neural mechanisms for attention III – input selection / functional anatomy
Brain mechanisms for sleep and attention overlap extensively. For example, the cerebral cortex, where conscious perception is realized, undergoes radical changes in the patterning of synaptic potentials (as revealed by EEG/LFP recordings) between the lowest-attention state (stage \( \frac{3}{4} \) non-REM sleep) and high attention states (waking, REM sleep).

Changes in sleep/wake state and attention are sometimes mediated by groups of neurons that are highly interconnected (brainstem reticular and thalamic reticular neurons).

The classroom can be very hot.

REM sleep appears to be associated with a maximal frequency of events associated with reorientation of attention (as in a startle response) while non-REM sleep is associated with a minimal frequency of such events. The frequency of such events in the waking state lies between the two sleep states. Oddly enough, a similar pattern is observed for brain metabolism.

Work attempting to uncover the function of sleep typically takes either a species-comparison approach, a sleep-deprivation approach, or an approach involving recording of specific neurobiological characteristics of sleep.

Theories as to the function of sleep nearly always suggest that the function pertains to the brain as opposed to the rest of the body.
Neurally, attention is associated with either changes in the overall patterns of firing across a group of neurons (increased action potentials in response to the attended stimulus, and fewer to the unattended stimuli) and/or changes in the temporal firing patterns of neurons (neurons responding to attended stimuli fire in tune with a gamma rhythm). Such changes may, in part, be brought about by changing the subset of synaptic inputs to which a neuron responds most strongly.
what do we know so far (since midterm 1 material)?

Neural mechanisms for attention fall into 3 basic categories. 1) changes in signal-to-noise ratio. Here differences in the selectivity for firing responses of neurons are accentuated, in one or another form, by attention; 2) changes in the temporal coherence of neurons. Here, attention increases the degree to which neurons fire with temporal relation to a gamma frequency. 3) changes in the functional anatomy of neurons. Although neurons usually have thousands of synaptic inputs, they are not always ‘listening’ to all of them. Even synapses that are strong (more depolarizing when activated) can be depressed temporarily.
ACh and NE depress responses of ventrobasal thalamic neurons (which relay input from vibrissae) to the inputs they receive from the cortical area (barrel field) to which they project.

But... this depression is specific to low-frequency inputs... responses to high frequency inputs are actually enhanced.

ACh and NE slightly depress responses of olfactory cortex neurons to inputs from the olfactory bulb (= direct sensory inputs = layer 1a inputs).

ACh and NE strongly depress responses of olfactory cortex neurons to inputs from other regions of cortex (=association inputs = layer 1b inputs).
ACh can differentially modify strong versus weak synaptic inputs. This may explain deficits in the following olfactory discrimination task:

- odor A versus odor B – choose A (once learned):
- odor A versus odor M – choose M
- odor D versus odor E – choose D

Animals with ACh lesions have trouble with the A/M, but not D/E, pairing. ACh is thought to permit A/M learning by depressing, specifically, those inputs potentiated during learning of the A/B pairing.

Linster et al., Neurobiology of Learning and Memory, 2003
attention within sleep – ACh may modulate what CA3 and cortex respond to in sleep versus wake and thereby form a mechanism for memory transfer