C83MAB: Mind and Brain

Neuroscience of Consciousness I

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What is consciousness?

Look at the grey panel, then flick your eyes between the two pictures. Can you spot the difference? These flicker images work better on the internet (see www.psych.uic.edu/~remsink/flicker/download).
Consciousness

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

• Contents of consciousness (‘consciousness of . . .’) – particular (reportable) experience at a certain level of consciousness

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)

The ‘hard’ problem:

- Subjective experience (‘phenomenal consciousness’, ‘qualia’)

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

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

Abbreviations: EEG, electroencephalogram.
Key brain substrates of consciousness

http://www.scholarpedia.org/article/Neural_correlates_of_consciousness
Wakefulness and sleep - the cardinal states of consciousness

<table>
<thead>
<tr>
<th>Behavioural state</th>
<th>Wake</th>
<th>NREM</th>
<th>REM</th>
</tr>
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<tbody>
<tr>
<td><strong>Cognitive consequences</strong></td>
<td>Acquisition of information</td>
<td>Iteration of informationi</td>
<td>Integration of information</td>
</tr>
<tr>
<td><strong>Conscious experience</strong></td>
<td>Sensation and perception</td>
<td>Dull or absent</td>
<td>Vivid, internally generated</td>
</tr>
<tr>
<td>Thought</td>
<td>Logical progressive</td>
<td>Logical perseverative</td>
<td>Illogical bizarre</td>
</tr>
<tr>
<td>Movement</td>
<td>Continuous voluntary</td>
<td>Episodic involuntary</td>
<td>Commanded but inhibited</td>
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<tr>
<td><strong>Surface recordings</strong></td>
<td>EMG</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>EEG</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>EOG</td>
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</tbody>
</table>

Neurochemistry of wakefulness and sleep
**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.

**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.

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

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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.

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

Widespread cortical projections of intra-laminar thalamus

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

Similarities between sleep and anaesthesia

Regional decreases in brain metabolic activity during sleep and anaesthesia

(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)

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 frontoparietal association cortex, leading to loss of consciousness. Reproduced with permission from Ref. 6. (In color in Annals online.)
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

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:**


**Sleep & anaesthesia:**


**Dissociated states of consciousness:**

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?