C83MAB: Mind and Brain

Neuroscience of Consciousness I

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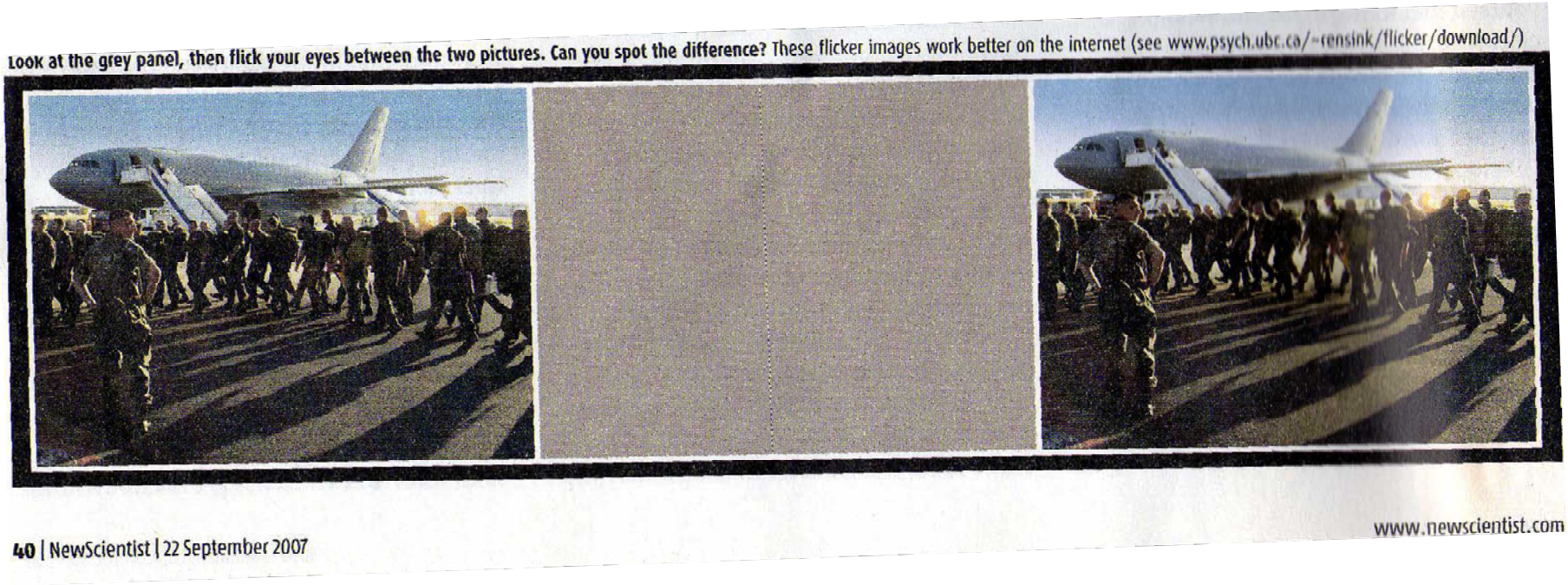
UNITED KINGDOM · CHINA · MALAYSIA

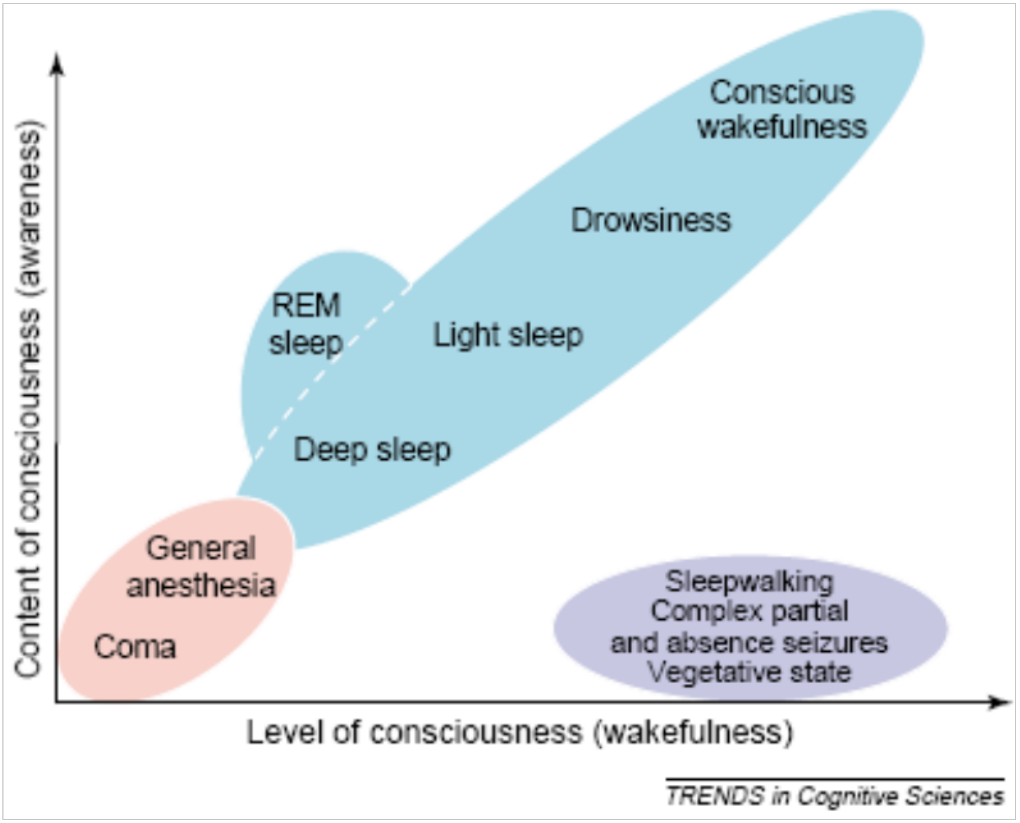
What is consciousness?



Consciousness

- **State of consciousness - Being awake/alert/attentive/responsive**

- **Contents of consciousness ('consciousness of . . .') – particular (reportable) experience at a certain level of consciousness**



Laureys (2005) Trends in Cogn. Sci. 9:556-559

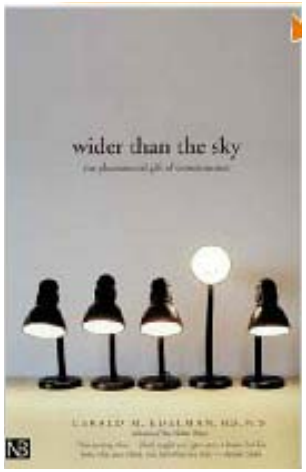
Does consciousness matter to you?

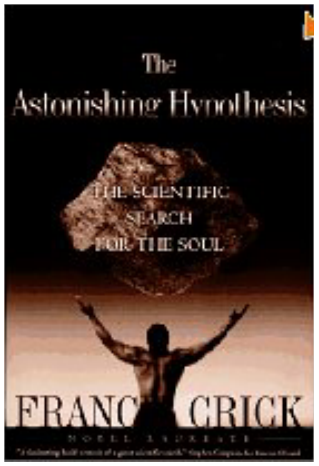
1)Yes, very much! Consciousness is central to my life!

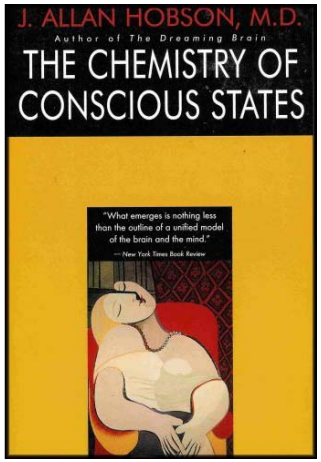
2) No, I can’t see what difference consciousness makes to my life!

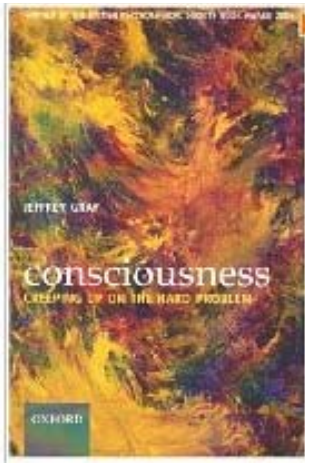
Great public interest in the brain substrates of consciousness . . .

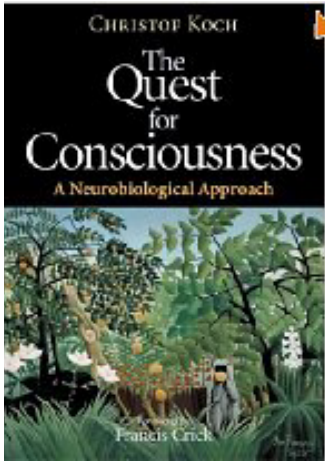
. . . reflected by an abundance of popular-science books on the topic.

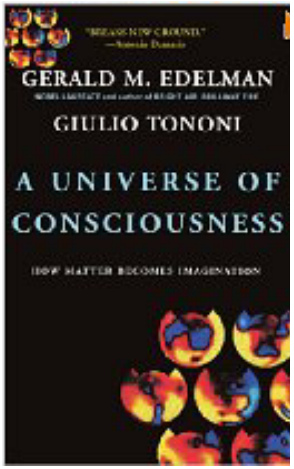












Etc.

Problems of consciousness – what is to explain?

- Difference between wakefulness and sleep
- Difference between being responsive and unresponsive
- Reportability of our mental states
- Integration of information
- Ability to access own internal states
- Focus of attention
- Deliberate control of behaviour (as opposed to automatic behaviour)

'Easy' problems

The 'hard' problem:

- Subjective experience ('phenomenal consciousness', 'qualia')

Chalmers (1995) "Facing up to the problem of consciousness" Journal of Consciousness Studies 2:200-219
http://consc.net/papers/facing.pdf

Today

- States of consciousness and neural correlates
- Neurological disorders of (the states of) consciousness

Next week

- Contents of consciousness and neural correlates
- Neurological disorders affecting contents of consciousness
- What's left to explain?

States of consciousness

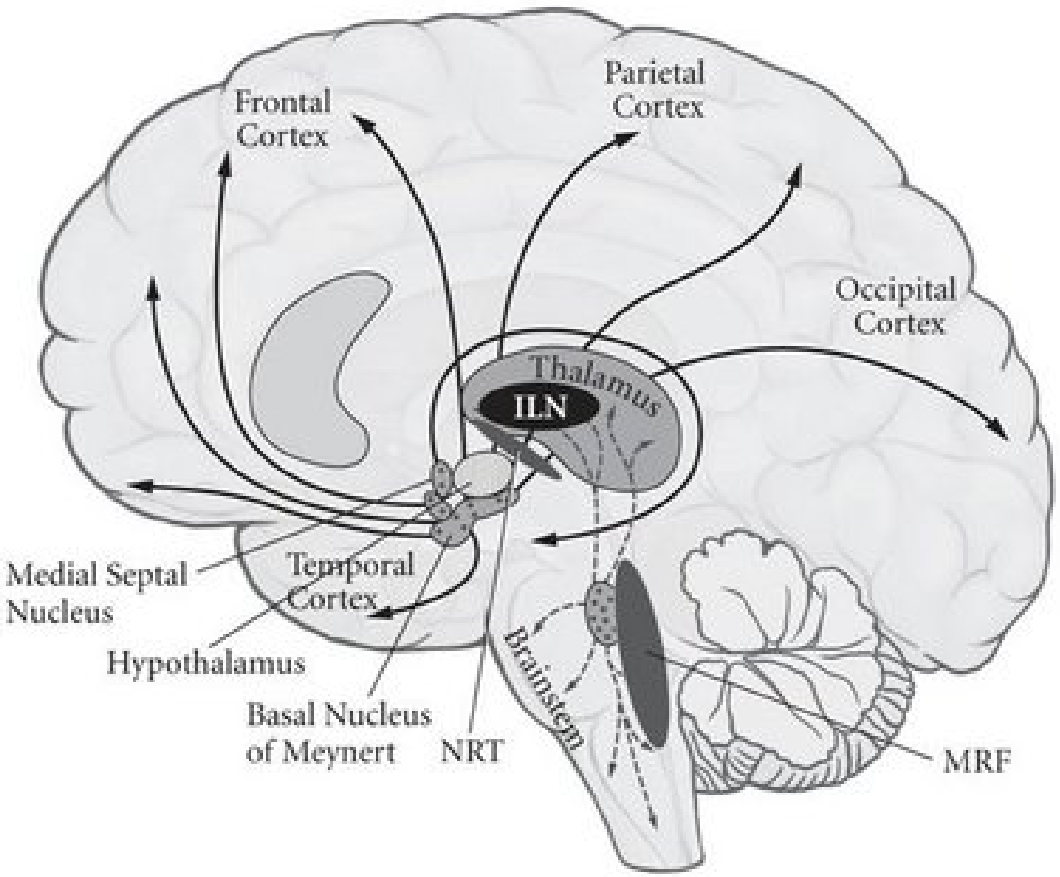
Table 1. Major properties of four types of unconscious state compared with conscious rest

State	Conscious resting state [19,20]	Deep sleep [22]	General anesthesia [24]	Vegetative state or coma [25]	Epileptic loss of consciousness [23]
Cause	Neuromodulation of the cortex by the brainstem, instructions to avoid deliberate tasks [26]	Physiological: neuromodulation of the forebrain by the brainstem	Pharmacological: a variety of chemical agents	Pathological: trauma, intoxication, anoxia, hypoglycemia	Pathological: slow, synchronized neuronal firing driven by brain foci [23]
Behavioral signs	Accurate reportability of attended stimuli; orientation to space, time, and self; visual images, inner speech, abstract thoughts; control of voluntary muscles	No reportability	No reportability	No reportability. Lower brainstem reflexes retained intact [25]	No reportability
Regional metabolism	High in frontoparietal cortex	Low in frontoparietal cortex [27]	Low in frontoparietal cortex [28]	Low in frontoparietal cortex	Low in frontoparietal cortex
EEG voltages	Low-amplitude, irregular high-frequency waves (8–100 Hz), or low-amplitude, regular alpha waves (8–12 Hz)	High-amplitude, regular, low-frequency waves (<4 Hz)	High-amplitude, regular, low-frequency waves	High-amplitude, regular, low-frequency waves	High-amplitude, spike-wave form, regular, low-frequency waves
Underlying neuronal mechanism (in cortical and thalamic neurons)	Firing irregularly at an average base rate of ~10 Hz	Slow, synchronized pausing of base-rate firing [22]	Slow, synchronous pausing of base-rate firing?	Slow, synchronous pausing of base-rate firing?	Slow, synchronous pausing of base-rate firing?
Functional connectivity	High and variable	Low between cortical regions, and between thalamus and cortex [22]	Low between cortical regions, and between thalamus and cortex	Low or absent between cortical regions, and between thalamus and cortex [29,30]	Low between cortical regions, and between thalamus and cortex [23]

Abbreviations: EEG, electroencephalogram.

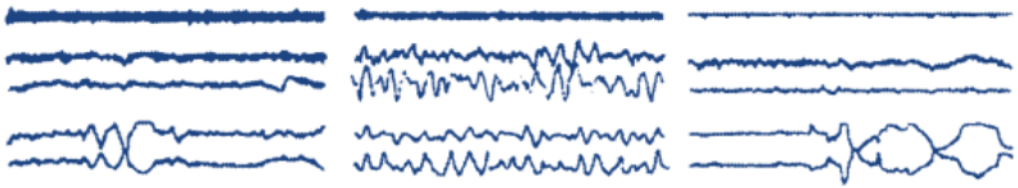
Baars et al. (2003) Trends Neurosci. 26:671-675

Key brain substrates of consciousness



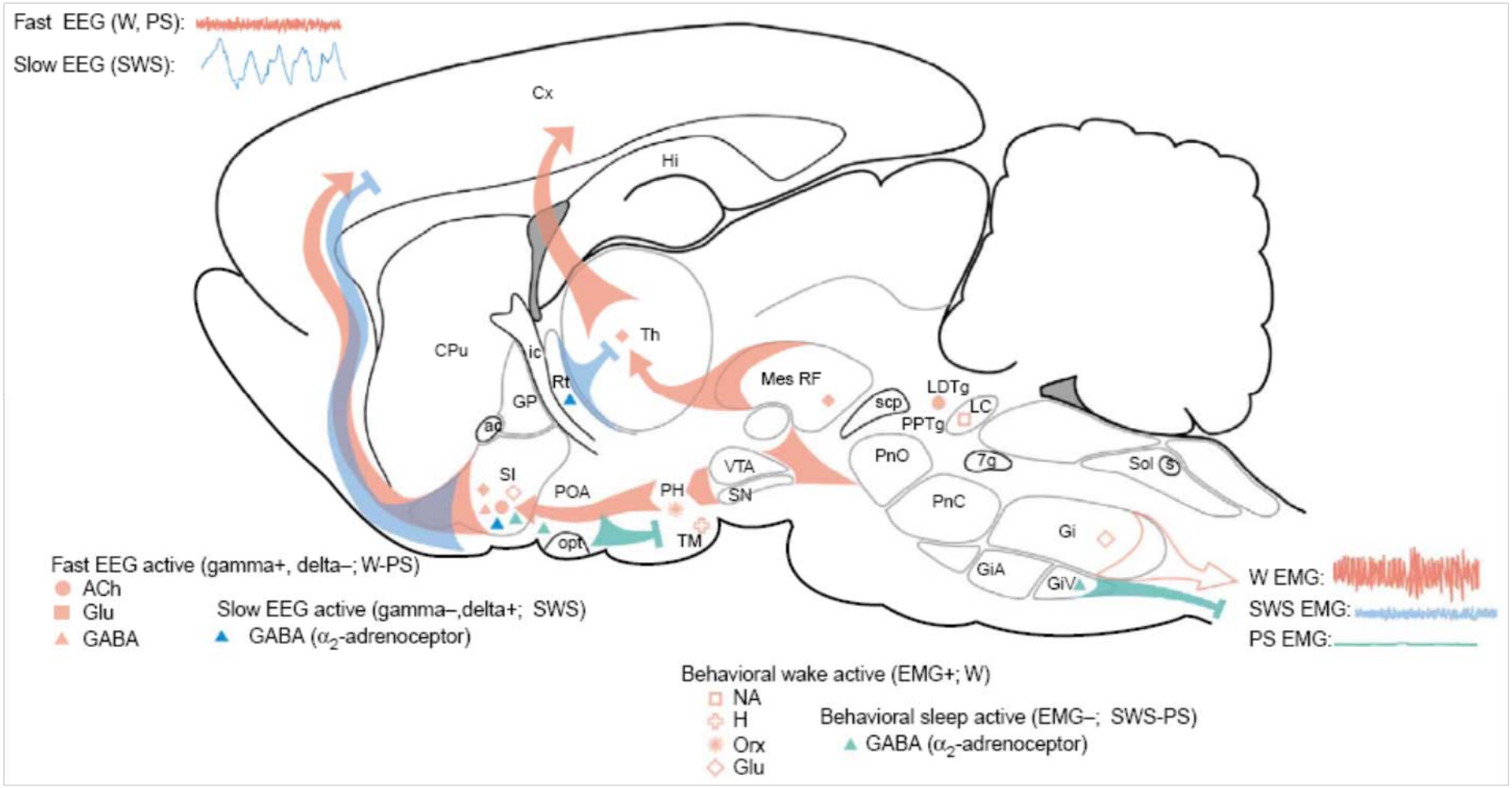
Mormann & Koch (2007) Scholarpedia 2(12):1740
http://www.scholarpedia.org/article/Neural_correlates_of_consciousness

Wakefulness and sleep - the cardinal states of consciousness

Behavioural state		Wake	Sleep: NREM	Sleep: REM
Cognitive consequences		Acquisition of information	Iteration of informatiion	Integration of information
Conscious experience	Sensation and perception	Vivid, externally generated	Dull or absent	Vivid, internally generated
	Thought	Logical progressive	Logical perseverative	Illogical bizarre
	Movement	Continuous voluntary	Episodic involuntary	Commanded but inhibited
Surface recordings	EMG			
	EEG			
	EOG			

Hobson & Pace-Schott (2002) Nature Rev Neurosci 3:679-693

Neurochemistry of wakefulness and sleep

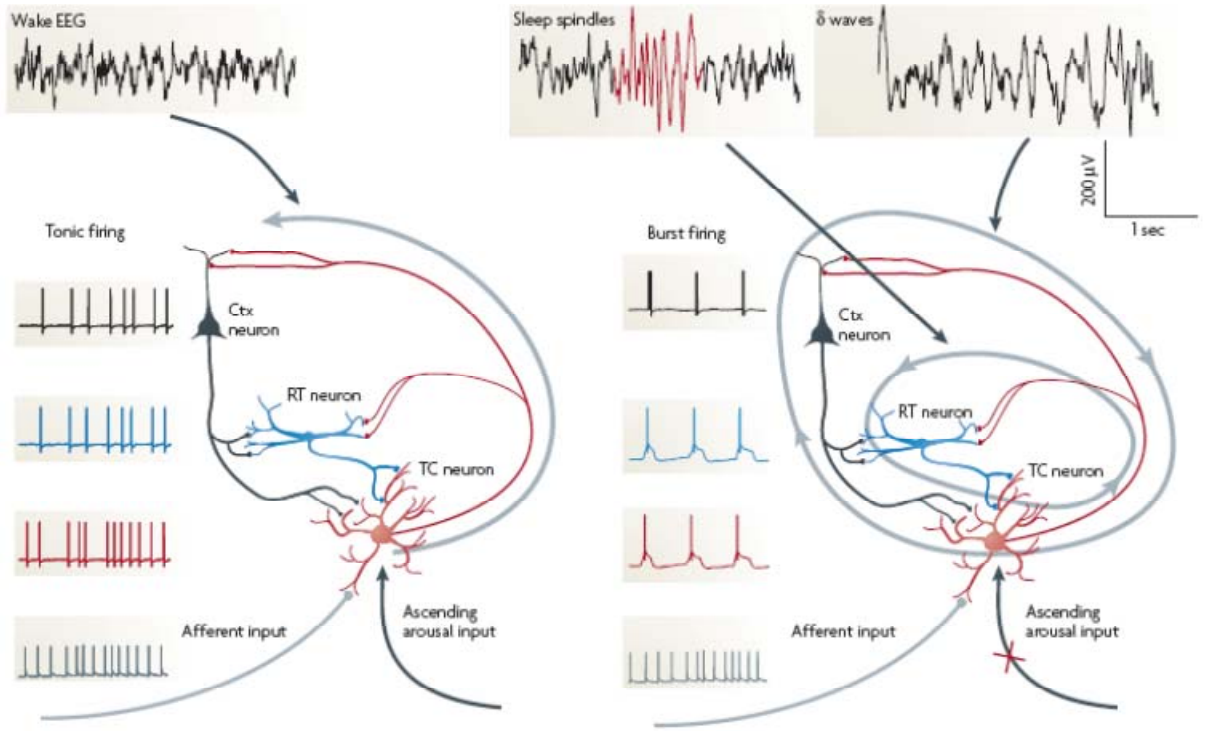


Jones (2005) Trends Pharmacol. Sci. 26:578-586

Cortico-thalamic mechanisms in wakefulness and sleep

Falling asleep: Reticular thalamic nucleus neurons fire rhythmic bursts - reflected by EEG spindles - which inhibits thalamo-cortical neurons, thereby blocking information transfer to cortex.

Awake: Cortical and thalamic neurons show tonic single-spike firing and afferent information is transferred to cortex.
EEG is largely desynchronised, but fast oscillations occur.



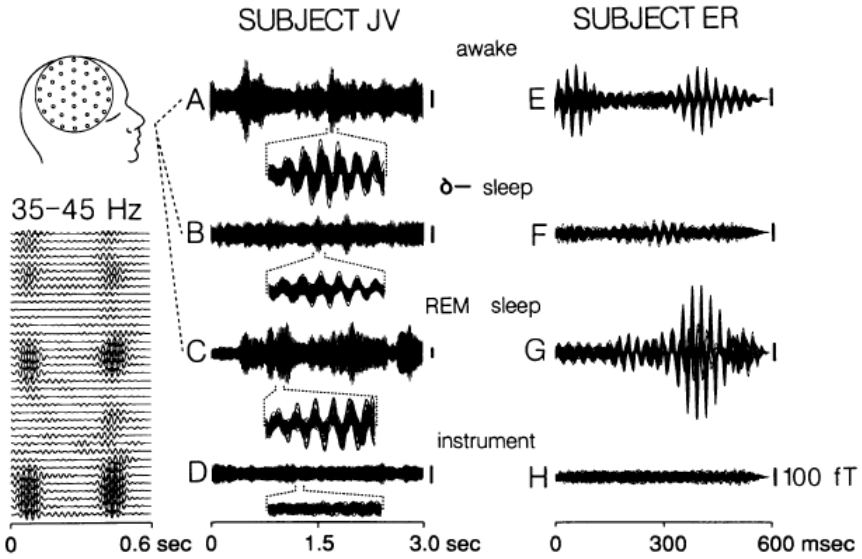
Sleep (non-REM/slow-wave): cortical and thalamic neurons show rhythmic slow changes between down and up states - reflected by slow EEG oscillations.

Waking up: Cholinergic neurons inhibit neurons in reticular thalamic nucleus and activate thalamo-cortical neurons – reflected by the replacement of slow oscillations by faster EEG oscillations –, thereby facilitating information transfer to cortex.

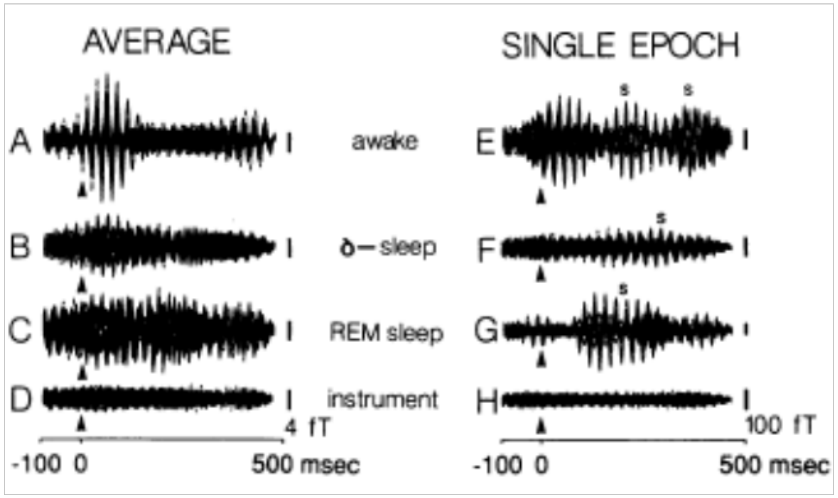
Figure from Franks (2008) Nature Rev. Neurosci. 9:370-386;
for detailed explanation see: Steriade (2003) Front. Biosci. 8:d878-899

Comparison of wakefulness and dreams: 40-Hz oscillations characterise conscious states

Spontaneous 40-Hz oscillations during wakefulness and REM sleep, but not slow-wave sleep



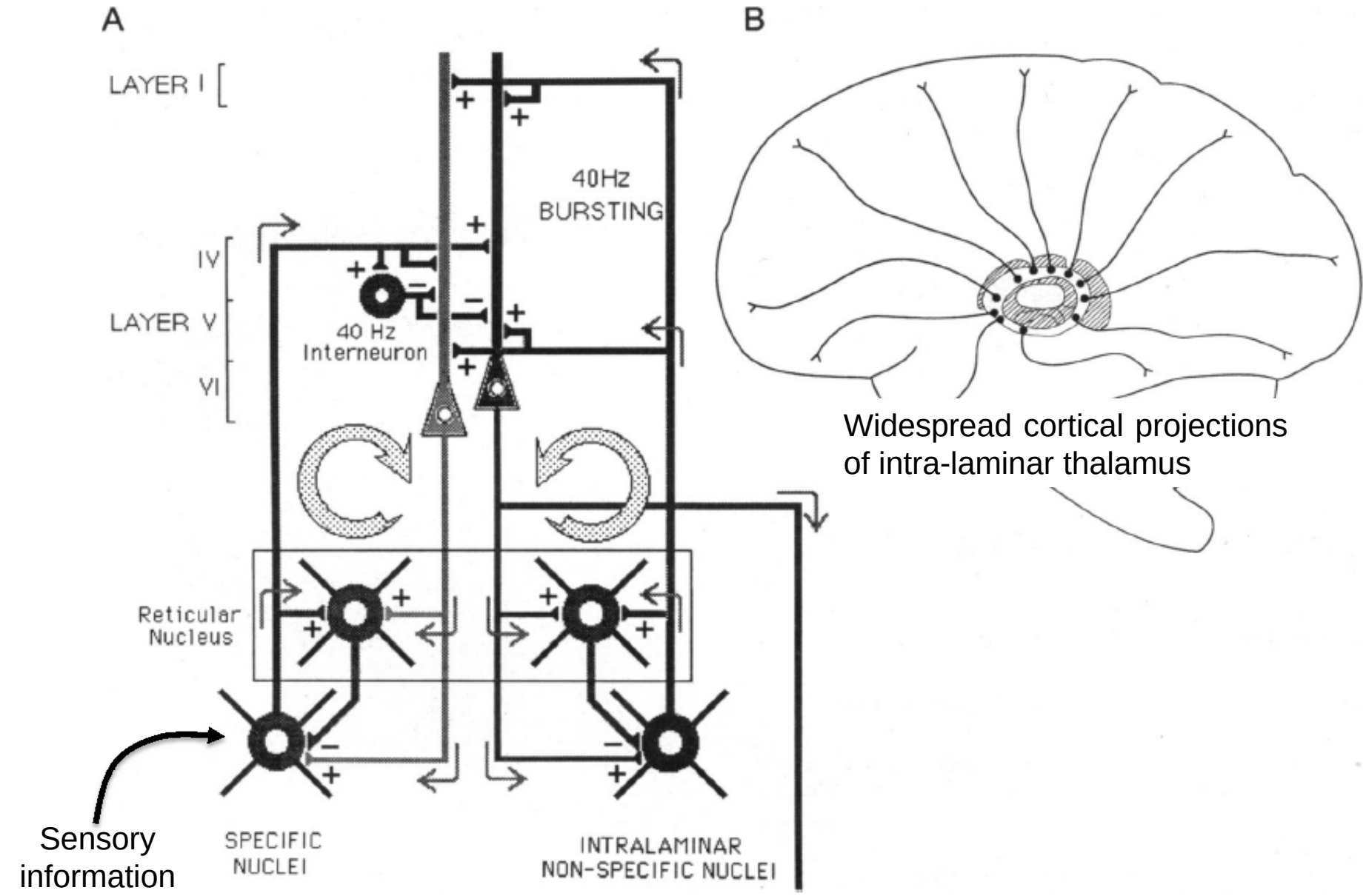
Re-set of 40-Hz oscillations by sensory stimulus (tone) during wakefulness



- Similar to the awake state, REM sleep – the main dream phase of sleep – is characterised by 40 Hz oscillations as measured by surface MEG or EEG.
- Thus, 40-Hz oscillations are a correlate of conscious processing and have been hypothesised to play a key role in such processing.
- Depth recordings in animal models suggest 40-Hz oscillations are generated by thalamo-cortical circuits.
- Only during wakefulness, but not REM sleep, 40-Hz oscillations are re-set by sensory stimulus (tone), in line with fast oscillations being critical for conscious perception of sensory information.

Llinas & Ribary (1993) Proc. Natl. Acad. Sci. USA 90:2078-2081

Thalamo-cortical circuitry hypothesised to underlie 40-Hz oscillations and consciousness

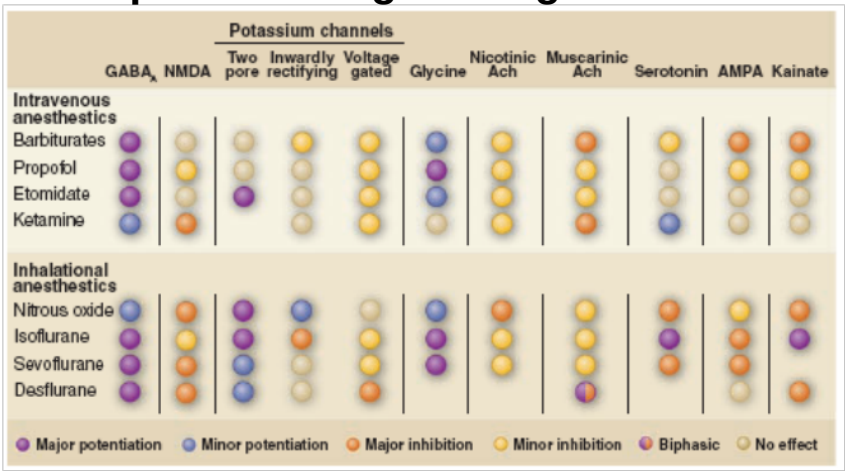


Llinas & Ribary (1993) Proc. Natl. Acad. Sci. USA 90:2078-2081

Anaesthesia and consciousness

•Loss of consciousness is the objective of anaesthesia. Loss of consciousness is not trivial to determine, as unresponsiveness and amnesia are no proof.

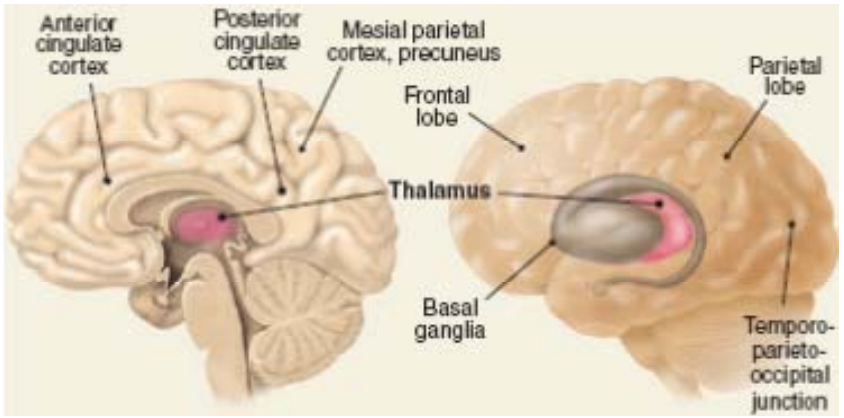
•Neuropharmacological targets of current anaesthetics



•Inhibitory neuro-transmission is enhanced, excitatory transmission decreased.

•Note: this can lead to excitation of certain brain regions due to network effects (e.g., ketamine).

•Brain sites associated with anaesthetic effects



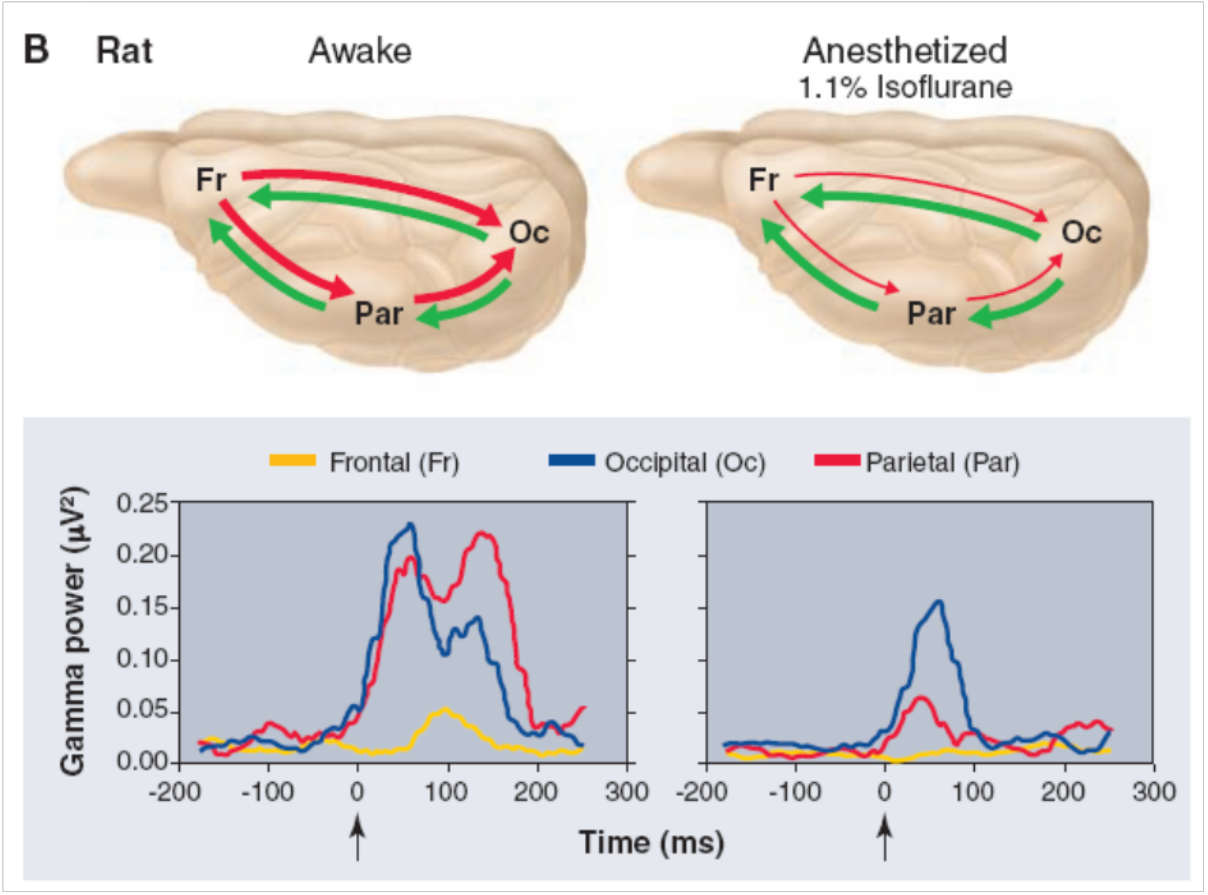
•*Thalamus*: Activity is decreased by many, but not all, anaesthetics (e.g., ketamine); may be secondary to cortical deactivation.

•*Neocortical areas*: Deactivation of, especially posterior, 'mesial cortical areas' and of a lateral 'temporo-parieto-occipital complex'. Note: Primary sensory cortices can often show unchanged responsiveness under anaesthesia!

•Anaesthesia may not necessarily involve cortical deactivation, but disruption of cortical integration, i.e. of the interaction of several cortical areas.

Alkire et al. (2008) Science 322:876-880

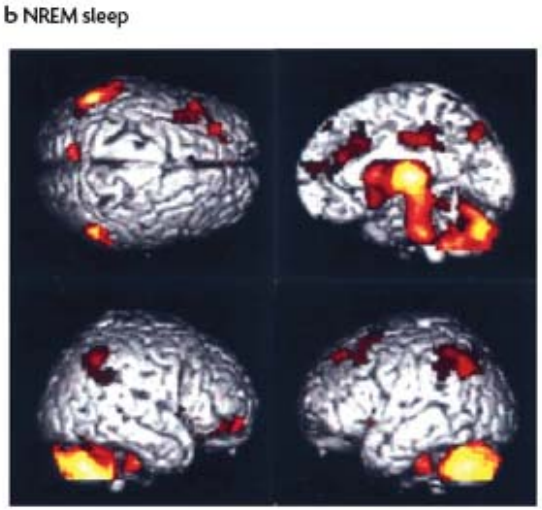
Anaesthesia disrupts cortico-cortical interactions organised by fast oscillations

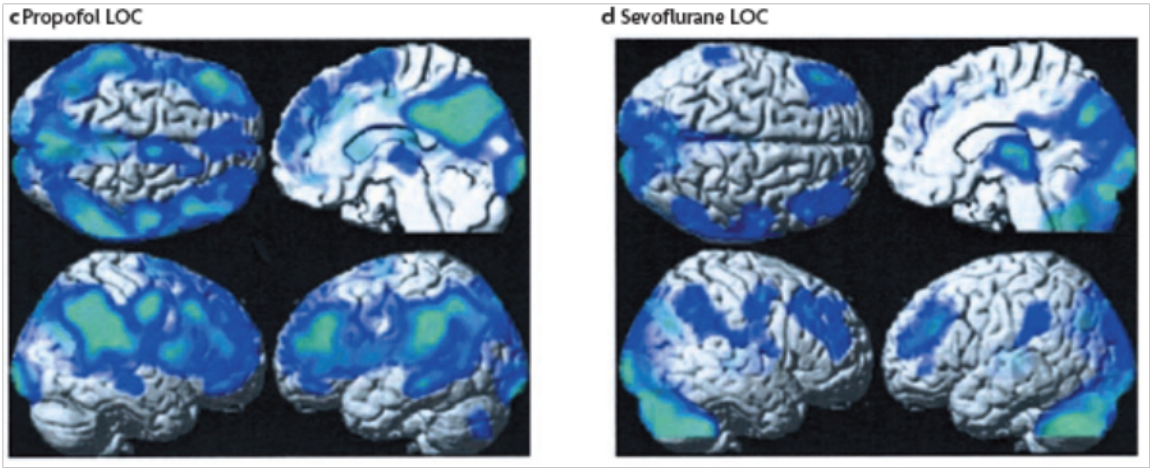


Alkire et al. (2008) Science 322:876-880, based on Imas et al. (2005) Neurosci. Letters 387:145-150

Similarities between sleep and anaesthesia

Regional decreases in brain metabolic activity during sleep and anaesthesia

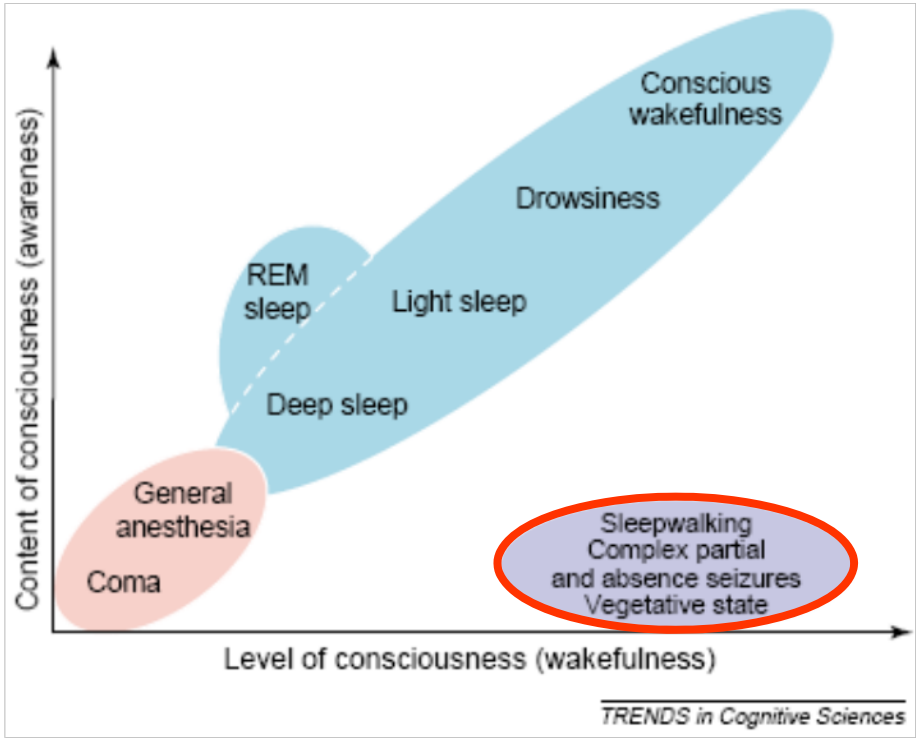




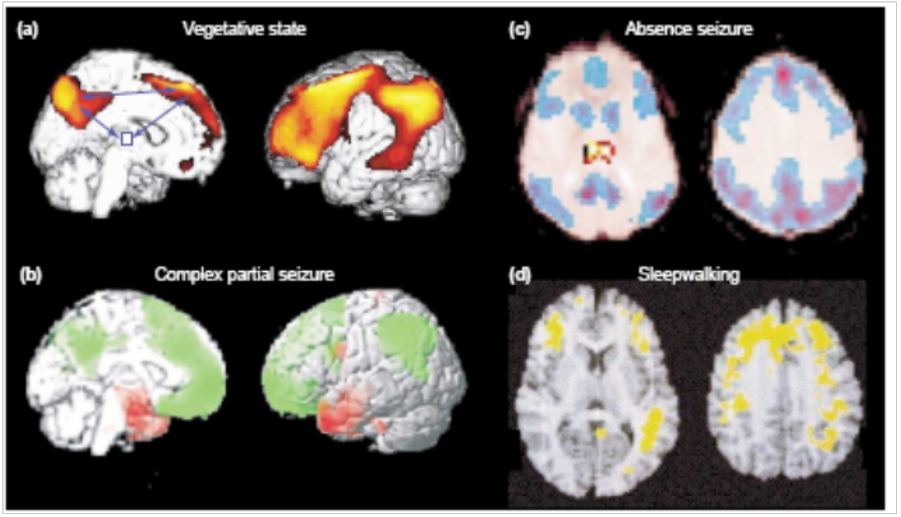
Franks (2008) Nature Rev. Neurosci. 9:370-386;

(Abnormal) Dissociated states of consciousness

There are several abnormal conditions in which wakefulness is relatively preserved whereas consciousness (of anything) appears to be largely absent.



Deactivated brain areas (in comparison to conscious wakefulness)



Laureys (2005) Trends in Cogn. Sci. 9:556-559

Loss of consciousness in epilepsy

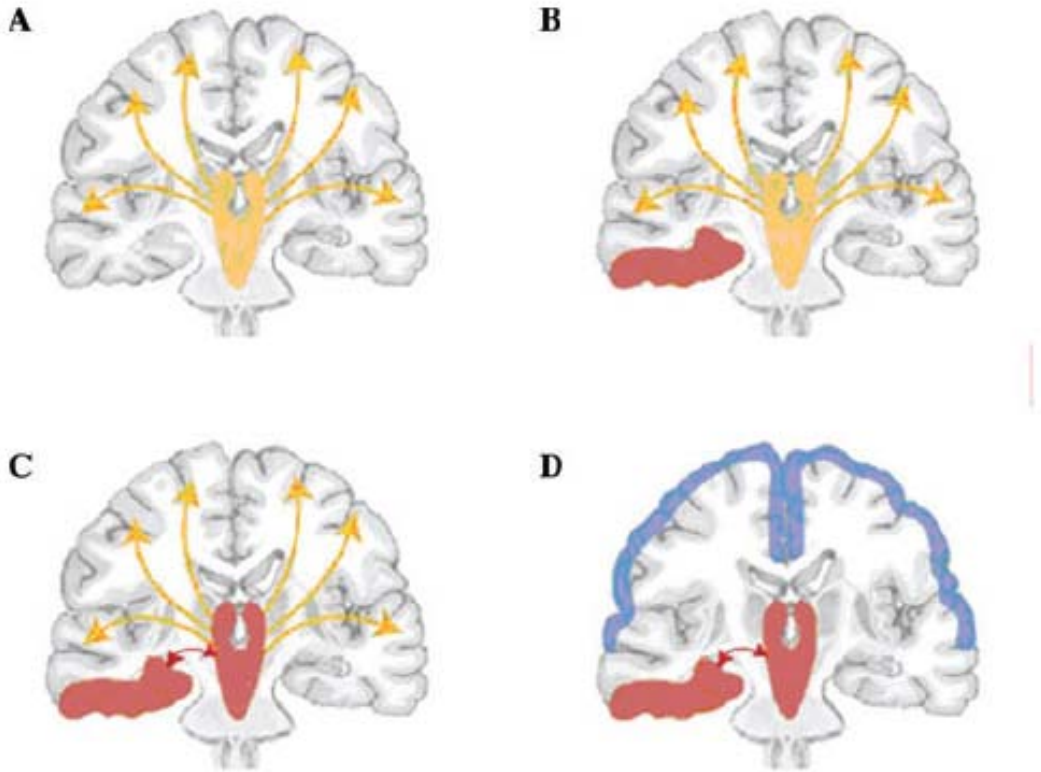
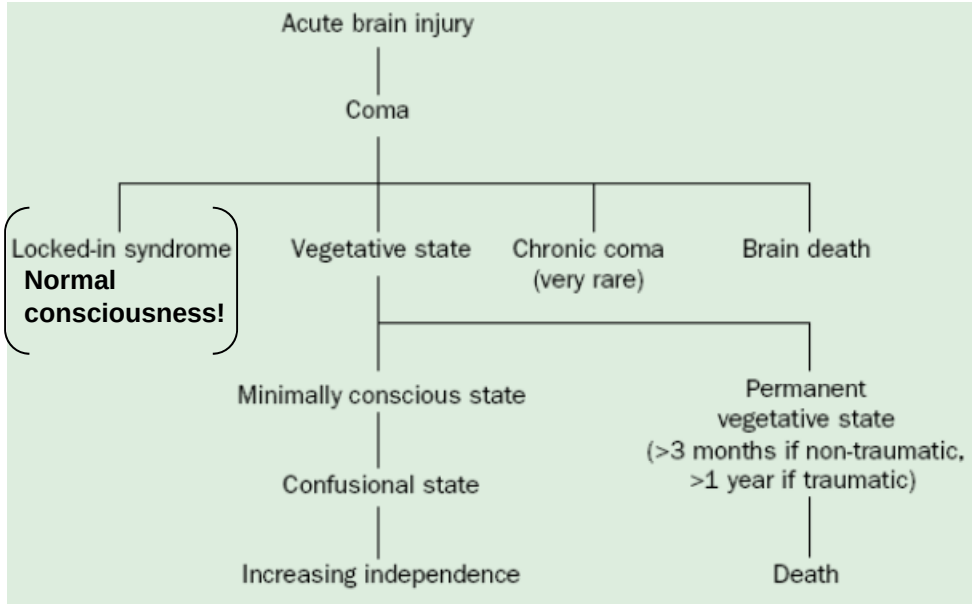


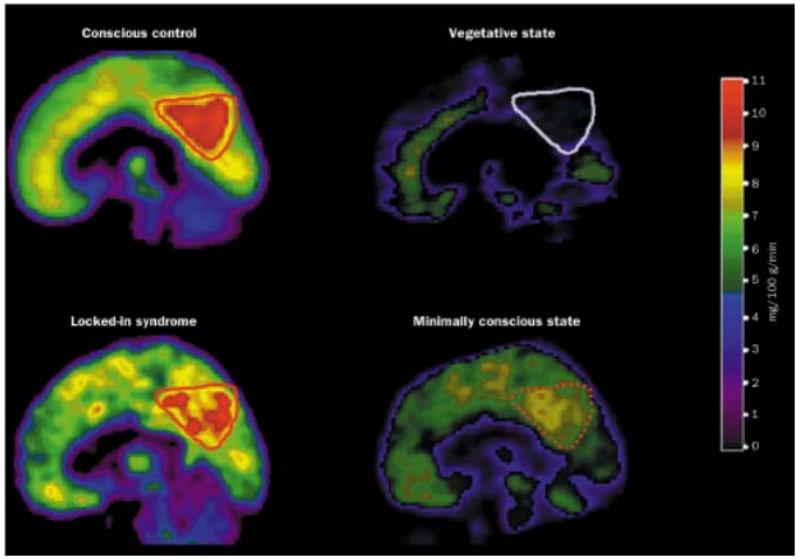
Figure 1. Network inhibition hypothesis for loss of consciousness in complex partial seizures. (**A**) Under normal conditions, the upper brain stem–diencephalic activating systems interact with the cerebral cortex to maintain normal consciousness (yellow represents normal activity). (**B**) A focal seizure (red) involving the mesial temporal lobe unilaterally. (**C**) Propagation of seizure activity from the mesial temporal lobe to midline subcortical structures. (**D**) Disruption of the normal activating functions of the midline subcortical structures leads to depressed activity (blue) in bilateral regions of the fronto–parietal association cortex, leading to loss of consciousness. Reproduced with permission from Ref. 6. (In color in *Annals* online.)

Yu & Blumenfeld (2009) Ann. N.Y. Acad. Sci. 1157:48-60

States of reduced consciousness following brain injury

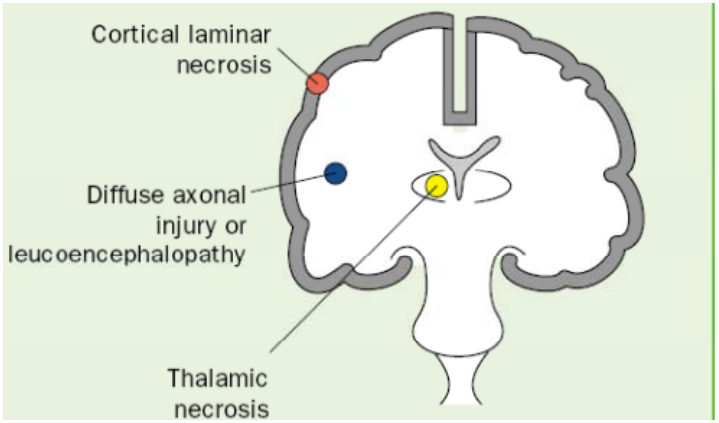


Resting brain metabolism in patients following brain injury and in healthy control



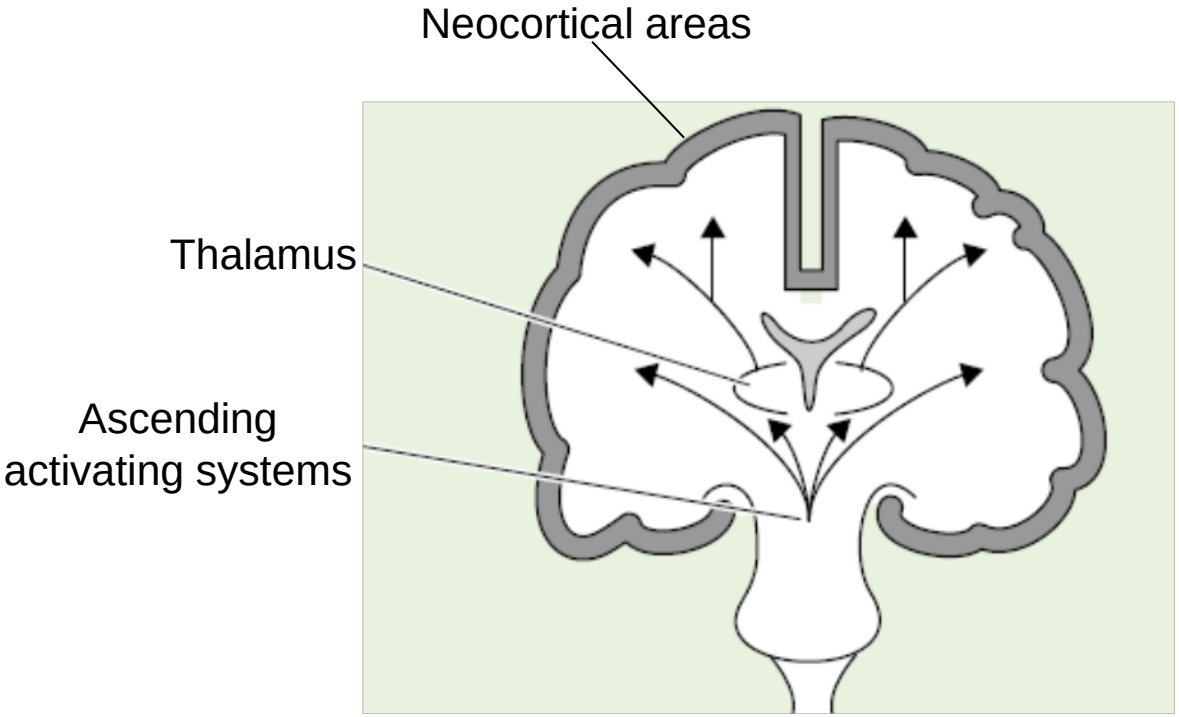
Laureys et al. (2004) Lancet Neurol. 3:537-546

Brain injury associated with vegetative state



Zeman (1997) Lancet 350:795-799

In a nut shell – states of consciousness and brain substrates



•Global states of consciousness depend on a distributed brain network, comprising ascending activating systems, thalamus and neocortex.

•Consciousness seems to involve the temporal coordination of distributed activity in thalamic and neocortical areas as reflected by ‘fast’ oscillations.

Neuronal correlates of specific contents of consciousness (e.g., specific percepts, specific plans, specific memories) . . .??? Next week!

Neuroscience of consciousness I – Selected Reading

***General overviews*:**

Chalmers D (1995) Facing up to the problem of consciousness. Journal of Consciousness Studies 2:200-219 (http://consc.net/papers/facing.pdf)

Zeman A (2001) Consciousness. Brain 124:1263-1289.

Tononi G & Koch C (2008) The neural correlates of consciousness: and update. Ann. N. Y. Acad. Sci. 1124:239-261.

***Sleep & anaesthesia*:**

Alkire MT, Hudetz AG, Tononi G (2008) Consciousness and anaesthesia. Science 322:876-880.

Franks NP (2008) General anaesthesia: from molecular targets to neuronal pathways of sleep and arousal. Nature Rev. Neurosci. 9:370-386.

***Dissociated states of consciousness*:**

Laureys S (2005) The neural correlates of (un)awareness. Trends Cogn. Sci. 9:556-559.

Neuroscience of consciousness I – Some questions to guide your revision

- What is consciousness, what are the problems of consciousness?
- What strategies can neuroscientists pursue to study the brain substrates of consciousness?
- In terms of brain substrates, what seem conscious states to have in common?
- What happens in your brain, so you wake up (‘regain consciousness’) after a night’s sleep?
- How do anaesthetics act in our brains to (hopefully!) result in loss of consciousness?
- What goes wrong in the brains of patients that suffer from disorders of consciousness?