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ABSTRACTS

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ES-3-4

FROM FLIP-FLOP TO CYCLES: NEURAL REGULATION MECHANISM OF SLEEP-WAKE STATES

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So far, many hypotheses have been proposed on regulatory mechanism of sleep-wake states. They are roughly classified into the following categories, although their objectives are more or less different from each other.

(1) Prey-predator dynamics: The reciprocal interaction between cholinergic and aminergic systems is considered to regulate nonREM-REM cycle (McCarley and Massaquoi, 1986).
(2) Multiple flip-flops: The regulatory mechanism of sleep-wake states is interpreted as a multiple flip-flops involving the nuclei in the preoptic hypothalamic area, the aminergic neurons, and the glutamatergic and GABAergic neurons in the brainstems (Saper et al., 2001).
(3) Neural diagram: The glutamatergic and GABAergic systems in the brainstem interacting with each other or their cascade are considered to regulate REM generation (Sakai et al., 2001; Luppi et al., 2006). Because (2) and (3) are rather conceptual, their realization and resulting dynamics are not given. Actually, there have been many mathematical models produced based on these idea. Mathematical modeling of regulatory mechanism of sleep-wake states tends to be more focused on circuit implementation to reproduce physiological phenomena rather than the time scale of state alternation. However, for biological reality what kind of actual process determines the time scale is essential. A flip-flop or bistable system is realized by self-excited systems mutually coupled by inhibitory connections, where excitation may come from external systems. Formation of flip-flop is not a sufficient condition for alternation of multiple states, but an unstabilizing mechanism of each state is essential to determine the time scale of state alternation, i.e., minutes in rodents and hours in human. Actual determinant factors for such a slow dynamics are not identified, which have not been seriously concerned so far. This situation is the same as in (1) and (3). Such slow dynamics cannot be attributed only to neural synaptic interactions. Possible biological processes are discussed from the modeling viewpoint.

ES-4-1

AROUSAL-RELATED LAPSES OF RESPONSIVENESS: CHARACTERISTICS, DETECTION, AND UNDERLYING MECHANISMS

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Lapses of responsiveness ('lapses') are complete transient disruptions in sensory-motor performance. They are a surprisingly frequent phenomenon in healthy subjects – even when not sleep-deprived – and particularly so when engaged in extended monotonous tasks. They are of particular importance in the transport, military, and medical sectors in which there is a need to maintain sustained attention for extended periods and in which lapses can lead to multiple-fatality accidents.
Lapses can be broadly divided into four main types:

- Sleep events (>15 s) – Extended loss of consciousness.
- Behavioural microsleeps (~0.5–15 s) – Brief loss of consciousness, with clear behavioural indications of drowsiness.
- Lapses of sustained attention – Not directly related to level of arousal and can occur when alert, fatigued, or drowsy.
- Lapses of task-orientated attention – Diverted attention.

Our primary focus is on microsleeps, with contributions covering aspects of behavioural detection and characterization, EEG-based characterization and detection, and determination of the underlying mechanisms in the brain via simultaneous recordings of whole-brain BOLD fMRI, 64-ch. EEG, eye video, and EOG, while performing a continuous 2-D visuomotor tracking task. In addition to improving our understanding of what happens in the brain during microsleeps, it is hoped that improved knowledge of the spatiotemporal dynamics of microsleeps will allow us to substantially improve the early detection, and even prediction, of microsleeps and to use this as the basis for a non-invasive early-warning systems with the potential to save many lives.
My talk will (i) provide an introduction to lapses, (ii) overview their importance in the real world, (iii) overview what several research studies have revealed about microsleeps, and (iv) summarize some of the remaining challenges in this fascinating and important area.

ES-4-2

CAPACITY LIMITS OF INFORMATION PROCESSING WHEN SLEEP DEPRIVED

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Our capacity to process information declines when sleep deprived. Perceptual processing capacity refers to our residual capacity to process peripheral distractors after processing a central task. Visual short term memory capacity refers to the number of visual items we can perceive and remember over a few seconds.
Perceptual processing capacity limitations may not be evident at lower perceptual load but can be uncovered at higher load using functional magnetic resonance adaptation. Indeed our ability to allocate attention to a specific visual category may be compromised during sleep deprivation but the extent to which this occurs relates to the temporal predictability of target appearance. Imaging visual cortex provides a means of uncovering loss of attentional selectivity, which in turn is a contributor to impaired cognition in the setting of sleep deprivation.
Visual short term memory is indeed affected by sleep deprivation but for reasons not obvious from observing behavior. We found deficits in engagement of fronto-parietal activation usually engaged in task performance that suggests a memoranda independent effect on neural activation that can be remedied in vulnerable persons by pharmacologic means.

ES-4-3

A MOBILE EEG DEVICE FOR ON-LINE ASSESSMENT OF SLEEP QUALITY

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The polysomnography (PSG) which senses multiple physiological signals including EEG, ECG, EMG and EOG signals is a traditional and common device used to diagnose the participants sleep problems in clinic. People who have the sleep problem will pre-register to do the