Sleep and memory

10-20 billion neurons In Brain generate recordable potentials

These recordable potentials Are 2 TYPES

1) Evoked c. potential

Caused by
SENSORY STIMULATION

2) spontaneous c. rhythm

It represents **EEG**
(Spontaneous electric brain activity)

EEG Finds *Changes in electrical brain activity*
To diagnose Brain:

- **Tumors**
- **Damage**
- **inflammation**
- **Stroke**
- **Sleep conditions**

⚙:
Tiny
Dogs
Ignore
SSleep

Sleep definition

Sleep: arousable unconscious state

Age	Sleep time (h)
Infant 👶	18
Adult 🧑	8
Old 🧓	6

Sleep types & cycle

Sleep types:
1) **rapid eye movement (REM)**
2) **Non rapid eye movement (NREM)** (Slow wave)
   (In NREM; waves are strong & low)

———	REM	NREM
Duration (m)	5-20	90
Represent	**25%** of our sleep	**75%** of our sleep
Dreams	*Usual* (Vivid - nightmares)	*Maybe* (Not remembered)
Muscle activity	**Marked** reduction (Almost paralyzed)	**Mild** reduction (Not fully relaxed)
Heart rate	Increased & irregular	Mild reduction
Rest	No	Yes
EEG waves	Rapid *waves* Low *voltage*	Slow *waves* High *voltage*
	Active brain β wave - like rythm	Becomes deeper & restful

Highest frequency **γ - β - α - θ - δ** lowest frequency
Highest consciousness — — — — — — — — — lowest consciousness

Sleep cycles:

1: **NREM sleep** (stages 1,2,3,4) for 90 minutes
2: **REM sleep** for 5-20 minutes

This repeats **4-5 times** at one night sleep for a normal adult
NREM -> REM -> NREM -> REM -> NREM -> REM... until you wake up

Sleep theories

- **ACTIVE INHIBITORY PROCESS** (active)
- **FATIGUE OF RAS** (passive)

1) Initiation of NREM

STIMULATION OF BRAIN AREAS
Result In SLEEP

THESE AREAS ARE:

- **Raphi nuclei** (in lower pons & medulla)
  It gives nerves to *brainstem reticular formation*, & up thalamus, hypothalamus, Limbic system & cortex. There they **Release serotonin** (transmitter causes sleep)
- **Nucleus tractus solitarius** (in medulla)

2) Initiation of REM

↑ **activity of ACH neurons**
In upper brainstem reticular formation

These neurons excite:
RAS/ THALAMUS/ CORTEX

3) termination of REM

↑ serotoninergic neurons activity in raphi nuclei -> inhibit
cholinergic neurons in RAS (upper brainstem reticular formation)

Sleep Significance

Sleep significance in general:
Restoring normal brain activity & different CNS functions

Stage 4 NREM Sleep:
 Brain recovery

(because ↓ cerebral metabolic rate & blood flow
  Result: ↓ **free radicals & their damage**)

REM Sleep:
- (Facilitate **Brain development** changes in **infants** & young children because they spend more rem time)
- (Enhances chemical & structural changes in **learning & memory**)

Less sleep = less higher brain functions

Sleep and memory

Memory definition

Memory: acquired recallable knowledge or skill

Memory
Has 2 TYPES
(According to nature of information)

1) Explicit (Declarative)

Facts & Events
(Conscious recall)

1) Implicit (Non- Declarative)

Skills & habits
(Driving, piano, memory writing)

Memory
Has 3 TYPES
(According to the time course of storage)

1) Short term (STM)

Seconds of minutes
(Then fades or turns to other types)

2) Intermediate term (ITM)

Hours or days

3) Long term (LTM)

Several years
(May become permanent)

Memory mechanism

Memories are stored by
FACILITATION IF SYNAPTIC TRANSMISSION
The new (Facilitated) pathways
are called **MEMORY TRACES,**
they can be selectively Activated to recall memories.

STM mechanisms

Short term memory: Achieved by either:

- **reverberating circuit activation**
- **Post-tetanic Potentiation**
- **Presynaptic terminals facilitation**

- **Presynaptic terminals facilitation**
  (Synaptic sensitization)
  - Presynaptic Facilitatory neuron release serotonin
  - Serotonin closes presynaptic K+ channels using cAMP
  - prolonged presynaptic AP
  - prolonged Ca2+ channel activation
  - more presynaptic Ca2+ entry -> <u>more transmitter release</u>
  - augmented post synaptic potential (PSP)

ITM mechanism

Intermediate term memory
Based on **Chemical changes**
In presynaptic terminal
Or post synaptic membrane
Both increase **SYNAPTIC SENSITIVITY**

LTM mechanism

Long term memory
Based on **structural changes** at the synapses
The changes <u>enhance/suppress</u> signal conduction
These changes include:

- ↑ vesicle release sites for transmitter release
- ↑ transmitters vesicles release
- ↑ presynaptic terminals
- Dendritic spines structural change **(Stronger transmission)**

Memory consolidation

Memory consolidation
A process to convert STM TO LTM
It takes 1 hour of rehearsal & repeating same info over & over

Memory consolidation involved organs
(Principal decision making of info importance)
(Associated with reward & punishment)

- **Hippocampus** : *DRIVE* makes mind rehears new info
- **Amygdala**

i: different kinds of sensory memories are stored
in different cortical association areas
Visual in visual, voices & words in auditory, faces in face recognition

Amnesia

Amnesia: memory impairment
due to lesion in Certain brain areas

Types of amnesia	Retrograde	Anterograde
Feature	Can't recall *past* LTMs	Can't store *new* LTMs
Cause	Brain trauma	Lesion in medial portions of temporal lobe & hippocampus (Consolidation areas)
Patient can recall:	*Present*	*Past*

Senile dementia (old age), **Alzheimer disease** (middle age)
But both can occur at any age, & characterized by:
1: memory impairment, 2: lack of concentration, 3:inattentive
Cause: loss of cholinergic terminals from nucleus basalis
To neocortex, amygdala & hippocampus