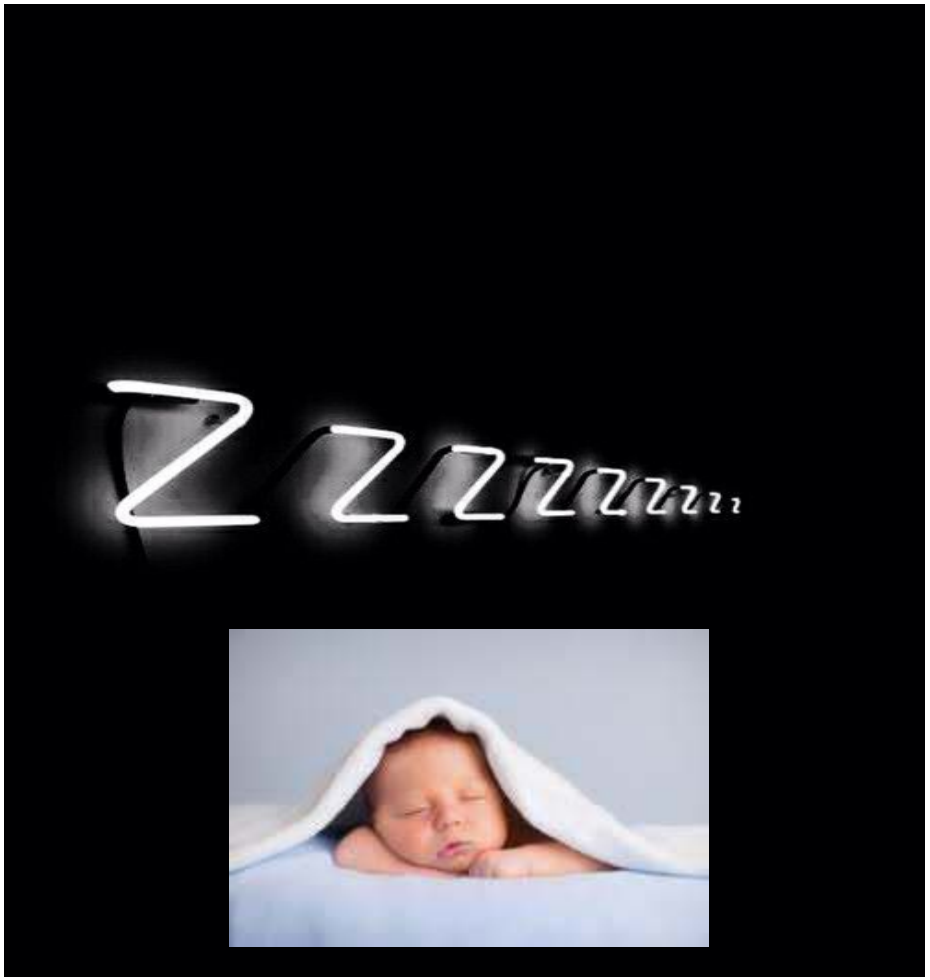
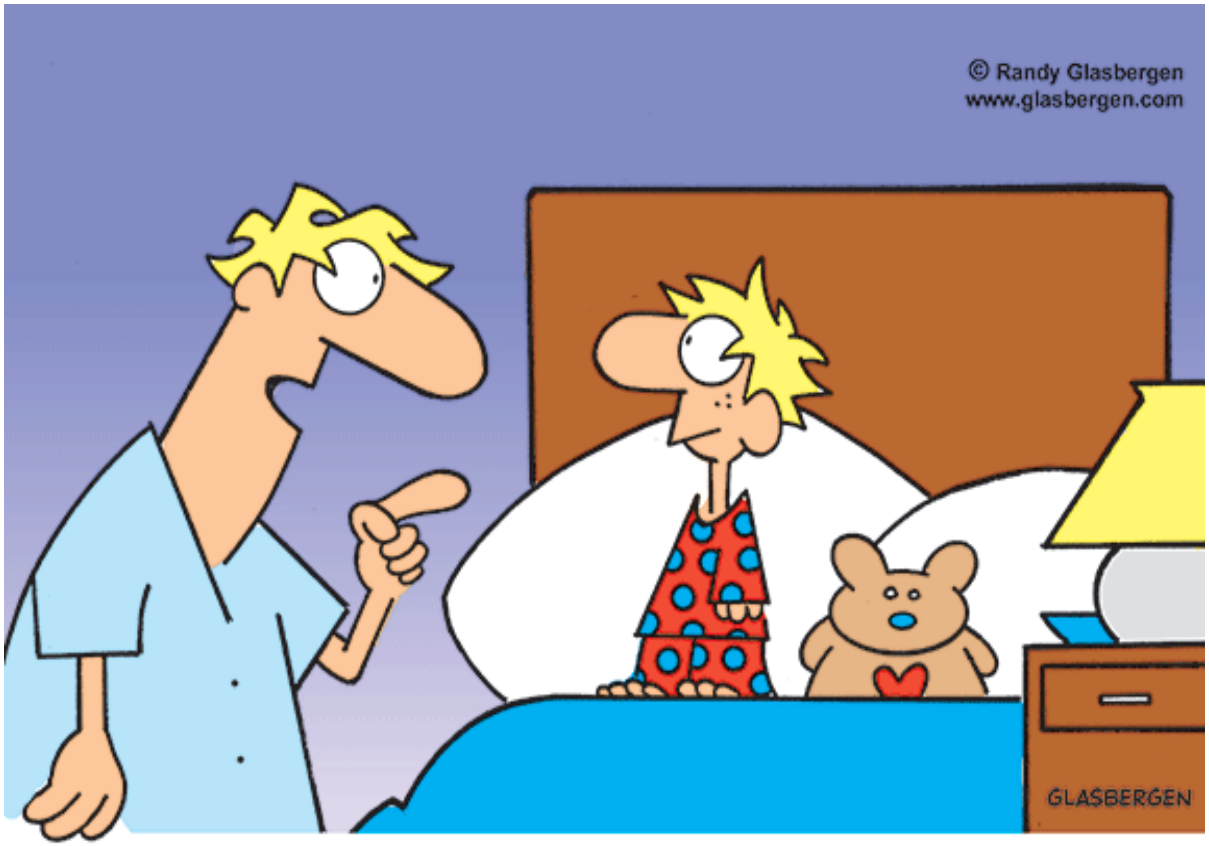
Sleep and Sleep Disorders

Dr Bharat R Shah M.D.

For all those who love sleep: What is Sleep?



What is Sleep?



"It's a time machine! You get in it tonight and when you wake up, it's *tomorrow!*"

What is Sleep?

A normal, reversible, recurring behavioral state of disengagement and unresponsiveness to the environment that is characterized by typical changes in the electroencephalogram.

- A natural periodic state of rest for the mind and body, in which the eyes usually close and consciousness is completely or partially lost, so that there is a decrease in bodily movement and responsiveness to external stimuli
- A state of unconsciousness but still able to awaken by normal sensory stimuli

How much sleep do we need?

- Infants 16-20
- Toddlers 12-14
- Pre School 11-13
- School Age 10-11
- Teens 9.5-10

- Most adults need 7 ½ -8 hours to function well
  - About 10% require more or less sleep
  - Pregnant women need more sleep

IF YOU ARE 30…

MY 12 YEARS ASLEEP IS EQUIVALENT TO THE...

2 years building the Eiffel Tower took

and the

2 years building the Titanic took

and the

5 years the film Gravity took to make

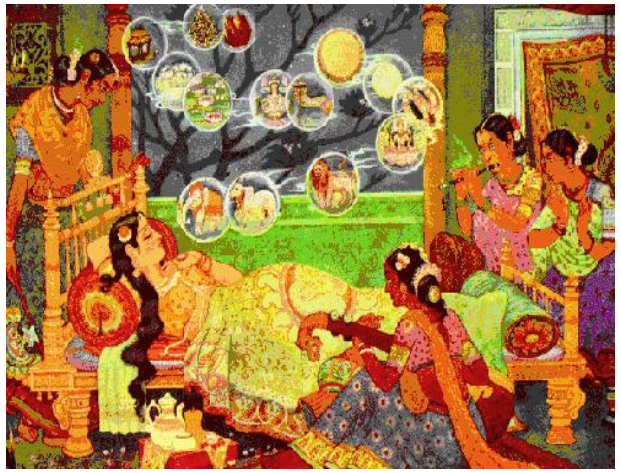
DREAM CONTENT

It is found that people all over the world dream of mostly the same things. Personal experiences from the recent past are frequently incorporated into dreams

1. Negative Emotional Content: 8 out of 10 dreams have negative emotional content.
2. Failure Dreams: People commonly dream about failure, being attacked, pursued, rejected, or struck with misfortune.
3. Sexual Dreams: Contrary to our thinking, sexual dreams are sparse. Sexual dreams in men are 1 in 10; and in women 1 in 30.
4. Dreams of Gender: Women dream of men and women equally; men dream more about men than women.

Meaning of Dreams

- In ancient societies, dreams guided political, social and everyday decisions.
- Early religious books are filled with references to divine visions during sleep.
- In the Mandukya Upanishad , a dream is one of three states that the soul experiences during its lifetime, the other two states being the waking state and the sleep state
- Greek philosophers attributed dream content to natural sources.



Dream Theories

- Activation Synthesis
- Information Processing
- Cognitive Theory
- Psychodynamic Theory

Dreams mean very little

Dreams mean quite a bit.

Dream Theories

DREAM THEORIES

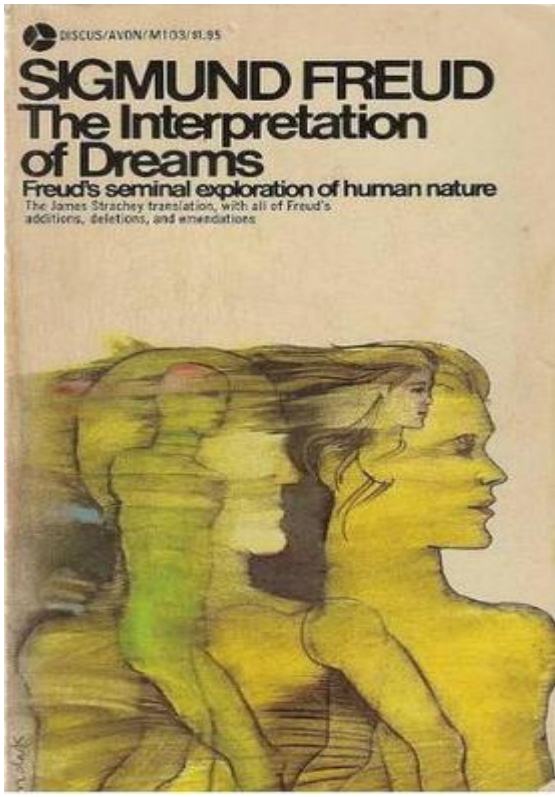
Theory	Explanation	Critical Considerations
Freud's wish-fulfillment	Dreams provide a "psychic safety valve"—expressing otherwise unacceptable feelings; contain manifest (remembered) content and a deeper layer of latent content—a hidden meaning.	Lacks any scientific support; dreams may be interpreted in many different ways.
Information-processing	Dreams help us sort out the day's events and consolidate our memories.	But why do we sometimes dream about things we have not experienced?
Physiological function	Regular brain stimulation from REM sleep may help develop and preserve neural pathways.	This may be true, but it does not explain why we experience *meaningful* dreams.
Activation-synthesis	REM sleep triggers impulses that evoke random visual memories, which our sleeping brain weaves into stories.	The individual's brain is weaving the stories, which still tells us something about the dreamer.
Cognitive theory	Dream content reflects dreamers' cognitive development—their knowledge and understanding.	Does not address the neuroscience of dreams.

Meaning of Dreams

- In the 19th century, Sigmund Freud promoted popular theory that dreams gave us access to our repressed conflicts.

  He called them "the royal road to the unconscious “

  He considered his book ‘The Interpretation of Dreams’ as his most important work.
- Jung described dreams as messages to the dreamer, with revelations that can uncover and help to resolve emotional or religious problems and fears
- Alfred Adler, believed that dreams reflect current lifestyle and offer solutions to contemporary problems.



Meaning of Dreams

- Freud proposed that dreams protect sleep, which might be disturbed by the arousal of unacceptable wishes and thoughts
- Hartmann said dreams may function like psychotherapy, by "making connections in a safe place" and allowing the dreamer to integrate thoughts that may be dissociated during waking life

- Bad dreams let the brain learn to gain control over emotions resulting from distressing experiences.

- Dreams may also create new ideas through the generation of random thought mutations or may help certain types of learning
- Ramanujan attributed his mathematical genius to dreams
- Neils Bohr finally got the structure of atom right after a dream of it!

Meaning of Dreams

- In 1977 Allan Hobson and Robert MacCarley proposed the “activation – synthesis hypothesis.

They suggested that dreaming consists of association and memories elicited from the forebrain (Neocortex and associated structure) in response to random signals from the brain stem (ponto-geniculo-occipital spikes).

Dreams were merely the “best fit” the forebrain could provide to this random bombardment from brain stem.

- During the night there may be many external stimuli bombarding the senses, the mind interprets the stimulus and makes it a part of dream in order for a continued sleep

Why do we sleep ?

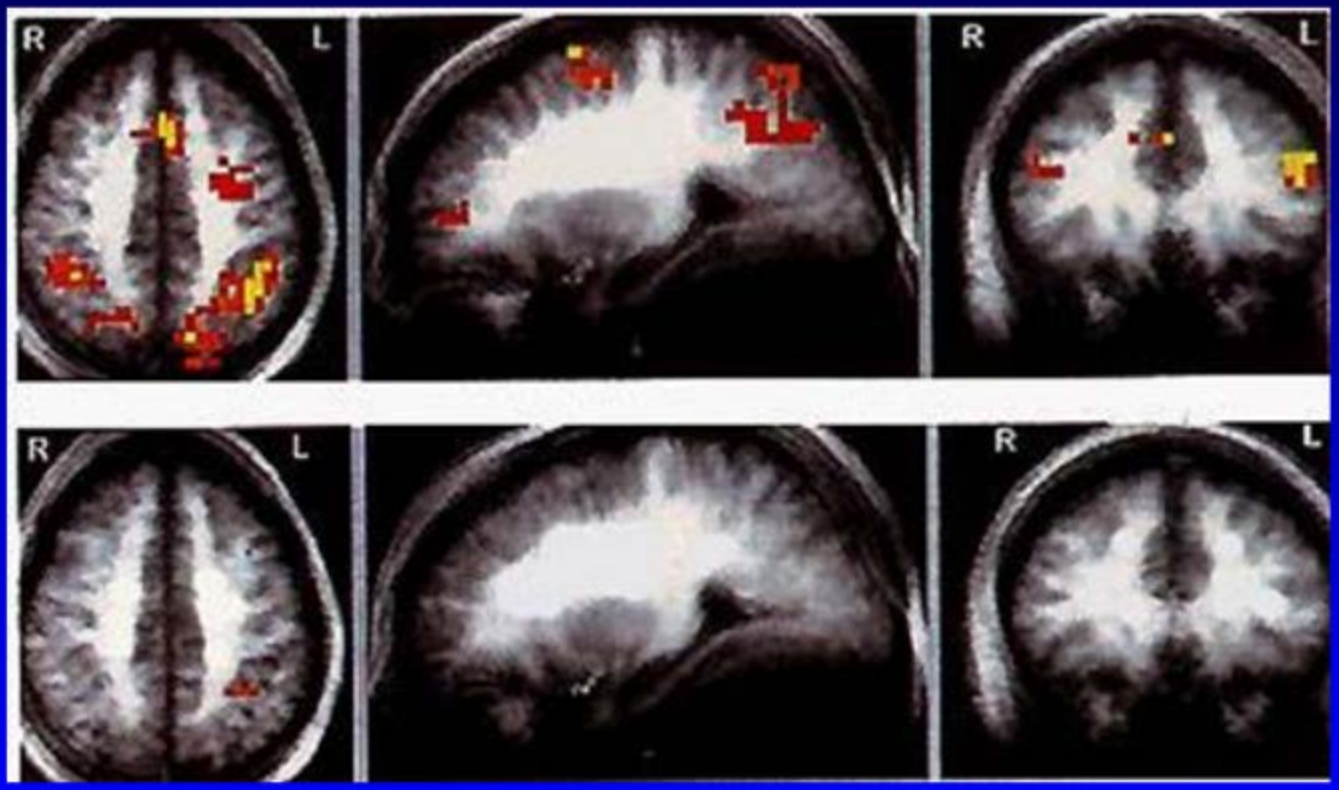
SLEEP DEPRIVATION

- Early reports of bizarre or psychotic behavior
- Wide individual variability (personality and age factors)
- Most common effects of sleep deprivation:
  - increased irritability
  - decreased concentration
  - Confusion/disorientation

Why do we sleep?

Sleep deprivation

Sleep Deprivation: Effect on cognition



fMRI during serial subtraction task

Normal sleep – Activation of PFC, parietal, pre-motor cortex

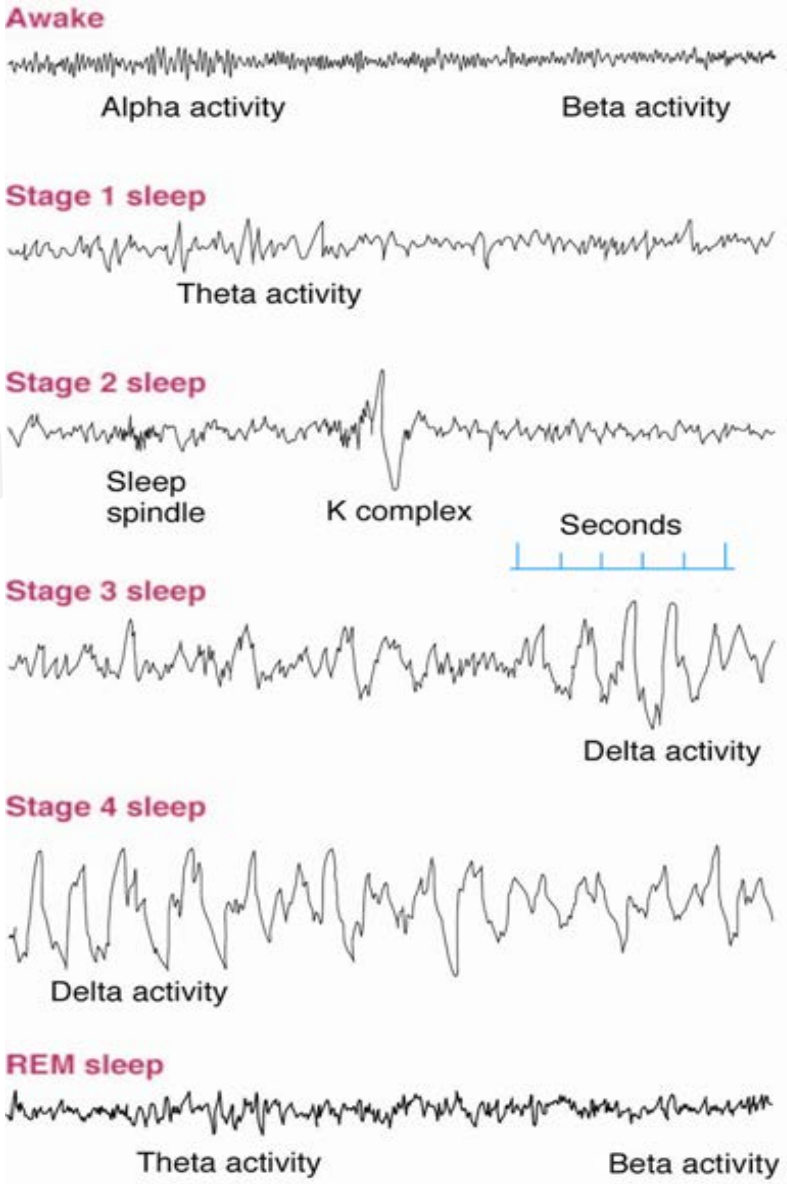
Following sleep deprivation – Decreased activation

Sleep deprivation in medical trainees

- **Surgery:** 20% more errors and 14% more time required to perform simulated laparoscopy post-call (two studies) Taffinder et al, 1998; Grantcharov et al, 2001
- **Internal Medicine:** efficiency and accuracy of ECG interpretation impaired in sleep-deprived interns Lingenfelser et al, 1994
- **Pediatrics:** time required to place an intra-arterial line increased significantly in sleep-deprived Storer et al, 1989

SLEEP PHYSIOLOGY

- SLEEP STAGES
  - Stage 1: alpha and theta waves (initial)
  - Stage 2: K complexes, sleep spindles
  - Stage 3: 20-50% delta (SWS)
  - Stage 4: > 50% delta (SWS)
  - REM (emergent stage 1)



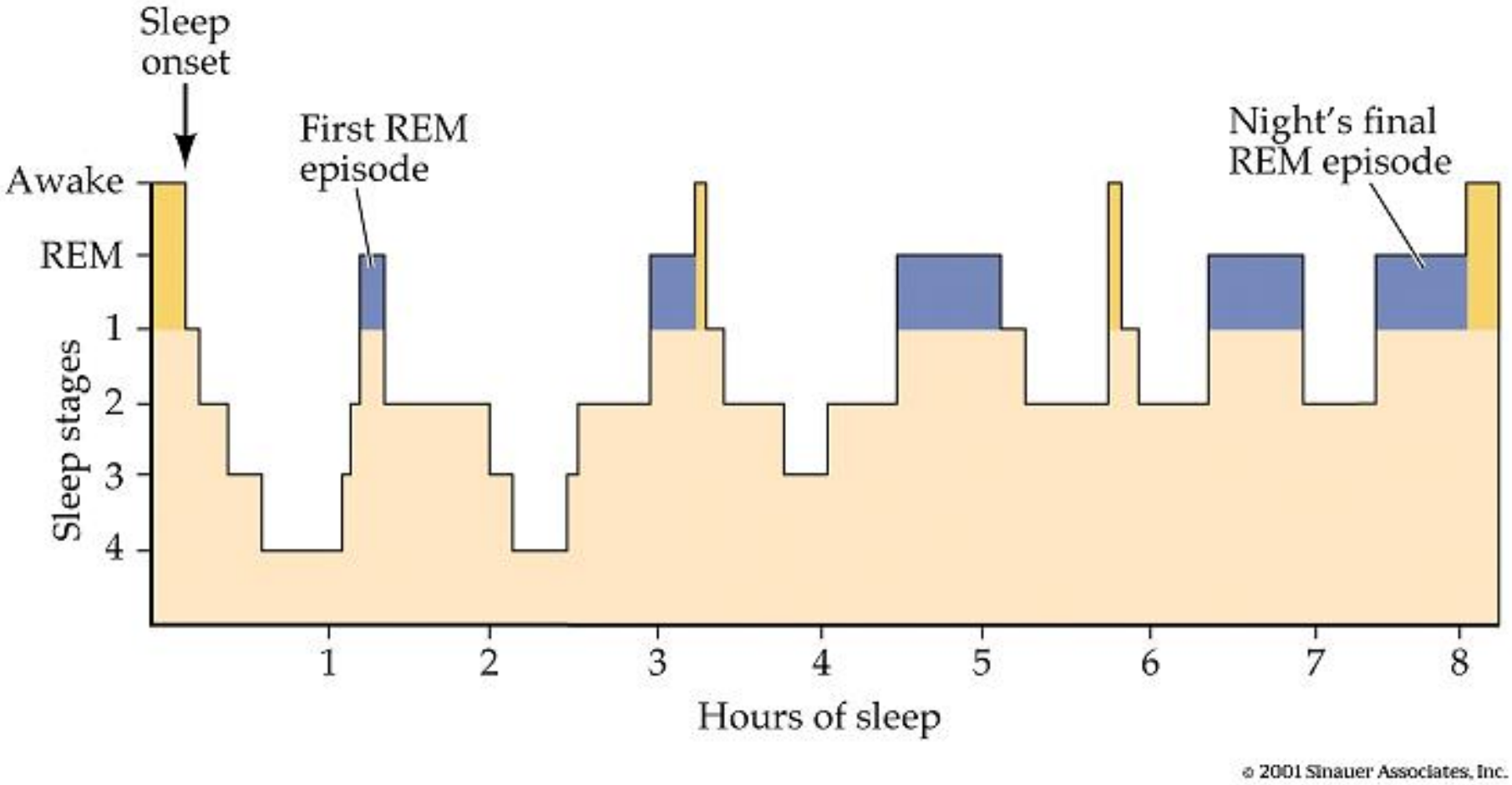
Sleep cycle

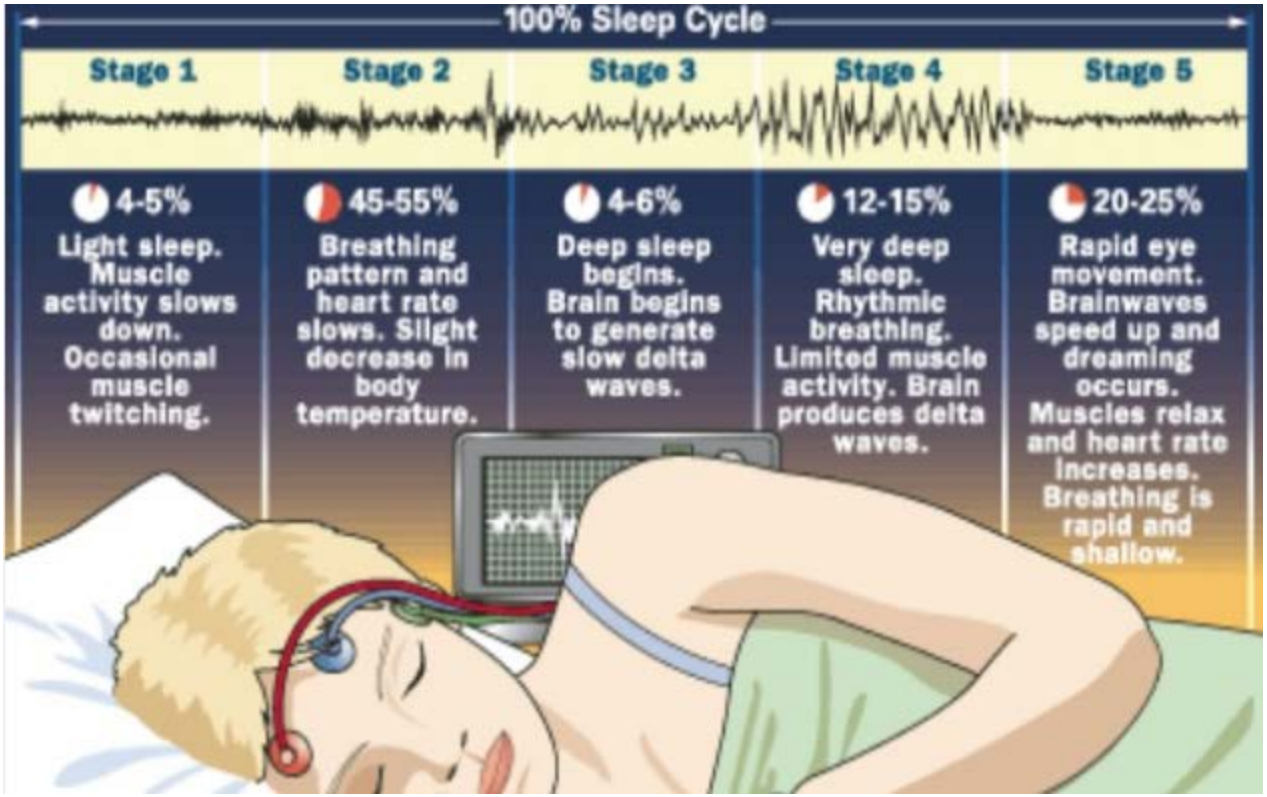
- About 90 minutes after sleep onset NREM - to 1st REM episode of night.
- The order normally being N1 → N2 → N3 → N2 → REM.
- REM latency of 90 min is normal
- Shortening of REM latency frequently occurs with depressive disorders and narcolepsy
- REM period occurs every 90-100min during night.
- 1st REM shortest of 10min; later 15-40min.

Sleep Cycle

- Typically, N3 sleep ----- first third of the night,
- Whereas REM sleep ------- last third of the night.
- This can be helpful clinically as NREM parasomnias typically occur in the first third of the night with the presence of N3 sleep.
- This contrasts with REM sleep behavior disorder (RBD), which typically occurs in the last half of the night.

SLEEP CYCLES




100% Sleep Cycle
Stage 1
Stage 2
Stage 3
Stage 4
Stage 5
4-5%
Light sleep. Muscle activity slows down. Occasional muscle twitching.
45-55%
Breathing pattern and heart rate slows. Slight decrease in body temperature.
4-6%
Deep sleep begins. Brain begins to generate slow delta waves.
12-15%
Very deep sleep. Rhythmic breathing. Limited muscle activity. Brain produces delta waves.
20-25%
Rapid eye movement. Brainwaves speed up and dreaming occurs. Muscles relax and heart rate increases. Breathing is rapid and shallow.

REM SLEEP

- REM sleep, accounts for 20–25% of total sleep time in most human adults.
- This is defined by rapid, relatively low voltage EEG with episodic rapid eye movement and absent EMG activity of all the skeletal muscles .
- EEG resembles waking state.
- The brain is highly active in REM sleep, and overall brain metabolism may be increased as much as 20 per cent
- Heart rate and respiration usually become irregular, which is characteristic of the dream state
- REM sleep is also called *paradoxical sleep because it is a paradox that a person can* still be asleep despite marked activity in the brain

REM Sleep

- During REM sleep, threshold for arousal by environmental stimuli is increased. So by criteria of external arousability, REM sleep is the deepest sleep.
- At the same time a sleeping human is more likely to awake spontaneously from REM sleep. By criterion of internal arousability REM sleep is the lightest stage of sleep.

Why REM sleep?

- The Ontogenetic Hypothesis of REM sleep states that this sleep phase is particularly important to the developing brain, possibly because it provides the neural stimulation that newborns need to form mature neural connections.
- REM sleep is necessary for proper central nervous system development. Supporting this theory is the fact that the amount of REM sleep decreases with age, as well as the data from other species
- A newborn baby spends almost 9 hours a day in REM sleep.
- By the age of five or so, only slightly over two hours is spent in REM.

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Memory Process and the Function of Sleep

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- Zhang hypothesizes that the function of sleep is to process, encode and transfer the data from the temporary memory to the long-term memory

- NREM sleep processes the conscious-related memory (declarative memory), and REM sleep processes the unconscious related memory (procedural memory and spatial memory). The purpose of body paralysis during REM stage is to prevent the sleeper from reacting to the procedural data being processed

Why REM Sleep ?

- There is evidence linking REM sleep with highly synchronised theta rhythm at the hippocampus. Theta rhythm has been linked with encoding of long term memories.
- REM sleep provides a mechanism, allowing memory processing to occur “off line”.
- Each species of mammal could process the information most important for its survival.
- In REM sleep, this information may be accessed again and integrated with past experience to provide an ongoing strategy for behaviour

REM Rebound

- When you are sleep deprived you lose out on two types of sleep, REM and NREM (non-REM).
- After sleep deprivation when allowed to sleep freely, there is earlier initiation and increase frequency and marked lengthening of REM sleep: REM rebound.
- The existence of such rebound suggests that REM sleep is physiologically necessary.
- It also affirms the common belief that dreaming serves some important need.

NREM Sleep

- Most sleep during each night is of the NREM variety
- This sleep is exceedingly restful and is associated with decrease in both peripheral vascular tone and many other vegetative functions of the body.
- There are 10 to 30 per cent decreases in blood pressure, respiratory rate and basal metabolic rate.

- The American Academy of Sleep Medicine (AASM) divides NREM into three stages : N1, N2 and N3, the last of which is also called slow-wave sleep (SWS)

NREM Sleep

- Stage N1 refers to the transition of the brain from alpha waves having a frequency of 8–13 Hz (common in the awake state) to theta waves having a frequency of 4–7 Hz. This stage is sometimes referred to as somnolence or drowsy sleep

- During N1, the subject loses some muscle tone and most conscious awareness of the external environment. Sudden twitches and jerks may be associated with onset of sleep

NREM Sleep

- Stage N2 is characterized by sleep spindles ranging from 11–16 Hz and K-complexes.
- During this stage, muscular activity as measured by EMG decreases, and conscious awareness of the external environment disappears.
- This stage occupies 45–55% of total sleep in adults

NREM Sleep

- Stage N3 (deep or slow-wave sleep) is characterized by the presence of a minimum of 20% delta waves ranging from 0.5–2 Hz.
- Many descriptions still show a stage N3 with 20–50% delta waves and a stage N4 with greater than 50% delta waves; these have been combined as stage N3

- This is the stage in which parasomnias occur.

Why Non REM sleep ?

Energy Conservation

- Brain neurons depend on glycogen for energy. NREM sleep uses much less energy than wakefulness. Some have suggested NREM sleep may provide time to restore our brain's glycogen stores, which are depleted by the demands of wakefulness. Sleeps targets areas of prior neuronal activities.

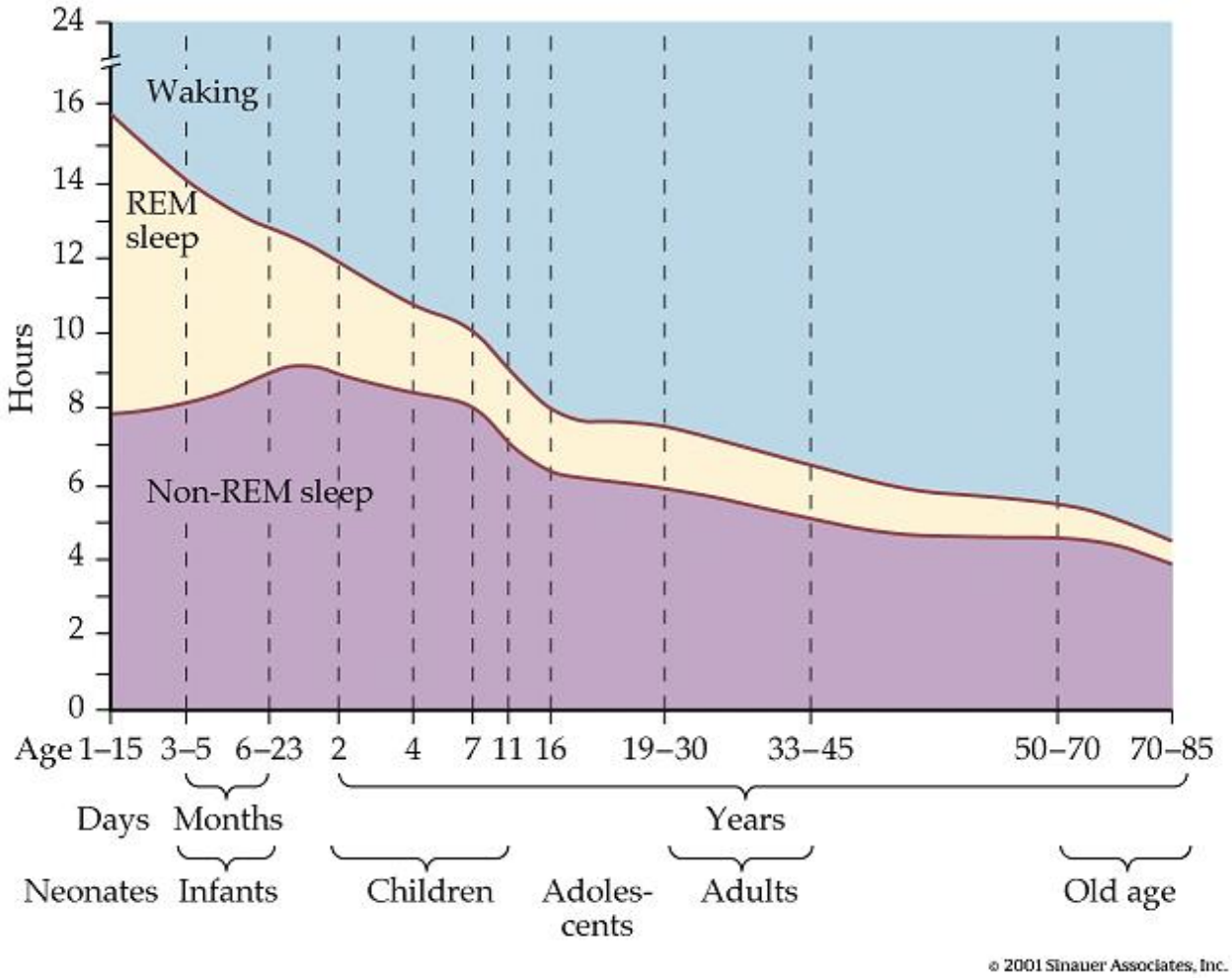
Brain Plasticity

- Synaptic efficiency and efficacy of the brain depends on keeping the synaptic connections refined integrating new neuronal firing patterns. Sleep periodically occurs to allow the brain to do this, thus maintaining brain plasticity.

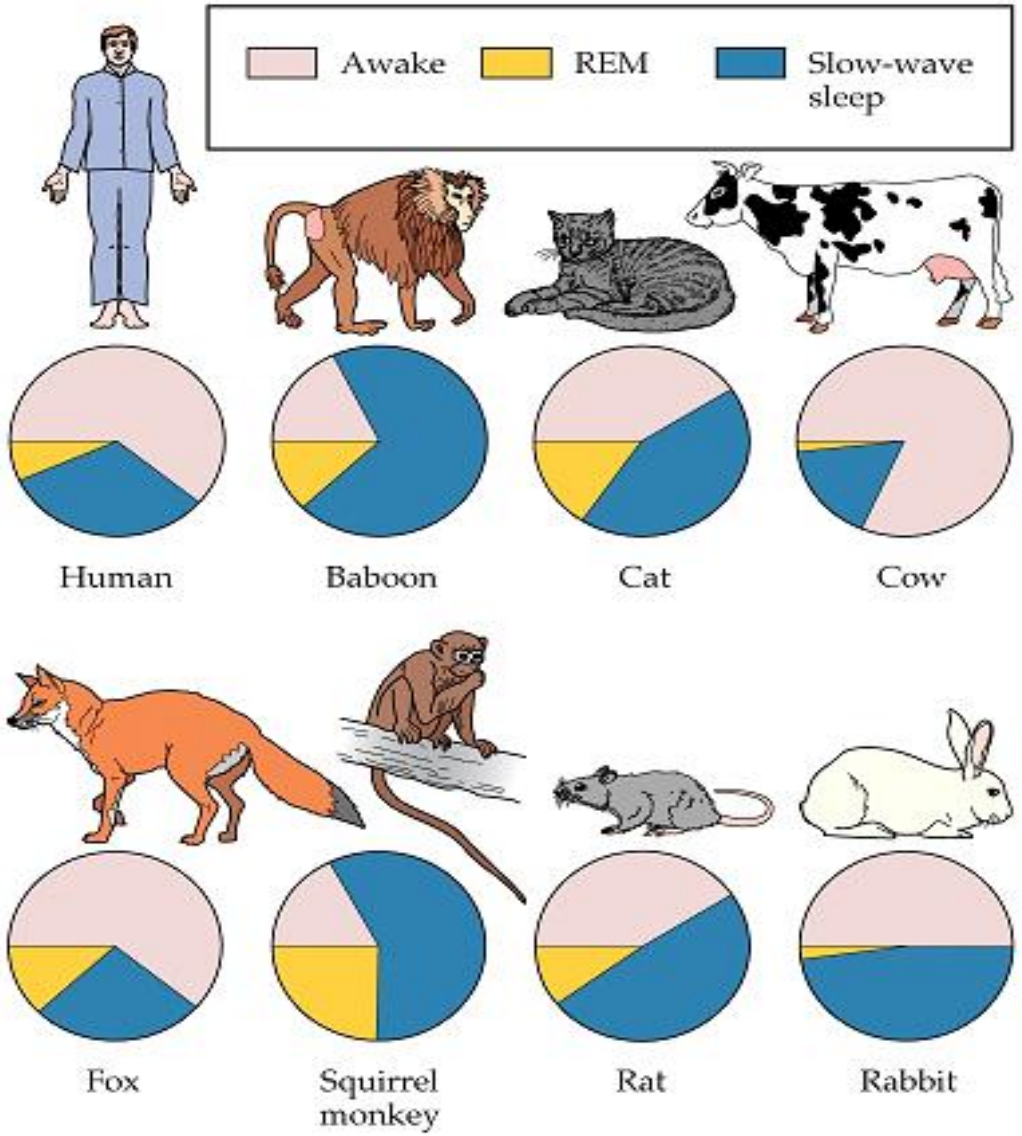
IMMUNE FUNCTION

- Recent studies suggest that sleep may strengthen our immune defenses and insufficient sleep impair them.

LIFE SPAN CHANGES IN SLEEP



COMPARATIVE STUDIES OF SLEEP

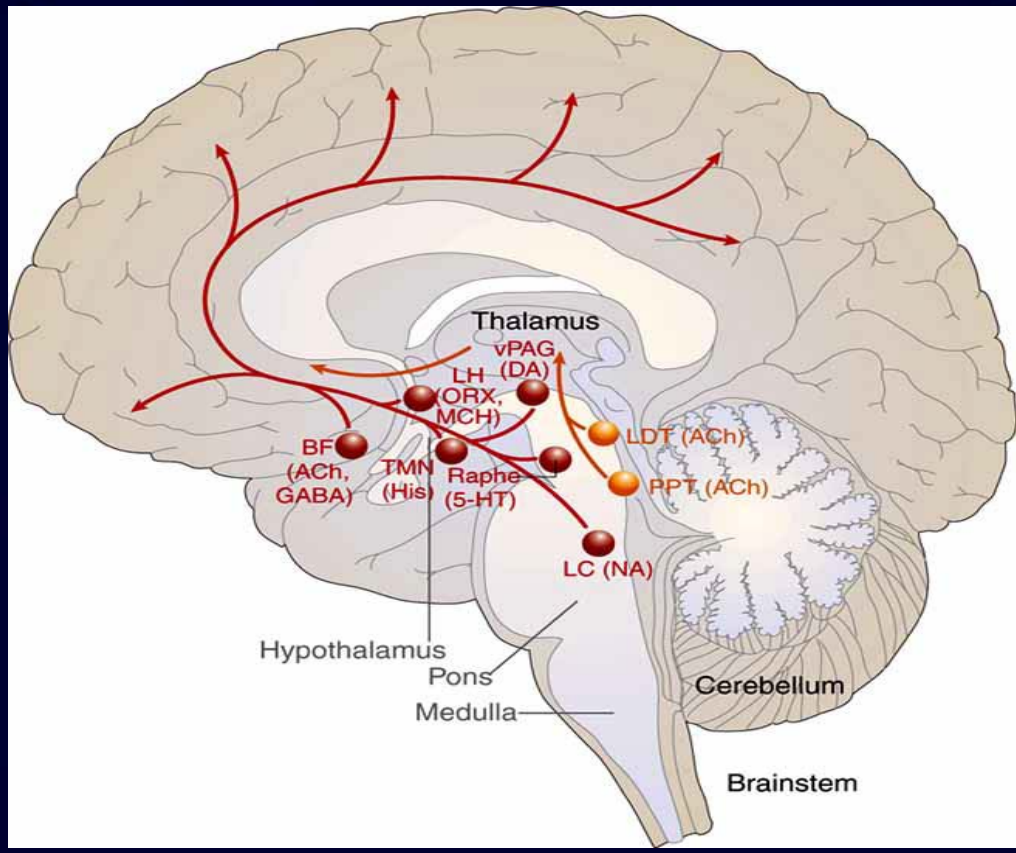


Mechanisms of Sleep

- **Sleep Is Believed to Be Caused by an Active Inhibitory Process.**
- An earlier theory of sleep was that the excitatory areas of the upper brain stem, the *reticular activating system*, simply fatigued during the waking day and became inactive as a result. This was called the *passive theory of sleep*.
- An important experiment changed this view to the current belief that *sleep is caused by an active inhibitory process :* it was discovered that transecting the brain stem at the level of the midpons creates a brain whose cortex never goes to sleep.

Brain Regions Involved in Sleep: Arousal Centers

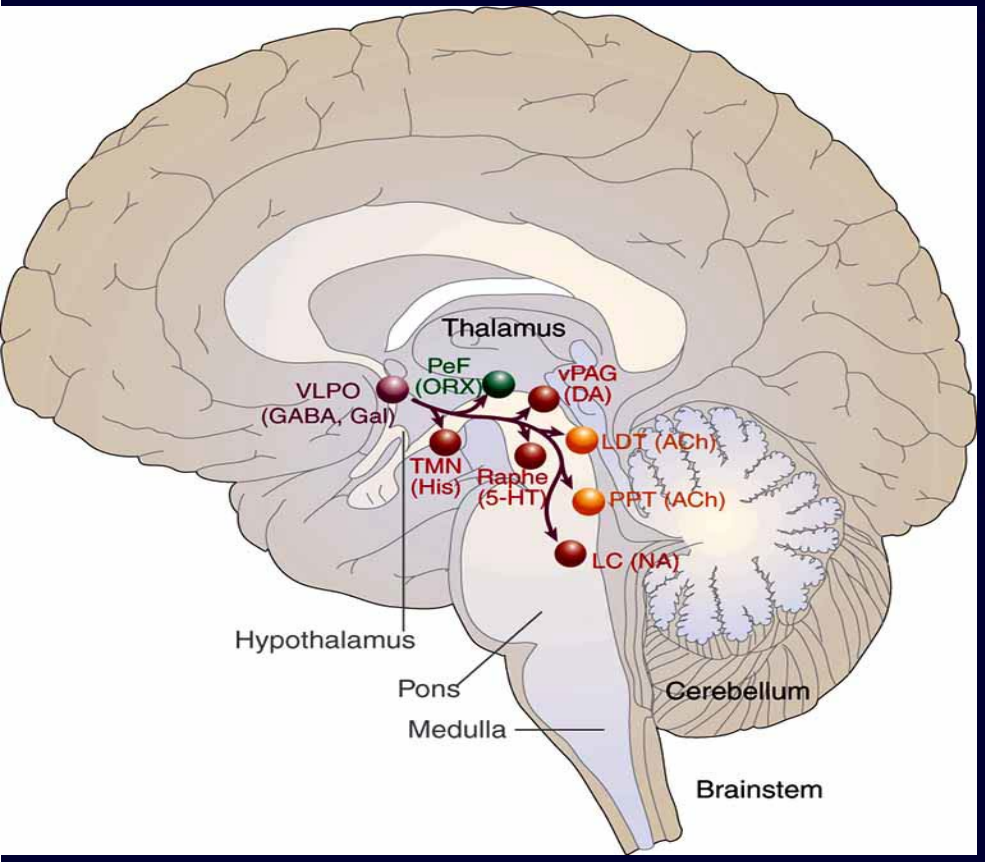
- In the wakeful state, ascending arousal centers in the brainstem, hypothalamus, and basal forebrain excite sensory and motor regions in the cerebral cortex and thalamus[1]
- Arousal centers[1]
  - PPT, LDT
    - Thalamocortical transmission
  - LC, Raphé nuclei, TMN, vPAG
    - Activation of the cerebral cortex
- Also contribute to arousal
  - LHA, BF
- SCN (circadian clock) modulates the balance between periods of high and low sleep propensity[2]



BF=basal forebrain; LC=locus coeruleus; LDT=laterodorsal tegmental nuclei; LH=lateral hypothalamus; PPT=pedunculopontine nuclei; SCN=suprachiasmatic nucleus; TMN=tuberomammillary nucleus; vPAG=A10 cell group.

- Two cholinergic , PPT/LDT neurons are most active during wakefulness and rapid eye movement (REM) sleep and discharge more slowly during non-REM (NREM) sleep.
- Second monoaminergic system: Neurons in these monoaminergic systems have broad action potentials, discharging most rapidly during wakefulness, slowing during NREM sleep, and showing little activity during REM sleep.
- A similar pattern was reported in orexin/hypocretin neurons of the lateral hypothalamus

Brain Regions Involved in Sleep-wake Regulation: VLPO



- VLPO
  - GABA and galanin transmitting
  - Innervates and inhibits arousal centers (eg, TMN, LC) to maintain the sleep state
  - During wakeful state, receives inhibitory signals from arousal centers: TMN, Raphé, LC
- Orexin neurons (PeF)
  - Directly inhibited by VLPO in sleep state
  - During wakeful state, help maintain excitatory signaling in the ascending arousal pathway

LC=locus coeruleus; LDT=laterodorsal tegmental nuclei; PeF=perifornical neurons; PPT=pedunculopontine nuclei; TMN=tuberomammillary nucleus; VLPO=ventrolateral preoptic nucleus; vPAG=A10 cell group.

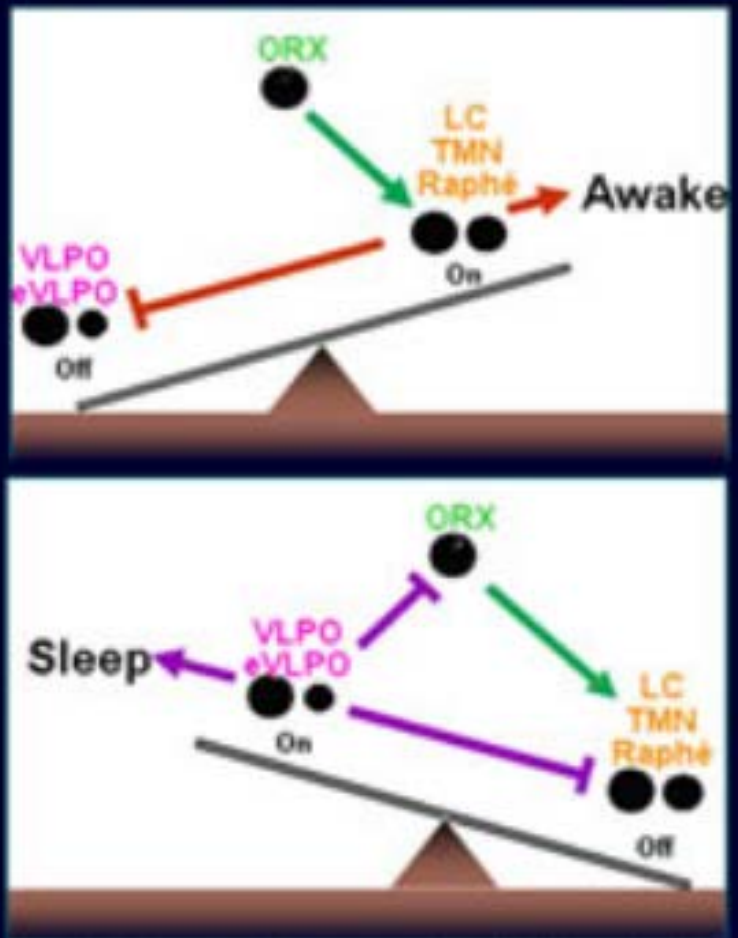
Neurochemical control of sleep-wake states

Neurotransmitter	Location	Action
Acetylcholine	LDT, PPT (pons)	REM, wake
Histamine	TMN (posterior hypothalamus)	Wake
GABA, galanin	VLPO	NREM sleep
Serotonin	Raphe nuclei	Wake, NREM
Norepinephrine	Locus coeruleus	Wake
Hypocretin	Later hypothal	Wake

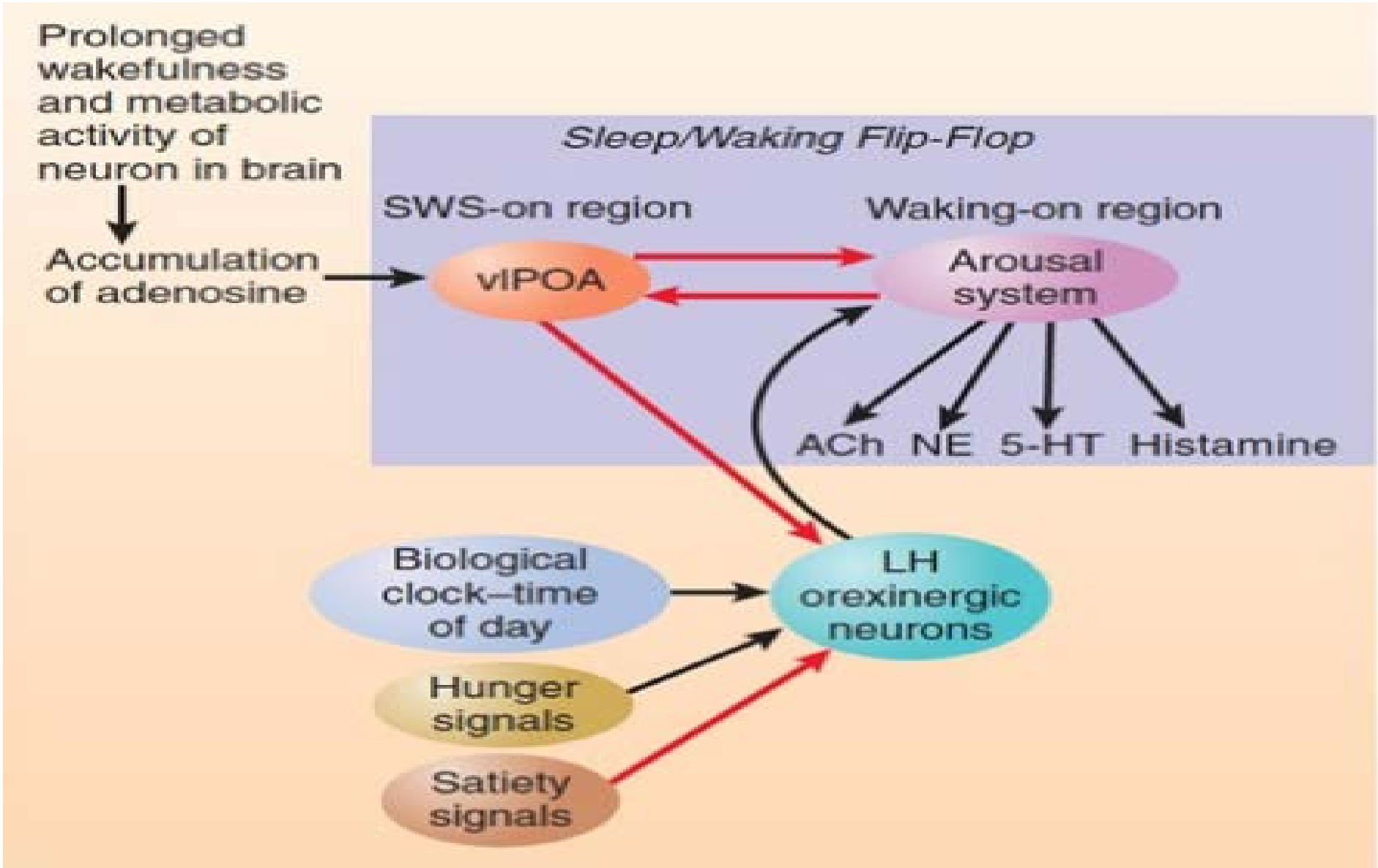
The "flip-flop" sleep switch

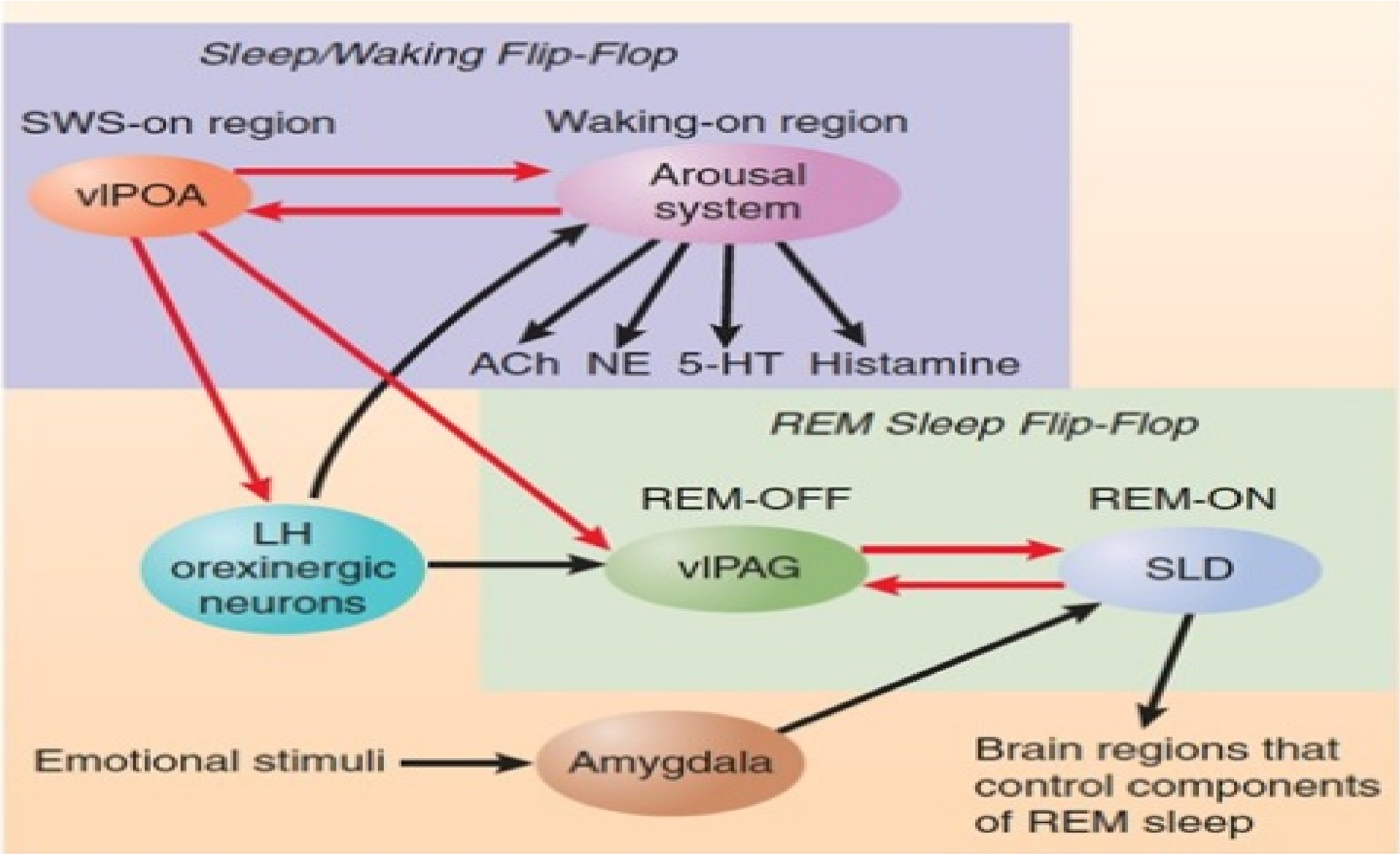
Saper, *Nature* 2005; 437:1257-63

The Sleep Switch: Mutual Inhibition

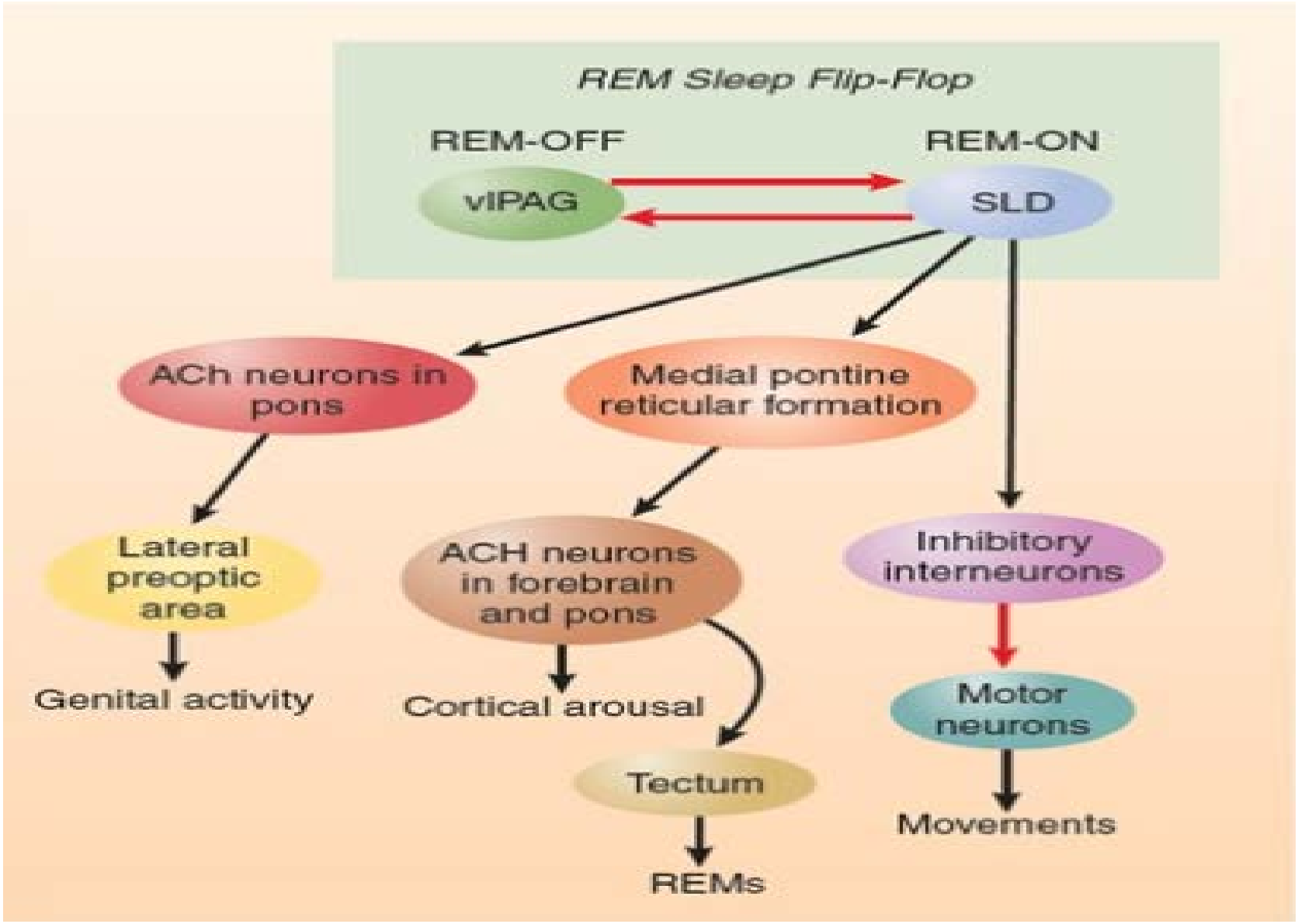


- During wakefulness, activity in arousal nuclei predominate and inhibit the VLPO
- VLPO inhibition relieves its suppression of arousal centers (eg, LC, TMN, Raphé) and orexin (ORX) neurons
- During sleep, VLPO activity predominates over the arousal centers and inhibits them
- VLPO activity inhibits LC, TMN, and Raphé, as well as PeF (ORX) activity

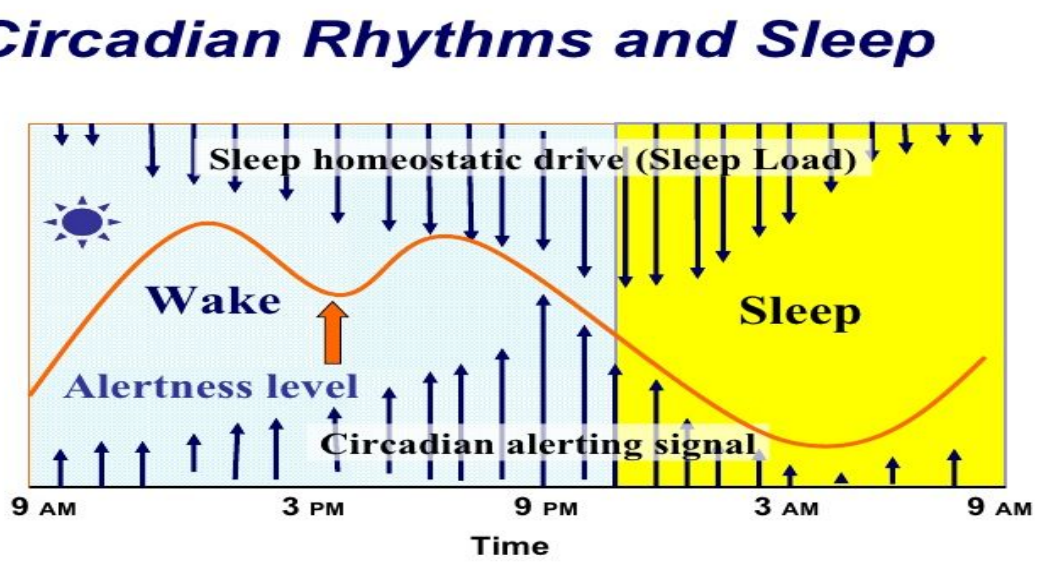

Prolonged wakefulness and metabolic activity of neuron in brain
Accumulation of adenosine
Sleep/Waking Flip-Flop
SWS-on region
Waking-on region
vlPOA
Arousal system
ACh
NE
5-HT
Histamine
Biological clock–time of day
Hunger signals
Satiety signals
LH orexinergic neurons



vlPAG: Ventrolateral periaqueductal gray matter, SLD: sublateralodorsal nucleus


REM Sleep Flip-Flop
REM-OFF
REM-ON
vlPAG
SLD
ACh neurons in pons
Medial pontine reticular formation
Lateral preoptic area
Genital activity
ACH neurons in forebrain and pons
Cortical arousal
Tectum
REMs
Inhibitory interneurons
Motor neurons
Movements

Sleep Regulation



Process S

During periods of wakefulness, glycogen, the body's principal store of energy, is exhausted . As glycogen is broken down into adenosine, extracellular levels of adenosine begin to accumulate in the basal forebrain

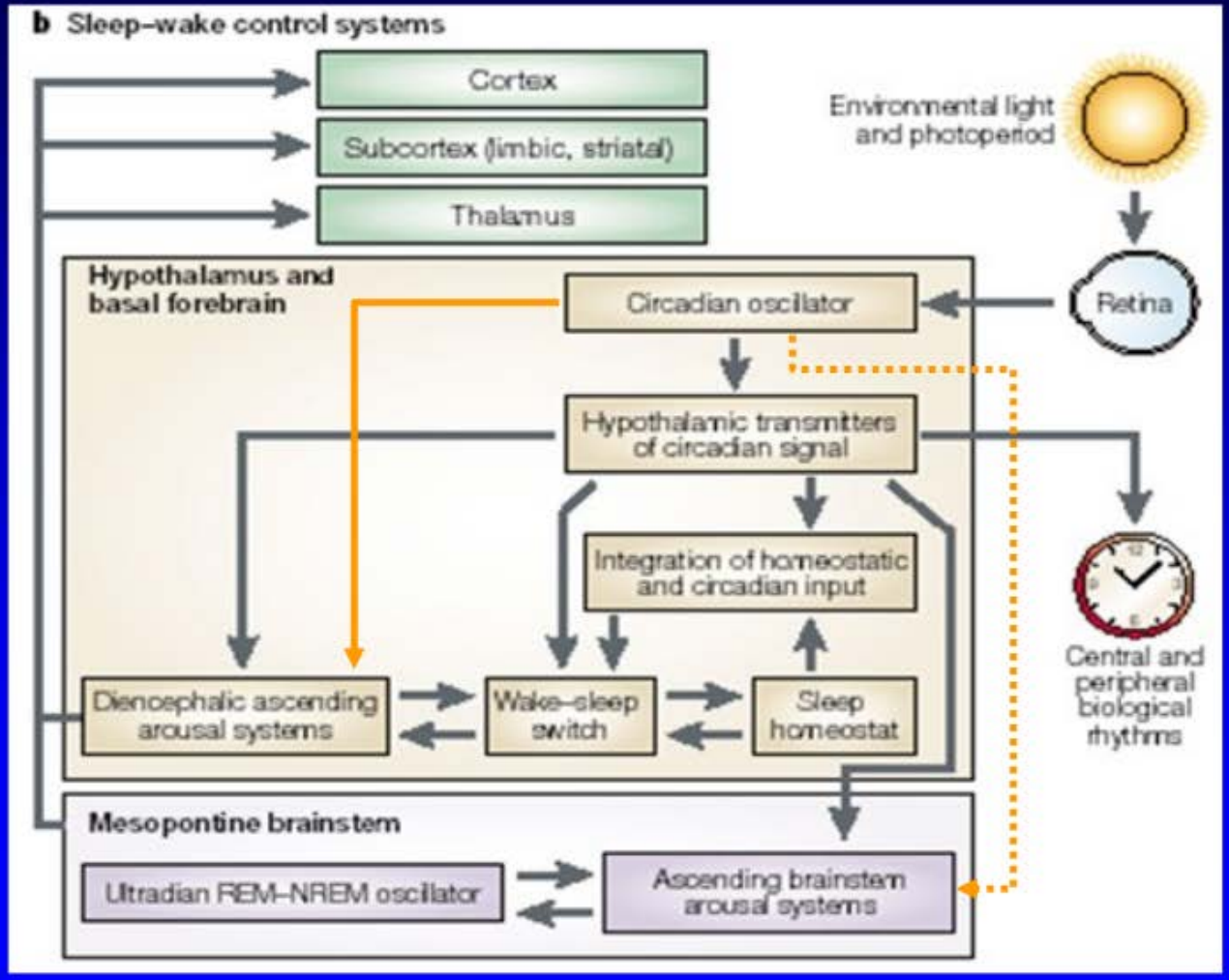
Adenosine also may excite VLPO neurons by disinhibiting GABAergic inputs . Therefore, by inhibiting the basal forebrain arousal system and triggering the VLPO nucleus, adenosine may act as homeostatic regulator of the sleep need.

Circadian rhythms and sleep regulation

- A second component of the sleep-wake regulatory mechanism, which Borbely called Process C, involves circadian influences, The locus of this endogenous circadian pacemaker is the suprachiasmatic nucleus (SCN) of the hypothalamus.
- The SCN, has been called the brain's "master clock".
- Circadian timing, in which neurons fire in a 24-hour cycle, is organized in a hierarchy of tissue-specific structures located throughout the body. These tissue-specific rhythms are coordinated by the SCN based on light input from the outside world during daytime and by melatonin secretion during the dark cycle

Integration of circadian rhythms and sleep-wake states

Pace-Schott and Hobson, 2002



Sleep Disorders

Outline of the *International Classification of Sleep Disorders*, Third Edition

I. Insomnia
   A. Chronic Insomnia Disorder
   B. Short-Term Insomnia Disorder
   C. Other Insomnia Disorder
   D. Isolate Symptoms and Normal Variants
      1. Excessive Time in Bed
      2. Short Sleeper

II. Sleep-Related Breathing Disorders
   A. Obstructive Sleep Apnea Disorders
      1. Obstructive Sleep Apnea, Adult
      2. Obstructive Sleep Apnea Disorder, Pediatric
   B. Central Sleep Apnea Syndromes
      1. Central Sleep Apnea with Cheyne–Stokes Breathing
      2. Central Apnea Due to a Medical Disorder without Cheyne–Stokes Breathing
      3. Central Sleep Apnea Due to High-Altitude Periodic Breathing
      4. Central Sleep Apnea Due to a Medication or Substance
      5. Primary Central Sleep Apnea
      6. Primary Central Sleep Apnea of Infancy
      7. Primary Central Sleep Apnea of Prematurity
      8. Treatment-Emergent Central Sleep Apnea
   C. Sleep-Related Hypoventilation Disorders
      1. Obesity Hypoventilation Syndrome
      2. Congenital Central Alveolar Hypoventilation Syndrome
      3. Late-Onset Central Hypoventilation and Hypothalamic Dysfunction
      4. Idiopathic Central Alveolar Hypoventilation
      5. Sleep-Related Hypoventilation Due to a Medication of Substance
      6. Sleep-Related Hypoventilation Due to a Medical Disorder
   D. Sleep-Related Hypoxemia Disorder
      1. Sleep-Related Hypoxemia
   E. Isolated Symptoms and Normal Variants
      1. Snoring
      2. Catathrenia

III. Central Disorders of Hypersomnolence
   A. Narcolepsy Type 1
   B. Narcolepsy Type 2
   C. Idiopathic Hypersomnia
   D. Kleine–Levin Syndrome
   E. Hypersomnia Due to a Medical Disorder
   F. Hypersomnia Due to a Medication or Substance
   G. Hypersomnia Associated with a Psychiatric Disorder
   H. Insufficient Sleep Syndrome
   I. Isolated Symptoms and Normal Variants
      1. Long Sleeper

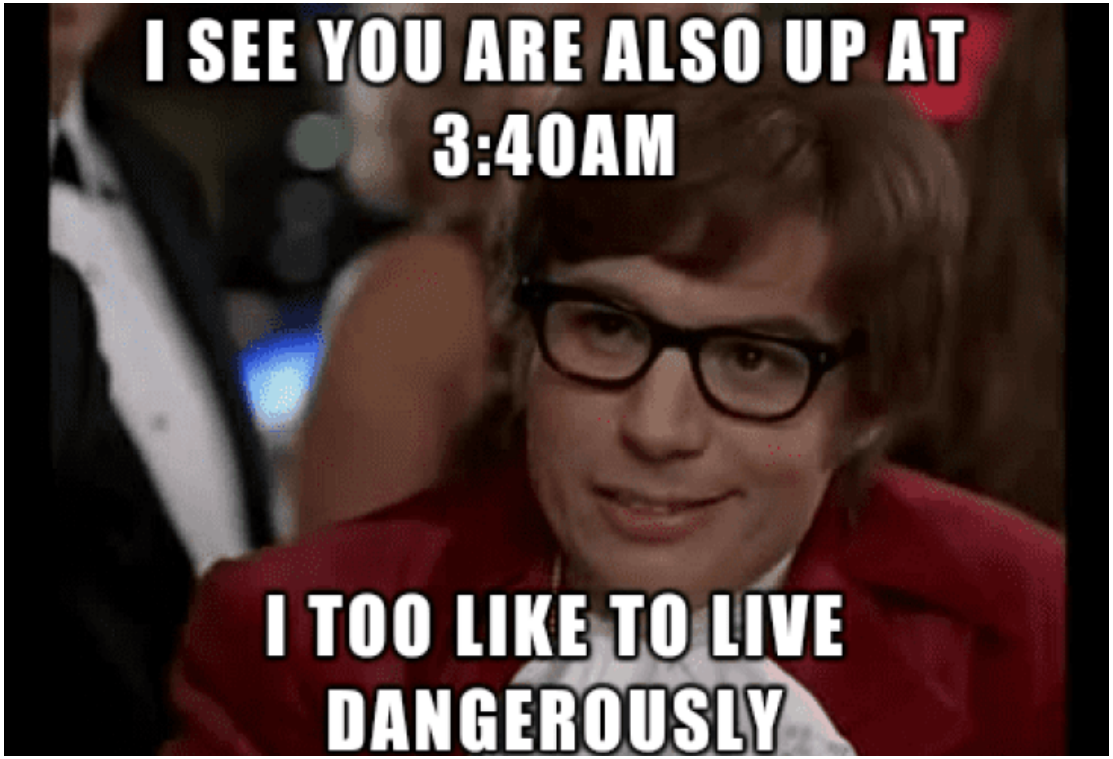
IV. Circadian Rhythm Sleep–Wake Disorders
   A. Delayed Sleep–Wake Phase Disorder
   B. Advanced Sleep–Wake Phase Disorder
   C. Irregular Sleep–Wake Rhythm Disorder
   D. Non–24-Hour Sleep–Wake Rhythm Disorder
   E. Shift Work Disorder
   F. Jet Lag Disorder
   G. Circadian Sleep–Wake Disorder Not Otherwise Specified

V. Parasomnias
   A. NREM-Related Parasomnias
      1. Confusional Arousals
      2. Sleepwalking
      3. Sleep Terrors
      4. Sleep-Related Eating Disorder
   B. REM-Related Parasomnias
      1. REM sleep behavior disorder
      2. Recurrent Isolated Sleep Paralysis
      3. Nightmare Disorder
   C. Other Parasomnias
      1. Exploding Head Syndrome
      2. Sleep-Related Hallucinations
      3. Sleep Enuresis
      4. Parasomnia Due to Medical Disorder
      5. Parasomnia Due to a Medication or Substance
      6. Parasomnia, Unspecified
      7. Isolated Symptoms and Normal Variants
         a. Sleep Talking

VI. Sleep-Related Movement Disorders
   A. Restless Legs Syndrome
   B. Periodic Limb Movement Disorder
   C. Sleep-Related Leg Cramps
   D. Sleep-Related Bruxism
   E. Sleep-Related Rhythmic Movement Disorder
   F. Benign Sleep Myoclonus of Infancy
   G. Propriospinal Myoclonus at Sleep Onset
   H. Sleep-Related Movement Disorder Due to Medical Disorder
   I. Sleep-Related Movement Disorder Due to a Medication or Substance.
   J. Sleep-Related Movement Disorder, Unspecified
   K. Isolated Symptoms and Normal Variants
      1. Excessive Fragmentary Myoclonus
      2. Hypnagogic Foot Tremor and Alternating Leg Muscle Activation
      3. Sleep Starts (Hypnic Jerks)

VII. Other Sleep Disorder

Insomnia



Insomnia Disorder

- A problem with sleep quantity or quality associated with : complaint of trouble initiating sleep or maintaining sleep
- which is associated with daytime consequences
- 3 nights/week, 3 months
- Despite opportunity to sleep
- Co existing medical or mental disorders do not explain predominant complaint of sleep

Insomnia

Occasional, short-term insomnia affects 30% to 50% of the population.

The prevalence of chronic insomnia disorder in industrialized nations is estimated to be at least 5% to 10%.

In medically and psychiatrically ill populations, as well as in older age groups, the prevalence is significantly higher.

Persistent insomnia has been identified in multiple studies as a significant risk factor for the development or relapse of psychiatric disorders, especially mood disorder, alcoholism, pain disorders

Chronic insomnia is associated with numerous adverse effects on function, health, and quality of life.

Treatment of Insomnia

Non-pharmacological

- 1) sleep hygiene
- 2) cognitive therapy
- 3) stimulus control therapy
- 4) sleep restriction
- 5) progressive muscle relaxation.

Pharmacological.

- Dietary supplements
  - melatonin
  - L-tryptophan
- Short acting benzodiazepines
- Z- drugs
  - zolpedium
- Low dose sedative antidepressants
  - mitrazepine
- Long acting medicines
  - flurazepam
  - quazepam
- Other medicines
  - nefazadone
  - qutiepine.

JCSM
Journal of Clinical
Sleep Medicine

SPECIAL ARTICLES

Clinical Practice Guideline for the Pharmacologic Treatment of Chronic Insomnia in Adults: An American Academy of Sleep Medicine Clinical Practice Guideline

Michael J. Sateia, MD[1]; Daniel J. Buysse, MD[2]; Andrew D. Krystal, MD, MS[3]; David N. Neubauer, MD[4]; Jonathan L. Heald, MA[5]

- 1. We suggest that clinicians use suvorexant as a treatment for sleep maintenance insomnia (versus no treatment) in adults. (WEAK)
- 2. We suggest that clinicians use eszopiclone as a treatment for sleep onset and sleep maintenance insomnia (versus no treatment) in adults. (WEAK)
- 3. We suggest that clinicians use zaleplon as a treatment for sleep onset insomnia (versus no treatment) in adults. (WEAK)
- 4. We suggest that clinicians use zolpidem as a treatment for sleep onset and sleep maintenance insomnia (versus no treatment) in adults. (WEAK)
- 5. We suggest that clinicians use triazolam as a treatment for sleep onset insomnia (versus no treatment) in adults. (WEAK)
- 6. We suggest that clinicians use temazepam as a treatment for sleep onset and sleep maintenance insomnia (versus no treatment) in adults. (WEAK)
- 7. We suggest that clinicians use ramelteon as a treatment for sleep onset insomnia (versus no treatment) in adults. (WEAK)
- 8. We suggest that clinicians use doxepin as a treatment for sleep maintenance insomnia (versus no treatment) in adults. (WEAK)

- 9. We suggest that clinicians not use trazodone as a treatment for sleep onset or sleep maintenance insomnia (versus no treatment) in adults. (WEAK)
- 10. We suggest that clinicians not use tiagabine as a treatment for sleep onset or sleep maintenance insomnia (versus no treatment) in adults. (WEAK)
- 11. We suggest that clinicians not use diphenhydramine as a treatment for sleep onset and sleep maintenance insomnia (versus no treatment) in adults.
- (WEAK)
- 12. We suggest that clinicians not use melatonin as a treatment for sleep onset or sleep maintenance insomnia (versus no treatment) in adults. (WEAK)
- 13. We suggest that clinicians not use tryptophan as a treatment for sleep onset or sleep maintenance insomnia (versus no treatment) in adults. (WEAK)
- 14. We suggest that clinicians not use valerian as a treatment for sleep onset or sleep maintenance insomnia (versus no treatment) in adults. (WEAK)

Table 23–2.
Do's and Don'ts for Good Sleep Hygiene

	DO	DON'T
Maintain regular hours of bedtime and arising	✓	
If you are hungry, have a light snack before bedtime	✓	
Maintain a regular exercise schedule	✓	
Give yourself an approximately an hour to wind down before going to bed	✓	
If you are preoccupied or worried about something at bedtime, write it down and deal with it in the morning	✓	
Keep the bedroom cool	✓	
Keep the bedroom dark	✓	
Keep the bedroom quiet	✓	
Take naps		✓
Watch the clock so you know how bad your insomnia actually is		✓
Exercise right before going to bed in order wear yourself out		✓
Watch television in bed when you cannot sleep		✓
Eat a heavy meal before bedtime to help you sleep		✓
Drink coffee in the afternoon and evening		✓
If you cannot sleep, smoke a cigarette		✓
Use alcohol to help in going to sleep		✓
Read in bed when you cannot sleep		✓
Eat in bed		✓
Exercise in bed		✓
Talk on the phone in bed		✓

Hypersomnia

- Excessive amounts of sleep, excessive day time sleepiness, or sometimes both.
- 3-5%
- Mostly secondary to loss of night time sleep

- **causes**

- Insufficient night sleep
  - Unsatisfactory sleep routine
  - Circadian Rhythm sleep disorder
  - Frequent parasomnias
  - Chronic physical illness
  - Psychiatric disorders
- Pathological sleep
  - Narcolepsy
  - Obstructive sleep apnea
  - CNS diseases
  - Drug effects
  - Kleine-Levin Syndrome.

Narcolepsy

- Episodes of irresistible sleepiness leading to 10-20 minutes of sleep
- Epidemiology: age of onset: 10-20years, prevalence : 0.02-0.16%
- after which person is fresh
- Occurs almost daily and in inappropriate times (talking, eating , driving or during sex)
- Other Features:

◦ Cataplexy – sudden loss of muscle tone (associated

with intense emotion & the sudden onset of REM sleep)

◦ Sleep paralysis

◦ Hypnagogic & hypnopompic hallucinations

Treatments:

- Follow regular routine, Planned naps, Avoid fatigue as it provokes catalepsy
- Stimulants, Antidepressants for cataplexy and sleep paralysis

Circadian Rhythm Sleep Disorder

Jet lag type

- Symptoms include varying degrees of difficulty in initiating or maintain sleep, daytime fatigue, decrements in daytime alertness and performance.
- Resolves without treatment after 2-7 days.

Delayed sleep phase type

- Late appearance of sleep(around 2a.m) but normal total sleep time and architecture which lead to sleep-onset insomnia and difficulty awakening at desired time.

Shift work type

- Symptoms of insomnia or excessive sleepiness.
- Adaptation takes 1-2 weeks

Circadian Rhythm Sleep Disorder Management

- 1) General measures.
  - education about sleep nature
  - establishing good sleep habits
  - regular sleep and meal times
- 2) Chronotherapy
  - establish regular waking time.
- 3) Medication.
  - short-acting BdZs
  - melatonin.

Restless leg syndrome

- Distressing & painful condition which can result in severe insomnia and periodic limb movement during sleep.
- Prevalence 10%, M=F
- Exacerbated by caffeine, fatigue or stress
- Sometimes associated with anemia, Vit B12 def
- Treatment: correct def, clonazepam,

dopamine agonists, L Dopa, Pramipexole, Ropinirole

Nightmares

- Awakening from REM sleep to full consciousness with detailed dream recall.
- Common in children 5-6years
- Stimulated by frightening experience during day.
- Frequent nightmares occur during period of anxiety.

- **Causes :** PTSD, Fever, Psychotropic drugs, Alcohol detoxification.

REM SLEEP BEHAVIOR DISORDER

- Characterized by failure to exhibit muscle paralysis during REM sleep
- Appears to be neurodegenerative disorder with some possible genetic component
  - Often associated with other neurodegenerative disorders, such as Parkinson's disease
- Usually treated with clonazepam, a benzodiazepine

Night Terrors

- Less common then nightmare. More familial
- Begins and usually ends in childhood
- Few hours after going to sleep. Within stage 3-4 NREM
- Sits up and appears terrified, screams and appears confused
- Marked increased in heart rate & resp rate
- Slowly settles and returns to normal calm
- Little or no dream recall.
- Prevalence : children 3%, adults 1% M>F

Somnambulism

- Automatism that occurs during deep NREM sleep usually in early part of night.
- 17% in childhood, 4-10% in adults. Familial
- sits up & makes repetitive movements or walk with eyes open, does not respond to questions
- Lasts for few seconds to minutes
- Management: Reassurance, Protect patient from harm, Relaxation techniques and minimization of stressors,
- Medications: small night dose of BDZ or AD

Sleep disturbances in MDD

- Problems with sleep, which could include insomnia or hypersomnia, is one of the diagnostic criteria for MDD . As such, it is not surprising that some type of sleep difficulty occurs in as many of 90% of MDD patients.

- Patients with MDD also appear to have alterations in their sleep stages that are evident with polysomnography. These include: shortened latency to the onset of REM sleep (REM latency); a longer duration of the first REM period; and decreased amount of slow-wave sleep.

Sleep disturbances in MDD

- Poor sleep appears to have an adverse impact on the course of MDD. Perhaps the most important and most concerning of these is that sleep disturbance appears to increase the risks of suicidal ideation, suicide attempts, and completed suicide.

- To date, at least 32 studies (10 – children and adolescents, 22 – adults)have identified that sleep disturbance is linked to suicidal ideation or completed suicide.

Sleep disturbances in MDD

- MDD patients with poor sleep have slower treatment response and lower remission rates than those without sleep disturbance.

- Greater sleep disturbance is also independently correlated (independent of depression severity) with poorer quality of life in those with MDD

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Interventions for Sleep Disturbance in Bipolar Disorder

Allison G. Harvey, PhD, Kate A. Kaplan, MA, and Adriane Soehner, MA

- Sleep disturbances are strongly coupled with inter-episode dysfunction and symptom worsening in bipolar disorder.
- Experimental studies suggest that sleep deprivation can trigger manic relapse.

- The clinical management of the sleep disturbances experienced by bipolar patients, including insomnia, hypersomnia delayed sleep phase and irregular sleep-wake schedule, may include medication approaches, psychological interventions, light therapies and sleep deprivation.
- Psychological interventions, are advantageous in that they are low in side effects, may be preferred by patients, are durable and have no abuse potential

Alzheimer's Disease

- Sleep disorders (SD) in patients with Alzheimer's disease (AD) are among the behavioral disorders that most interfere in the quality of life of the patient and of the caregiver.
- Negative repercussions on cognition, functionality, and behavior of these patients.
- Increased risk of institutionalization
- 40% of the patients with AD have some SD

- The factors that contribute : neuronal loss, atrophy of the suprachiasmatic nucleus of the hypothalamus, and in reduction of cholinergic activity, since acetylcholine participates in REM sleep.

Alzheimer's Disease

- In AD, the most common symptoms related to SD are perambulation, confusion, and nocturnal awakening, besides sleepiness during the day and inversion of the sleep-wake cycle, with reports of night waking being the most stressful aspect for caregivers

- Treatment

Sleep apnea


WHY IS IT THE ONE WHO
SNORES FALLS ASLEEP
FIRST???
ZZZZZZZZZZZZ


SOX

Sleep Apnea

- Sleep apneas are divided into:

- Central sleep apnea: neural drive to all respiratory muscles is abolished

- Obstructive sleep apnea: airflow ceases despite continuing respiratory drive because of occlusion of the oropharyngeal airway

- Occlusion of the oropharyngeal airway results in progressive asphyxia until there is a brief arousal from sleep, whereupon airway patency is restored and airflow resumes.
- The patient then returns to sleep and the process is repeated, up to 300-400 x per night – sleep becomes fragmented.

Risk factors for OSA

- Obesity
- Age
- Male gender
- Positive family history of OSAHS
- Alcohol consumption before bedtime
- Sedatives
- Craniofacial anomalies
- Hypothyroidism

Symptoms- OSA

Day

- Excessive daytime sleepiness
- Unrefreshing sleep
- Memory disturbances
- Morning headache
- Depression

Night

- snoring
- apneas
- choking, gasping
- arousals
- sweating
- dry mouth
- palpitation
- Frequent micturation

Diagnosis

Excessive daytime sleepiness	+	Obstructive breathing events (more than 5 per hour of sleep)

or

Two or more of the following:
1. Choking or gasping during sleep
2. Recurrent awakenings from sleep
3. Unrefreshing sleep
4. Daytime fatigue
5. Impaired concentration

Clinical: typical patient is male, 30-60y,
snores, has daytime sleepiness, nocturnal choking or gasping,
witnessed apneas during sleep, moderate obesity,
large neck circumference
and mild to moderate hypertension

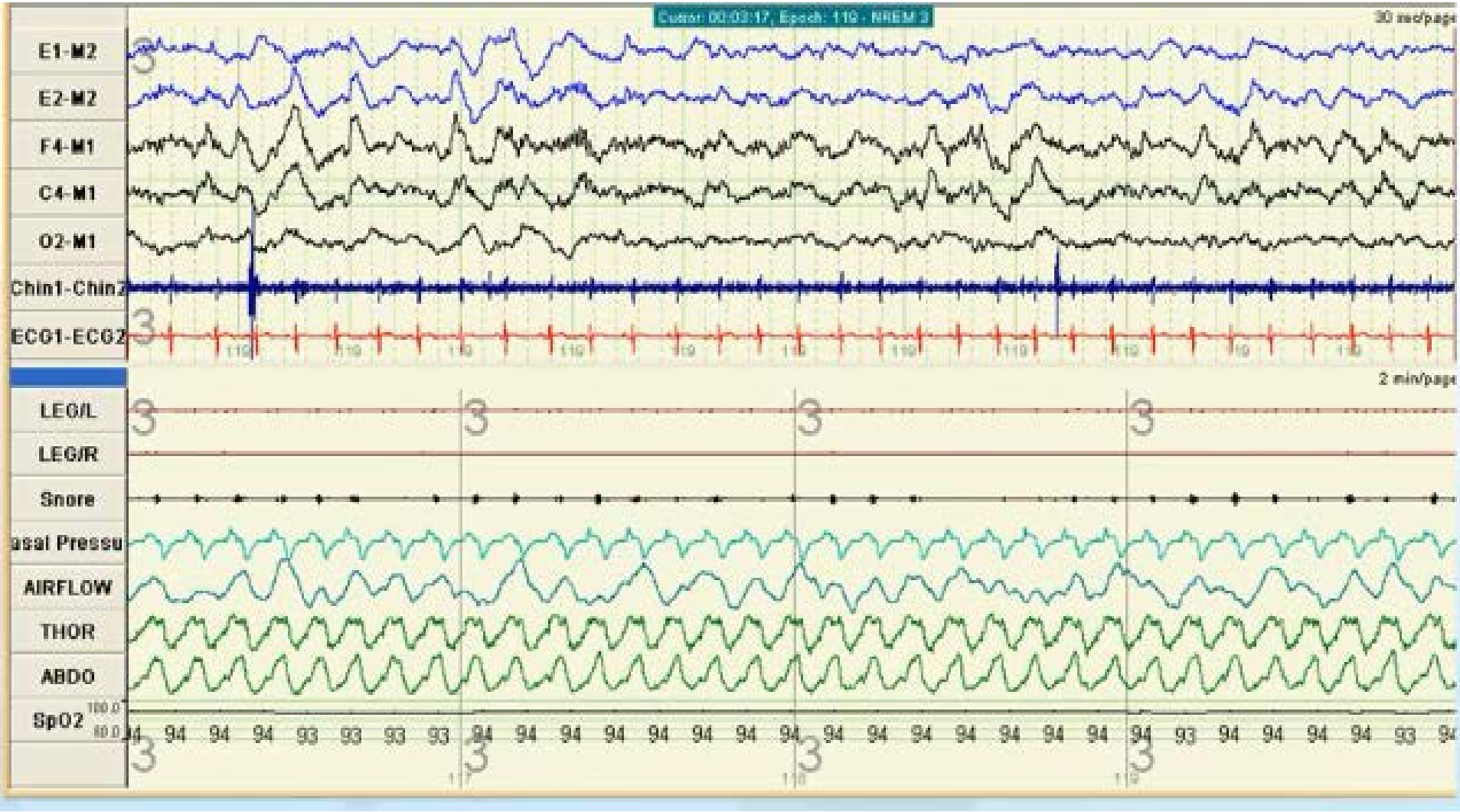
Apnea-hypopnea index (AHI): - Numbers of apneas and hypopneas/ 1 sleeping hour

Severity of sleep related breathing disorders:

- Normal: AHI<5/h
- Mild: AHI: 5-15/h
- Moderate: AHI:16-30/h
- Sever: AHI>30/h

Polysomnography montages

- EEG F4-M1, EEG C4-M1, EEG O2-M1, EOG-L(E1), EOG-R(E2), EMG (chin)
- AIRFLOW, both Thermal and Nasal Pressure
- THOR EFFORT, ABDO EFFORT
- SpO2,
- ECG,
- LEG(L), LEG(R)
- SOUND, POSITION
- CPAP pressure and flow
- Optional: additional EEG, dB meters, temperature, blood pressure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F4-M1
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O2-M1
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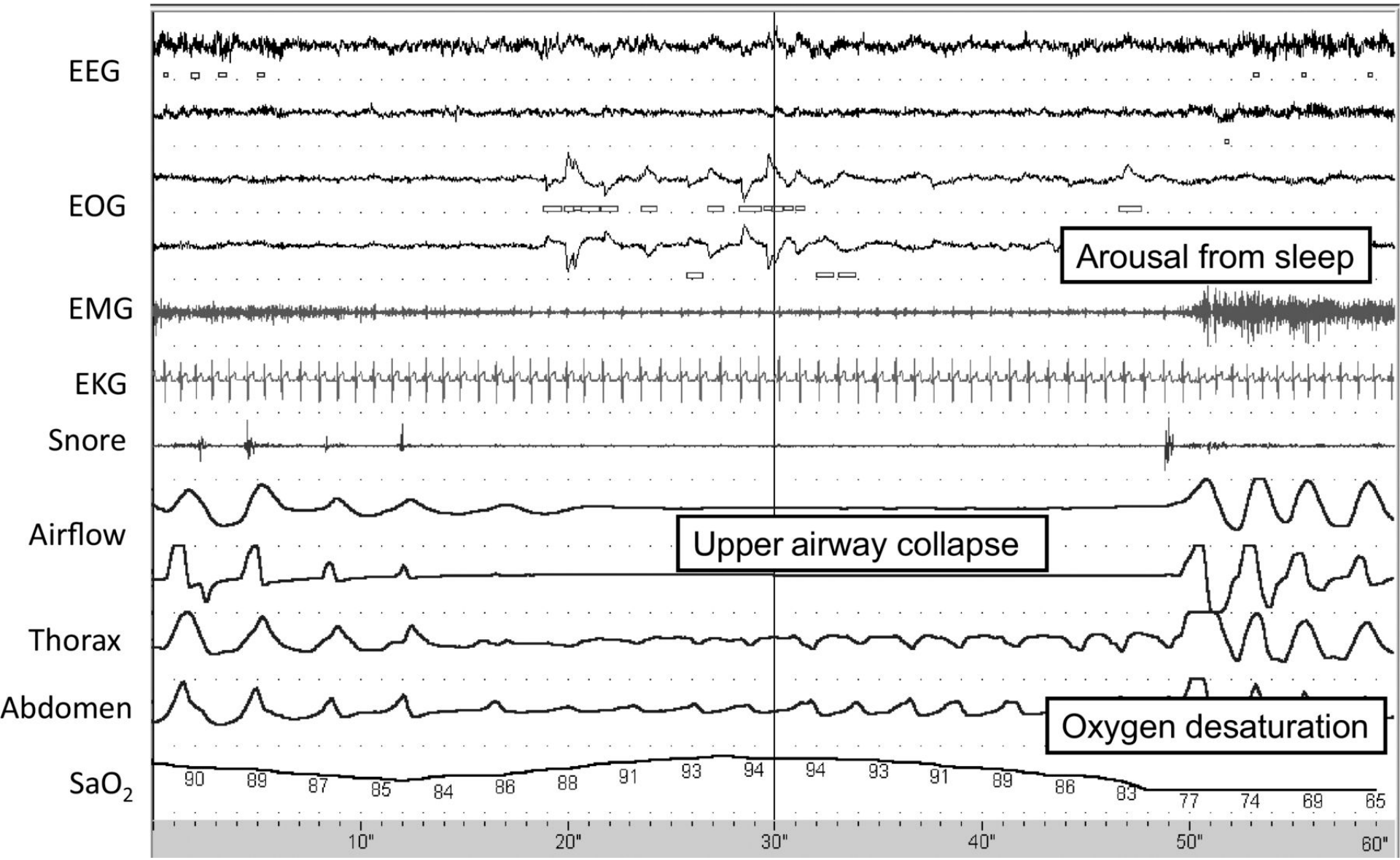
EEG
EOG
EMG
EKG
Snore
Airflow
Thorax
Abdomen
SaO2
Arousal from sleep
Upper airway collapse
Oxygen desaturation
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10"
20"
30"
40"
50"
60"

Treatment of OSA

- Weight loss, alcohol withdrawal
- Position training
- Nasal, pharyngeal surgery (UPPP)
- Oral appliances
- Treatment of OSAHS: nasal CPAP

Thank You