# BRAIN MECHANISMS and SLEEP – p.1

## 1. Wakefulness & Arousal

***Reticular Formation*** (Reticular Activating System) (in midbrain)

Including the ***Pontomesencephalic*** system

In ***pons & midbrain***

Receives sensory input from many ascending pathways

Sends its input to the ***Thalamus*** and the ***Basal Forebrain***

Releases ***Acetylcholine*** and ***Glutamate*** (✍ EPSPs)

If RAS stimulated in a sleeping subject ✍ subject awakens

If RAS stimulated in an awake subject ✍ subject becomes more alert, aroused

If basal forebrain is damaged ✍ subject shows impaired alertness, impaired learning, poor attentional focus, increased NREM (e.g. in Alzheimer's Disease)

Thus, increased activity in the pontomesencepalon, thalamus, and basal forebrain keeps us awake, alert, and ready to respond to new challenges

***Locus Coeruleus*** (in midbrain, in region of pons)

Also involved in arousal, becomes active during "meaningful" events (most likely as determined by cortex)

Has widespread axons to the cortex, incl. basal forebrain

Secretes (is the major source of) ***Norepinephrine***

Suppresses REM sleep

If stimulated ✍ strengthens storage of recent memories

***Hypothalamus*** also has some pathways to basal forebrain

Secretes ***Histamine*** ✍ arousal increases

Note: basal forebrain lies just anterior and dorsal to hypothalamus

Anterior hypothalamus has neurons that secrete ***Orexin/Hypocretin***

✍ stimulate release of ACh in forebrain and brainstem ✍ wake

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2. **Getting to Sleep (esp. NREM sleep)**

Have to decrease arousal:

Decrease temperature of brain (and body)

Decrease level of incoming stimulation (or use exposure to repetitive, non-meaningful stimuli)

**Actively inhibit** brain's arousal systems (sleep is an active process, is not just the lack of something)

During wakefulness, AMP (adenosine monophosphate) breaks down into ***Adenosine***, which begins to accumulate in brain (has little effect in most of brain)

However, there are RSs for adenosine in basal forebrain ✍ Basal forebrain is inhibited ✍ long-lasting suppression of arousal ✍ sleep

During wakefulness, ***Prostaglandins*** also build up in brain ✍ inhibition of hypothalamic cells that normally cause increased arousal

During a high fever ✍ increased Protaglandins secreted ✍ more likely to sleep

Cells in the Hypothalamus and in the Basal Forebrain also increase the release of ***GABA*** ✍ sleep

Note: these GABA-releasing cells get much of their input from cells in the ***preoptic*** and ***anterior hypothalamus*** (areas that control temperature of body)

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### 3. **Getting Into REM Sleep**

In general, in REM sleep activity in brain is **increased** in the Pons, Limbic System, & parts of the Parietal and Temporal Lobes

Activity is **decreased** in the Occipital Lobe, Motor Cortex, and Dorsolateral Prefrontal Cortex

Activity in the **Pons triggers REM sleep – PGO "spikes"**

P – Pons

G – (lateral) Geniculate (nucleus of thalamus)

O – Occipital lobe

If subject is REM deprived, PGO waves begin to occur in NREM

And even during wake (while subject's behavior looks "strange" as if subject were hallucinating…)

Areas near pons also send axons to spinal cord alpha motor neurons

✍ inhibit these neurons ✍ no contractions ("paralysis") of major muscles of body

***Acetylcholine*** increases during REM sleep (as it does in wakefulness)

Both ***Norepinephrine*** and ***Serotonin*** will interrupt or decrease REM