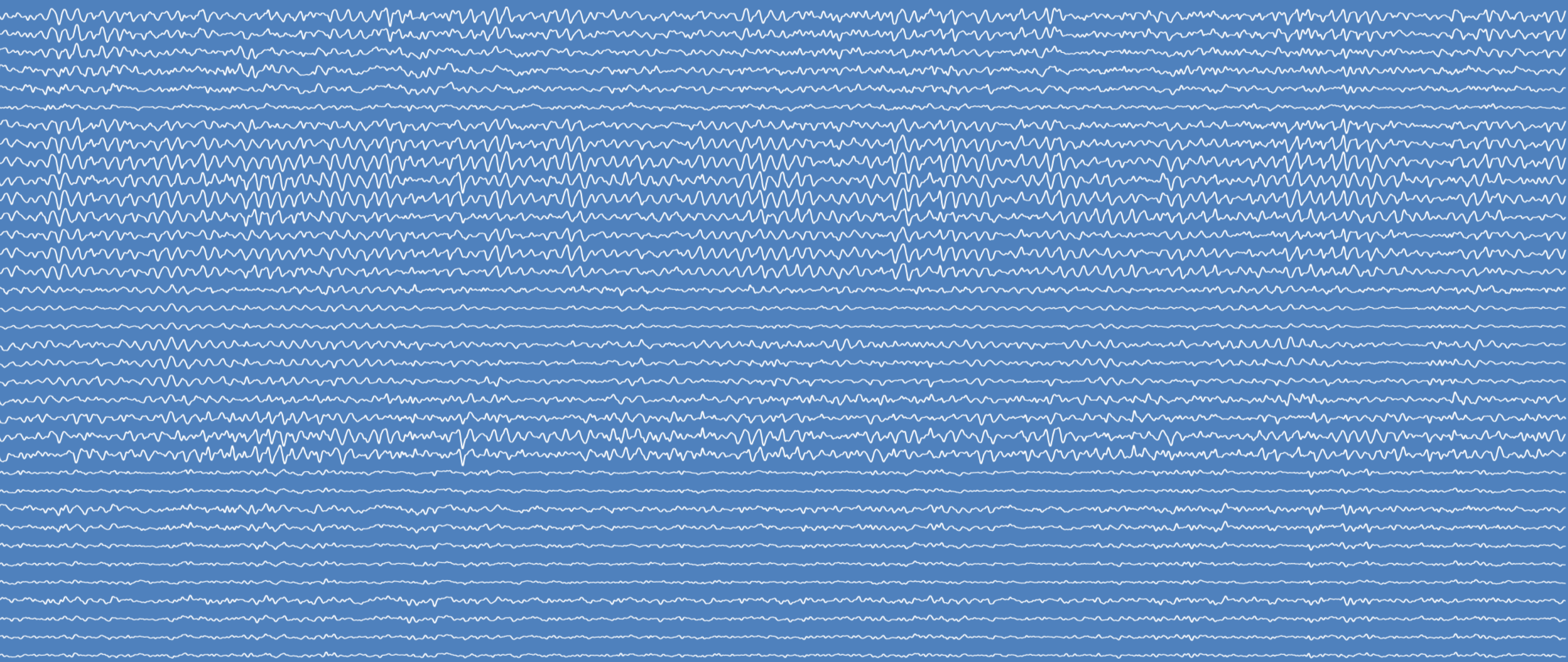

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Outline

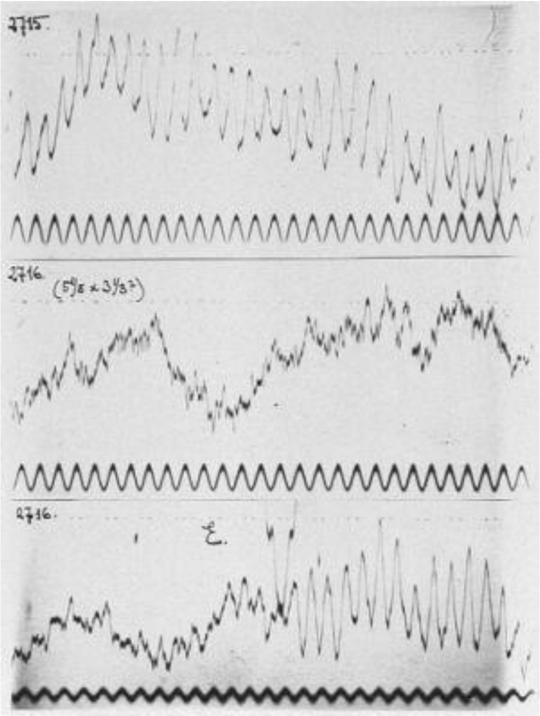
- *Background on EEG*
- Neural field theory of the EEG
- EEG dynamics during general anesthesia

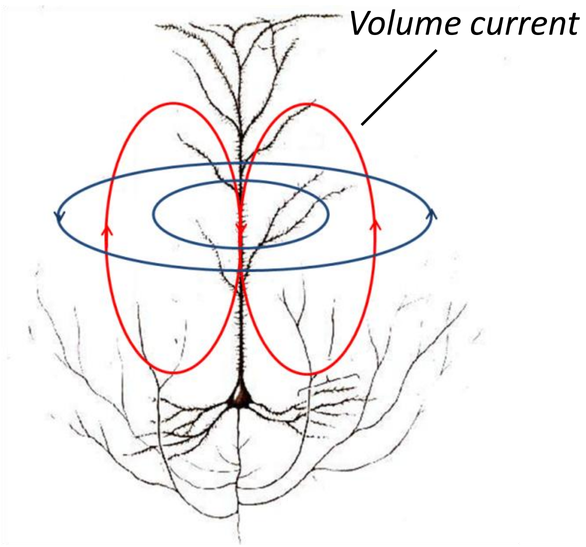
EEG recordings

- *Electro-encephalography (EEG)* refers to electrical potentials recorded from the scalp
- First recorded in the 1920s by the German psychiatrist Hans Berger
- EEG reflects the total synaptic activity of large numbers of cortical pyramidal neurons

EEG electrode cap

First EEG recordings

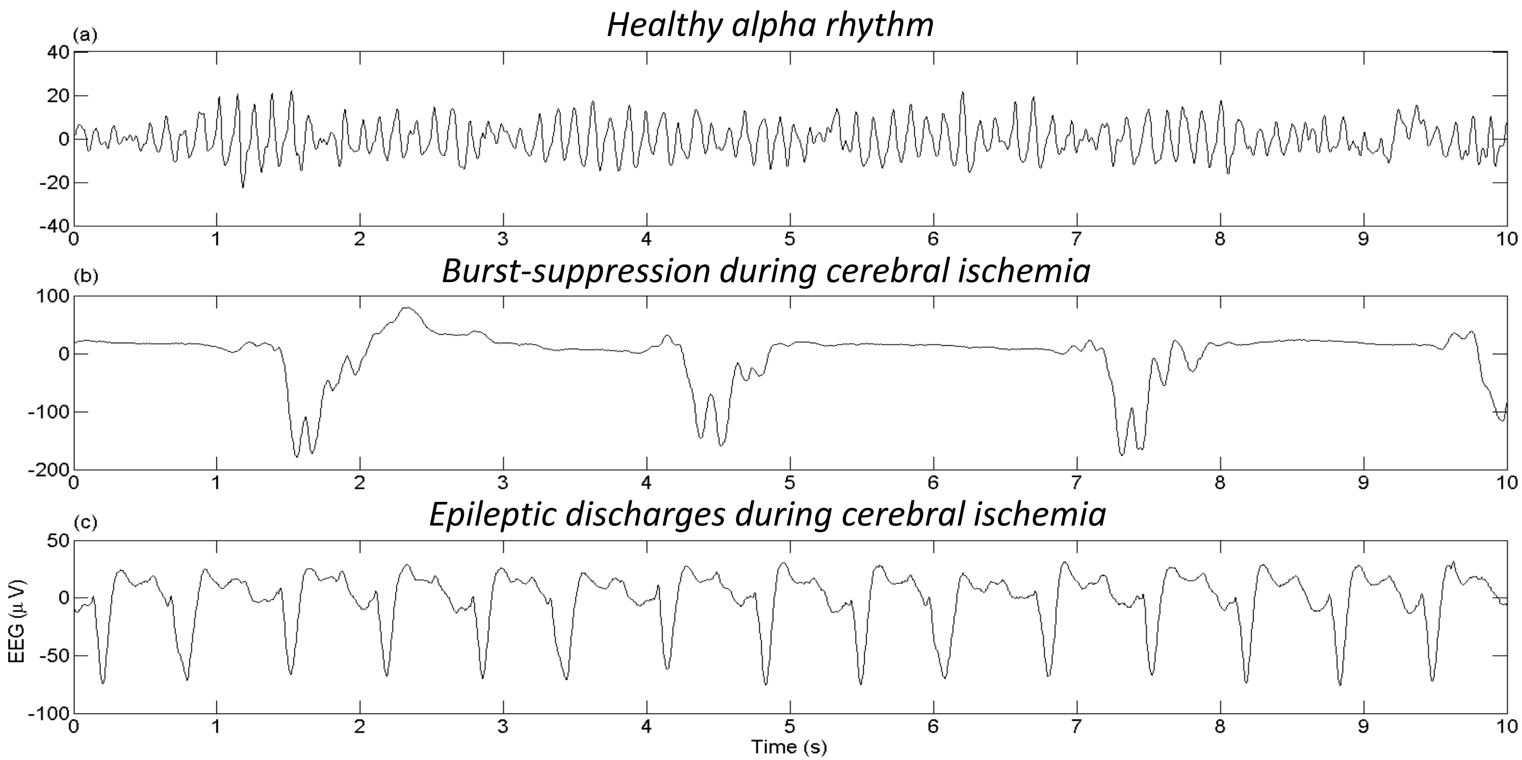




Hans Berger (1873-1941)

Varieties of EEG rhythms

- There exist a wide variety of EEG rhythms
- Well documented correlations with cognitive and perceptual processes and neurological and psychiatric syndromes
- Functions and physiological mechanisms of generation remain poorly understood



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- *Neural field theory of the EEG*
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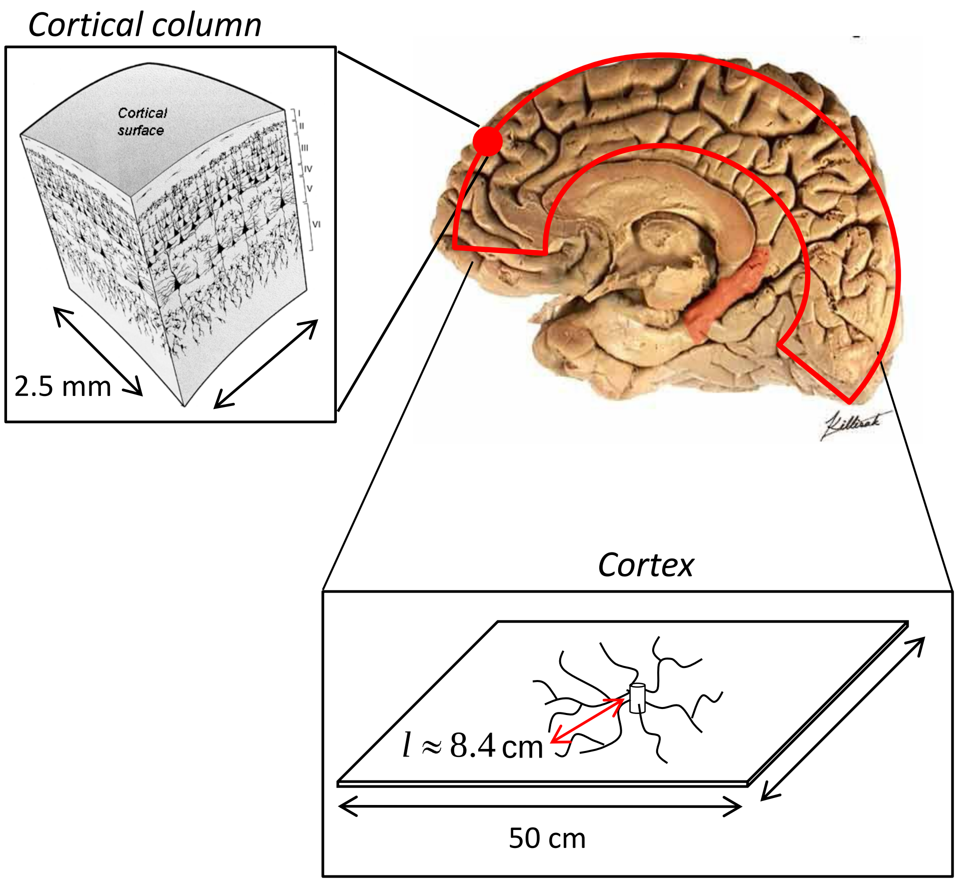
Neural fields



- Neural fields describe the *macroscopic* spatio-temporal dynamics in cortical tissue

- State-variables are *mean membrane potential* (mV) and *mean firing-rate (1/s)* within cortical columns

- **Local dynamics:**

- synaptic filtering: $V(t) = \nu h \otimes Q_{in}(t)$

- synaptic response: $h(t) = \frac{\alpha\beta}{\beta - \alpha}[e^{-\alpha t} - e^{-\beta t}]$

- neural activation: $Q(t) = S(V(t))$

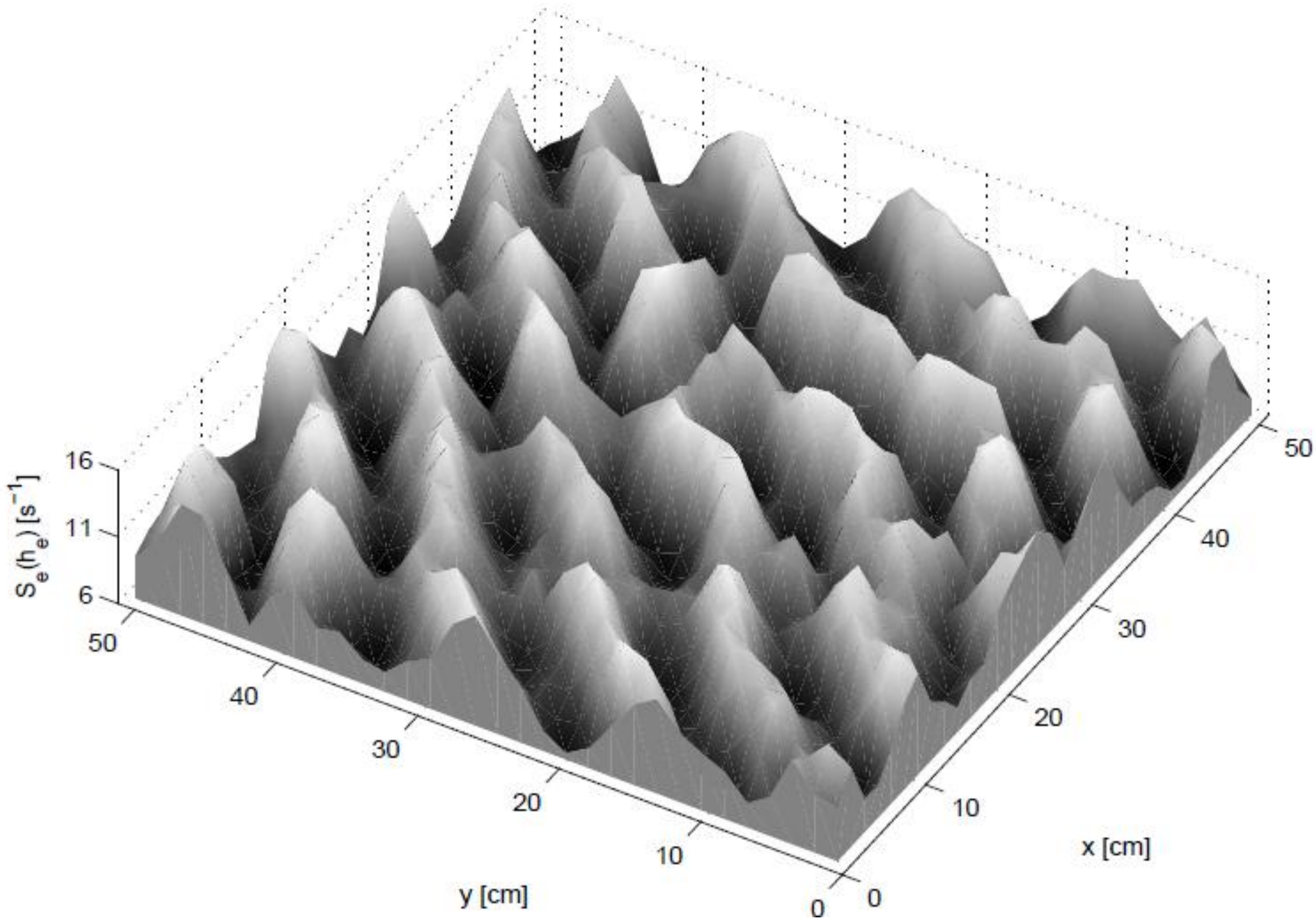
- **Global dynamics:**

- Long-range cortico-cortical projections are modeled by

$$\phi(r,t) = \int_{-\infty}^{t} \int_{cortex} G(t,t',r,r')Q(t',r')dr'dt'$$

-Typically one assumes isotropy and constant conduction velocity: $G(||r - r'||, t - \frac{||r - r'||}{v})$

Simulation of alpha rhythm



Bojak, I., Neurocomputing 2004

Beyond cortical chauvinism

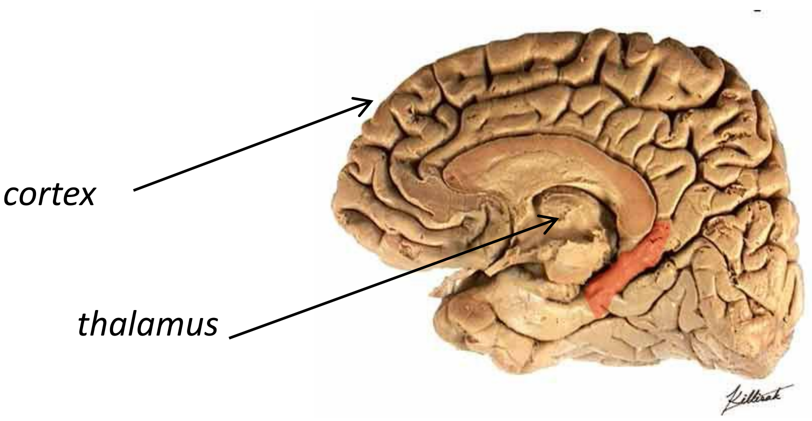
- Most field theories of large-scale brain dynamics (EEG) treat the cortex as an anatomically isolated structure (Liley, 2002)

- However, cortex is densely and reciprocally connected to the thalamus

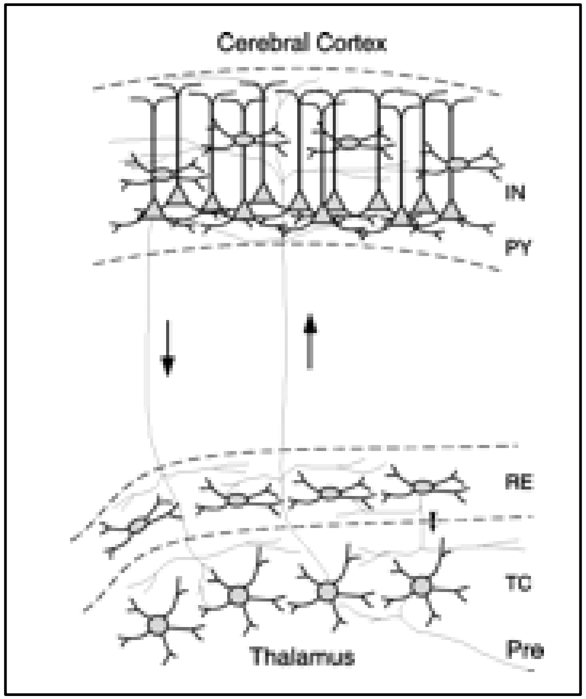
- In (Robinson, 2001; Rennie, 2002) a new direction was initiated by developing a thalamo-cortical field theory of the EEG

- Besides spontaneous EEG rhythms, the model can reproduce sleep spindles, evoked responses, and generalized seizures (Robinson, 2001; Rennie, 2002)

- Propofol targets subcortical structures and imaging studies (PET and fMRI) suggest that the thalamus is involved in anesthesia-induced functional changes in cortex



Thalamo-cortical connectivity



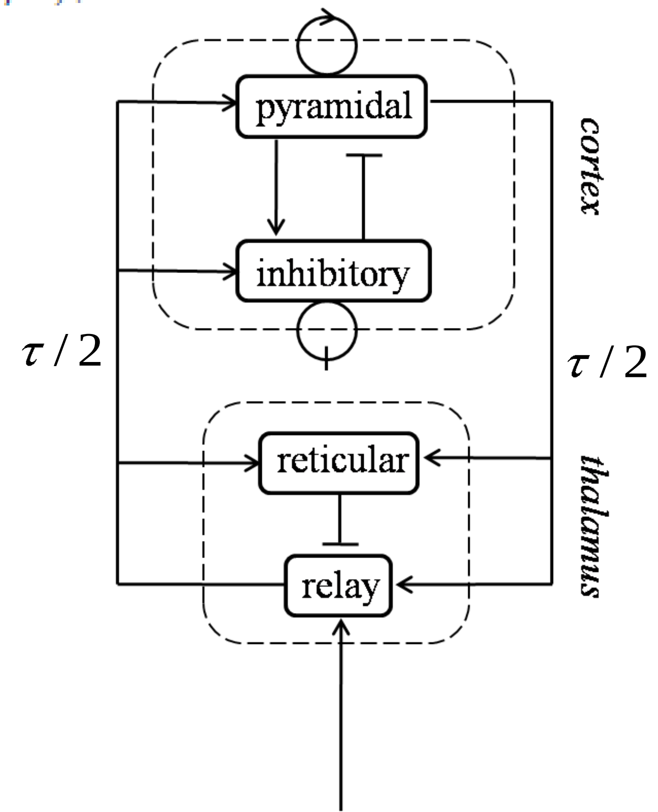
Thalamo-cortical field equations

$$
\begin{aligned}
V_e(x,t) &= \bar{h} \otimes v_{ee}\phi_e(x,t) + \bar{h} \otimes v_{es}S(V_s(x,t-\tau/2)) + \bar{h} \otimes v_{ei}S(V_i(x,t)), \\
V_i(x,t) &= \bar{h} \otimes v_{ie}\phi_e(x,t) + \bar{h} \otimes v_{is}S(V_s(x,t-\tau/2)) + \bar{h} \otimes v_{ii}S(V_i(x,t)), \\
V_s(x,t) &= \bar{h} \otimes v_{sn}\phi_n(x,t) + \bar{h} \otimes v_{se}\phi_e(x,t-\tau/2) + \bar{h} \otimes v_{sr}S(V_r(x,t)), \\
V_r(x,t) &= \bar{h} \otimes v_{rs}S(V_s(x,t)) + \bar{h} \otimes v_{re}\phi_e(x,t-\tau/2), \\
D(x,t)\phi_e &= S(V_e),
\end{aligned}
$$

where D is the wave operator

$$D = \left(\frac{1}{\gamma}\frac{\partial}{\partial t} + 1\right)^2 - l^2\nabla^2$$

with $\gamma = v/l$ the cortical damping rate (1/s)



- Approximately, the EEG signal is proportional to ϕ_e

Linearization and EEG spectra

- The transfer function from ϕ_n to ϕ_e is given by

$$\frac{\phi_e}{\phi_n} = \frac{\zeta_{eisn} + (1-\zeta_{ii})\zeta_{esn}}{[(D-\zeta_{ee})(1-\zeta_{srs})-\zeta_{ese}-\zeta_{esre}](1-\zeta_{ii})-\zeta_{eie}(1-\zeta_{srs})-\zeta_{eise}-\zeta_{eisre}}$$

Laplace transform of wave operator (k = (0,0))

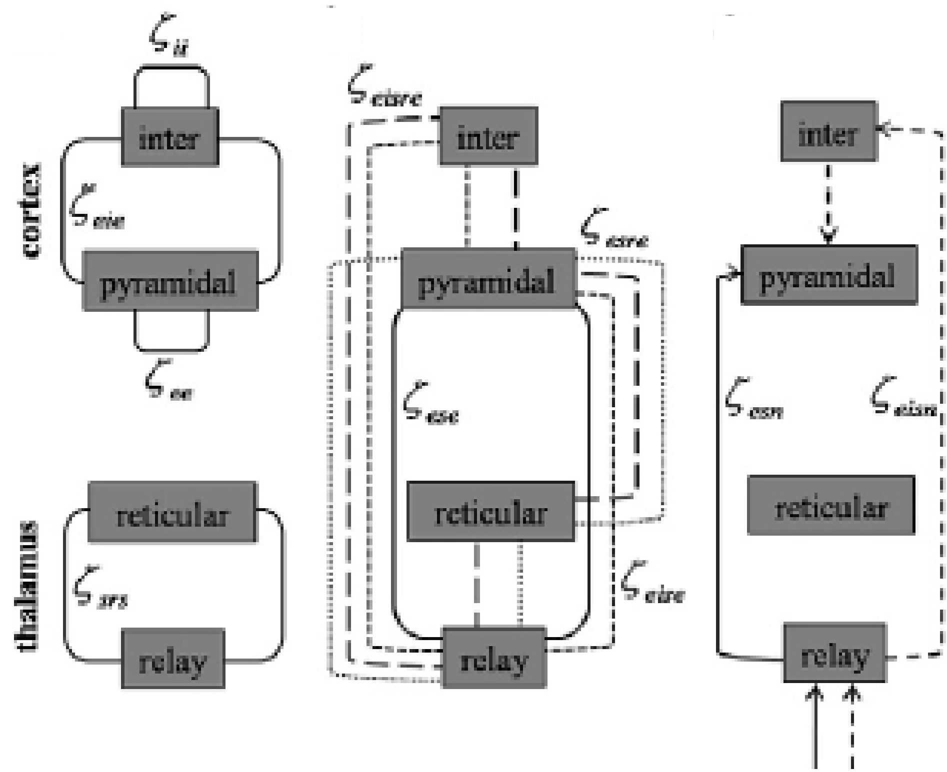
frequency response in the anatomical loop e -> s -> e

- For ϕ_n white noise:

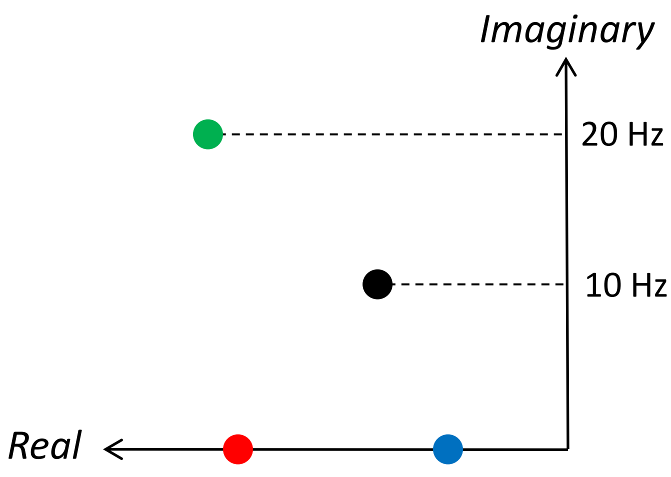
$$\phi_n = \langle \phi_n \rangle + \sigma_n \xi(t)$$

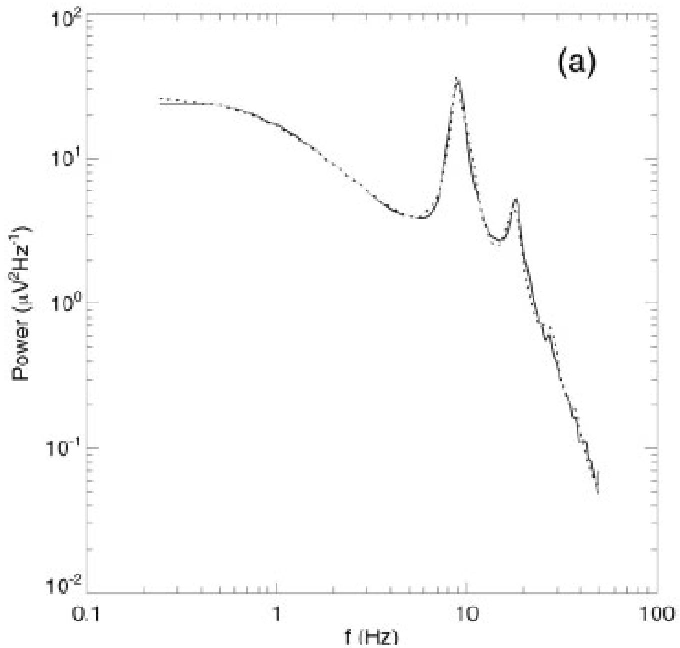
the EEG power spectrum is given by

$$P(\omega) = \sigma_n^2 |\phi_e(\omega)|^2$$



Resonances underlying EEG spectra





(Robinson, P. et al., Human Brain Mapping 2004)

The EEG power spectrum during wakefulness is dominated by the following resonances:

● ● - ***a pair of non-oscillatory resonances*** (underlying the power-law decay of EEG spectra)

● - ***alpha resonance*** (underlying the spontaneous alpha rhythm)

● - ***beta resonance*** (underlying the first harmonic of the alpha rhythm)

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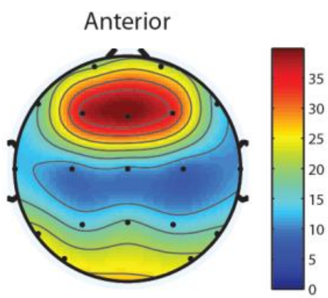
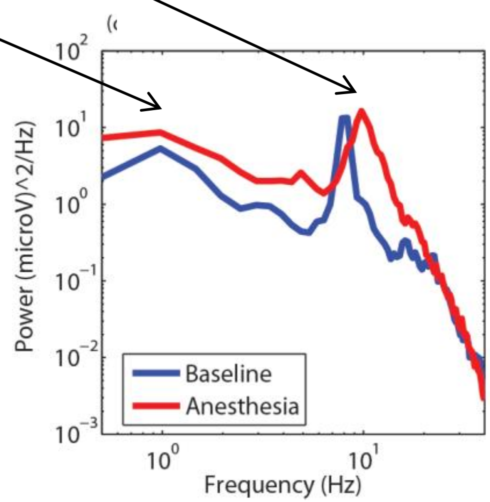
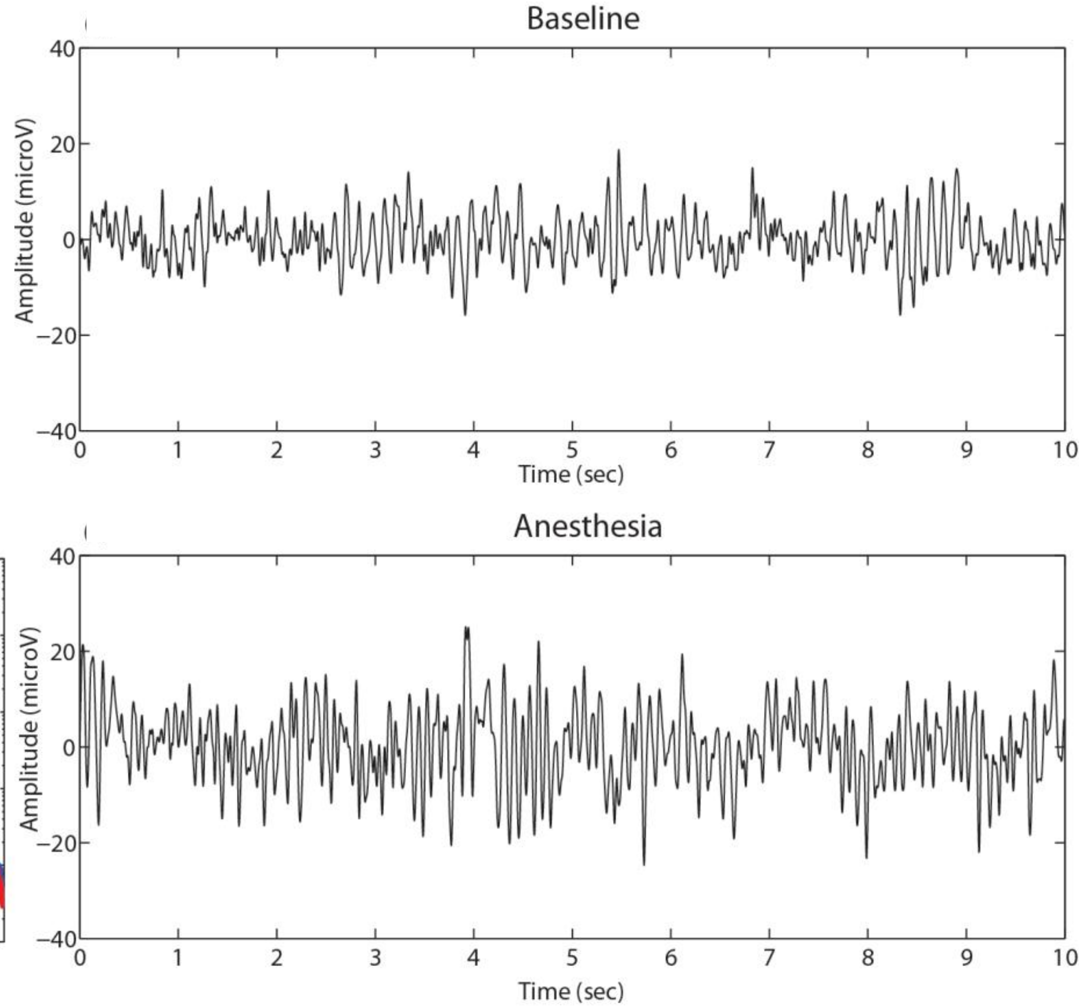
EEG phenomenology



Pronounced frontal alpha oscillations (7 – 13 Hz) with increased peak-frequency

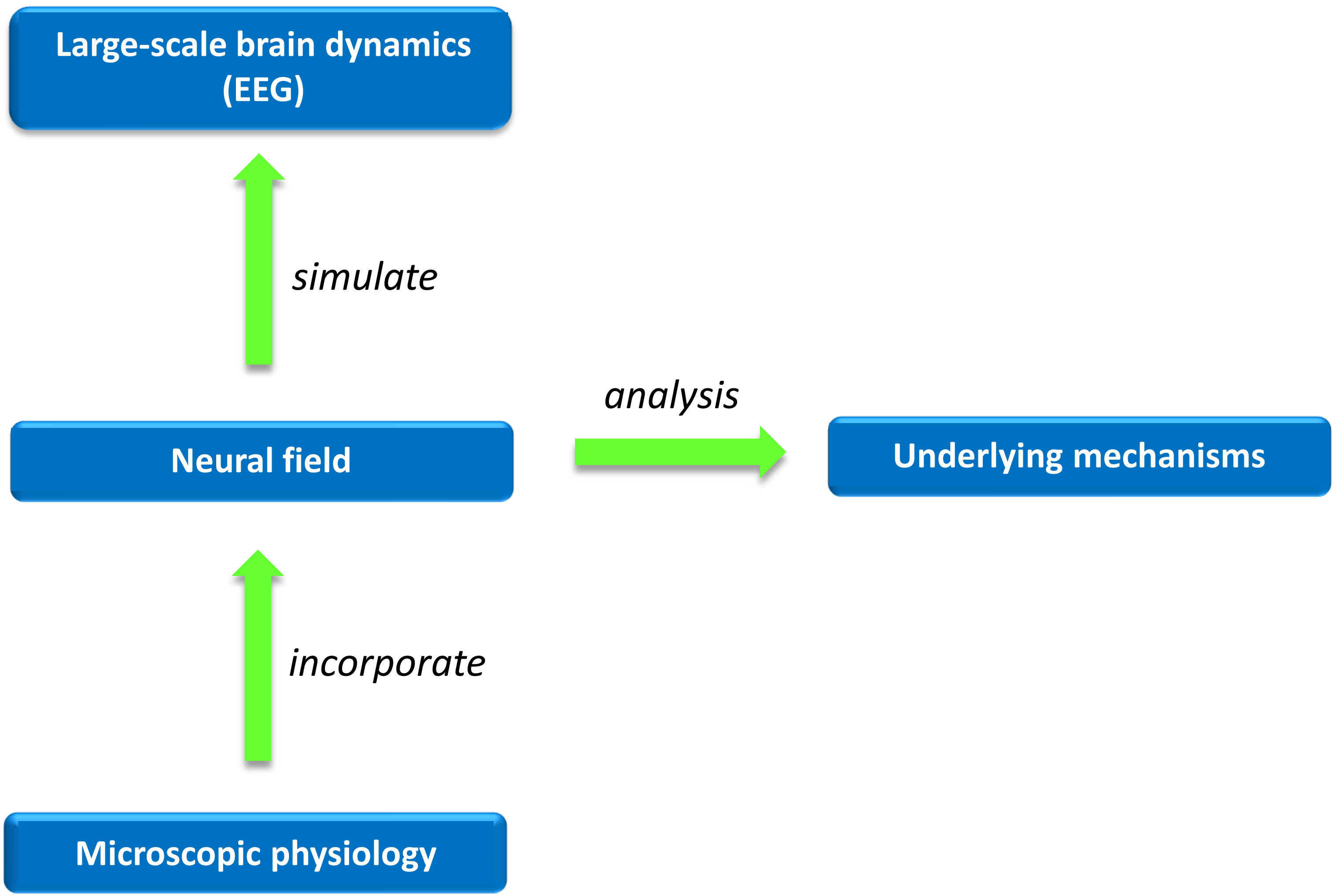
Increased delta (0.5 – 3 Hz) and t heta (3 – 7 Hz) power





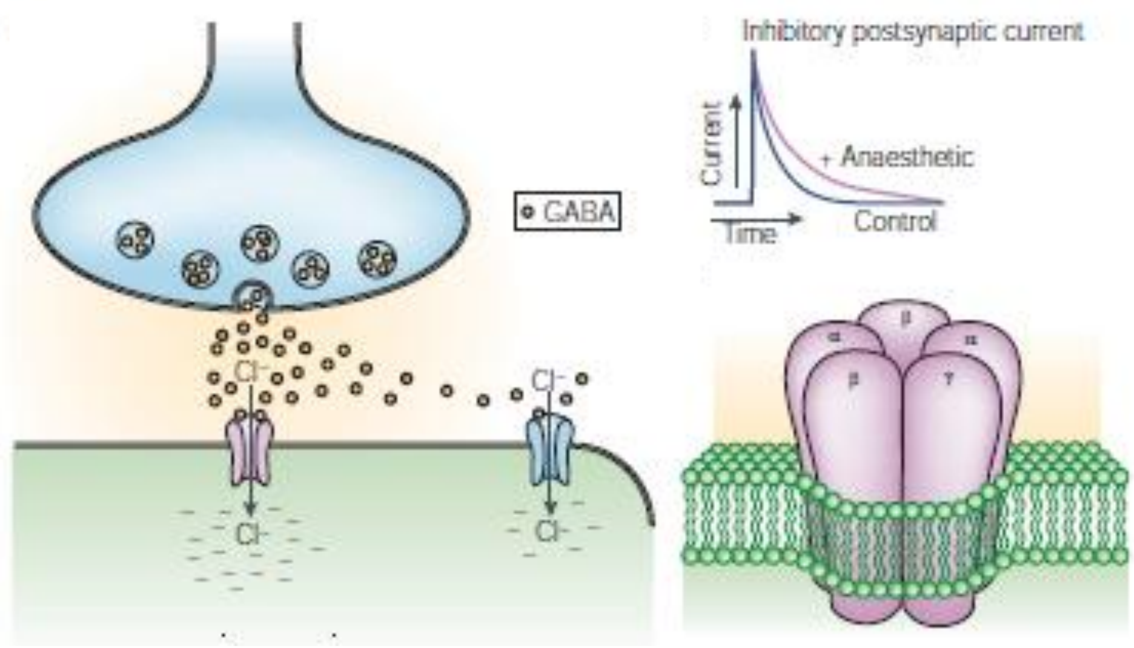
Hindriks, R., and van Putten, M.J.A.M., NeuroImage 2012

Crossing scales



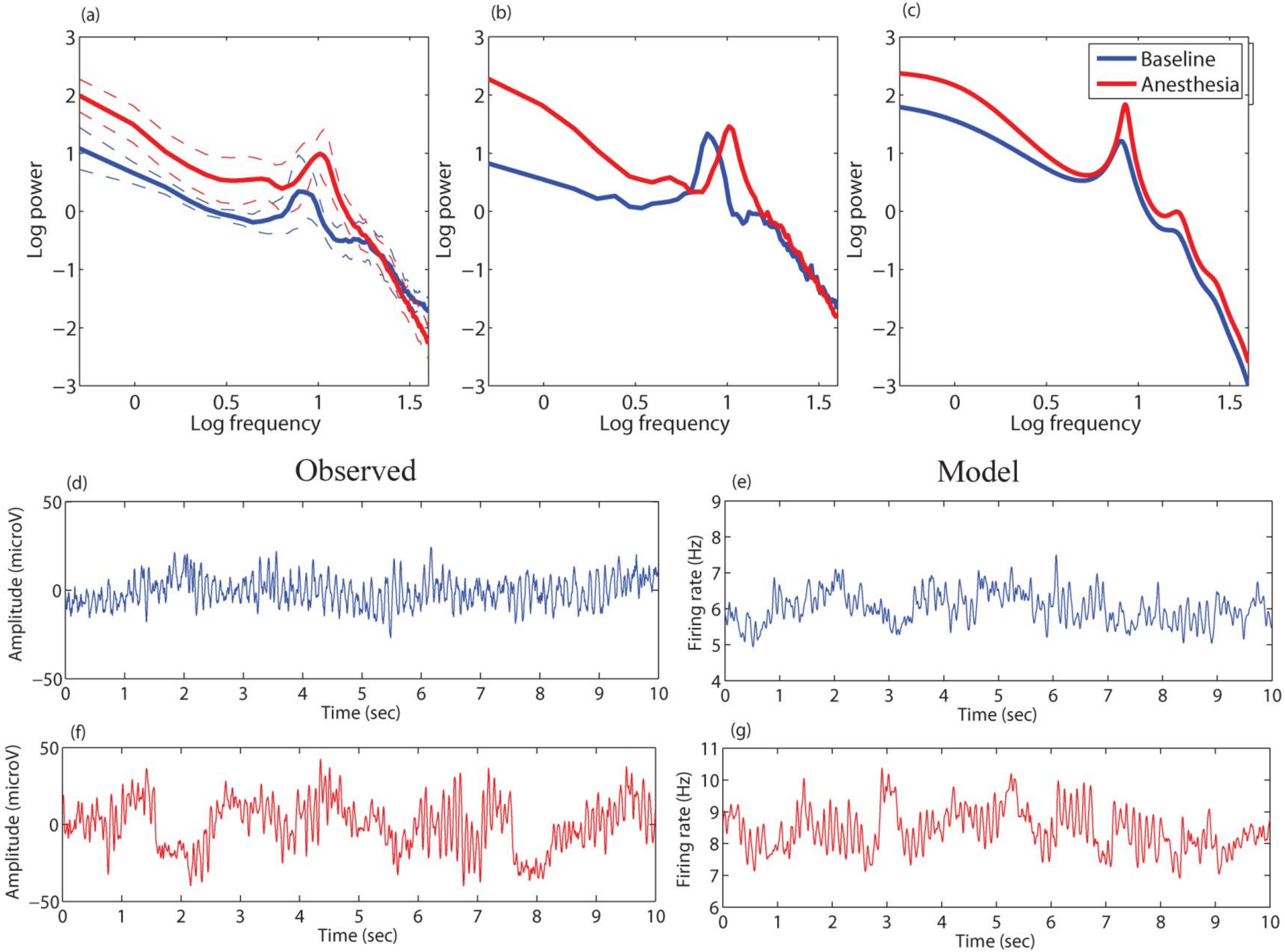
The action of propofol

- Propofol pre-dominantly targets GABA_A receptors
- It increases the time-constant of receptor de-activation
- About a dozen GABA_A receptor subtypes
- Differential affinity is not completely known



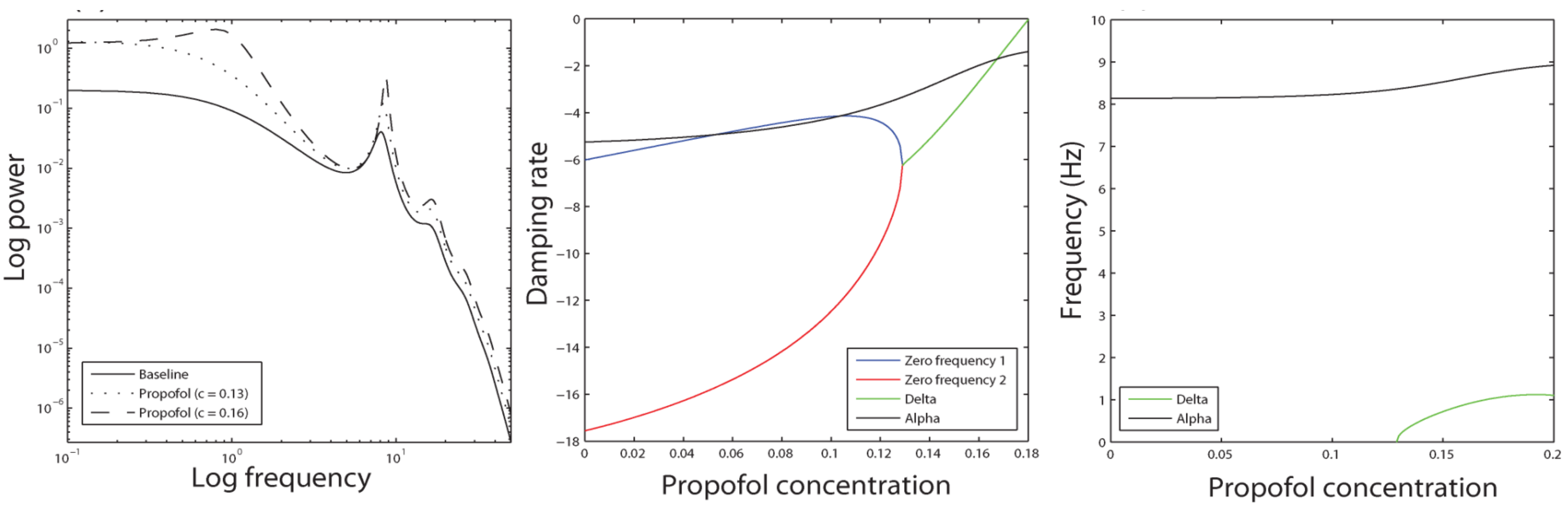
Rudolph, U. and Antkowiak, B., Nature Neuroscience Reviews, 2004

Reproduction of experimental observations

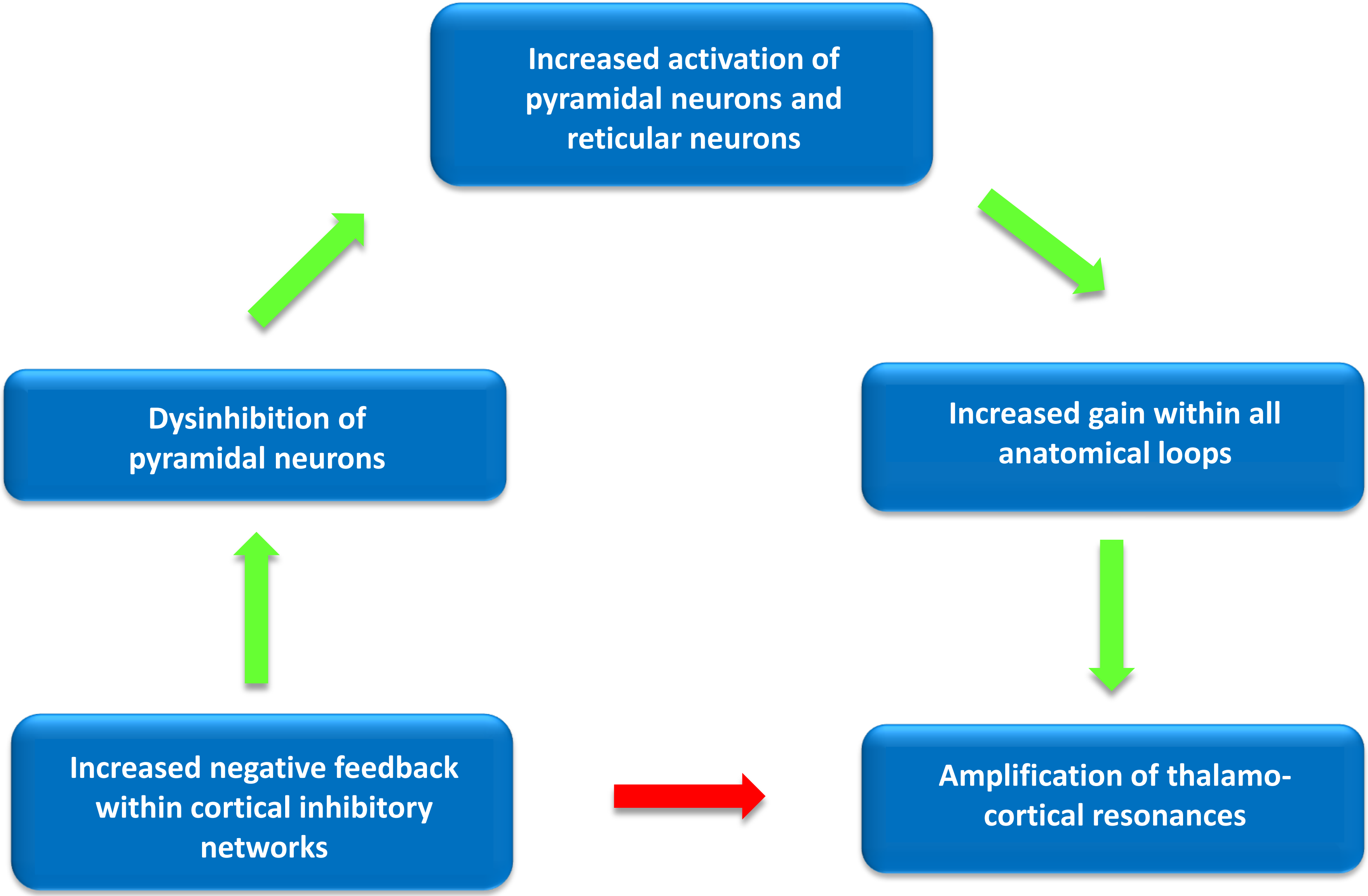


Modulation of resonances

- Pronounced EEG alpha (7-13 Hz) oscillations are caused by amplification of the alpha resonance
- Increased EEG delta power (0.5-3 Hz) is caused by the net amplification of the pair of non-oscillatory resonances
- Increased EEG theta power (3-7 Hz) is caused by spectral leakage



Underlying mechanisms



Model predictions

- *Propofol has a higher affinity for receptors on interneurons than for receptors on pyramidal neurons*
- *Selective administration of propofol to cortical tissue leads to the observed EEG phenomenology*
- *Increased firing-rates of pyramidal neurons in frontal cortex and neurons within reticular nuclei*
- *Increased synchrony of alpha oscillations between frontal cortex and thalamic nuclei*