22 -years legacy teaching collaborative care

SEC Sleep Education Consortium

The 22th Annual Dental Sleep Conference: Transformative Advances in Dental Sleep Medicine: Leading Innovations from the Womb to the Tomb

April 23th-25th, 2026
Greenway Plaza Hilton DoubleTree,
Houston, Texas

The SEC web page for downloading the Syllabus

www.SleepEducation.net

Password = SEC26InfoCloud

WiFi = Hilton Meetings Password = hougwstandard

1

House keeping issues

- There is a break with food during the morning and again in the afternoon sessions.
- Lunch will be served in the same room as the breaks.
- Please talk with the vendors. They help support the events and have clinically relevant information
- CME Opportunities: Sleep Education Consortium is partnering with Learner+ to offer clinicians access to an AI-powered reflective portfolio that rewards AMA PRA Category 1 Credits™ CME credits. You MUST digitally provide reflections to the content in order to obtain CME / CE credits. You do not get credits just by attending. You will have 16 specified reflection points / opportunities to provide your input (reflections) and obtain CME credits.
- You should have already signed up for the Learner+ platform. The instructions were sent via an email last week and again this week.

2

Financial Relationship Disclosure

☐ **No,** I do not have a financial interest, arrangement, or affiliation with a corporate organization offering financial support or grant monies for or related to the content of my presentation.

☒ **Yes,** I do have a financial interest, arrangement, or affiliation with a corporate organization offering financial support or grant monies for or related to the content of my presentation..

Harmony Biosciences : Research funding for medication trial

Jazz Pharmaceuticals: Research funding for medication trial

Avadel Pharmaceuticals: Research funding for medication trial, Speaker Honorarium

SleepArchiTx: Advisory Board

REST Technologies, Inc: Owner, Developing Sleep Tracking Tools, Owner

Ely Lilly - Speaker Honorarium

3

Example of the CME / CE process during the conference

Official Learner+ Champion | SEC 2026 Sleep Education Consortium

Sleep Education Consortium (SEC) partners with Learner+, a clinician-centric reflective learning platform that rewards CME/CE credits to busy clinicians anytime and anywhere learning happens. Learn more about how you can **reflect to unlock credits** below. View CME Credit Info

REFLECT NOW

https://champions.learner.plus/sec/

Medications to treat OSA

What inspired you to reflect?

Pick the context and a clinically relevant concept or phrase that inspired you to reflect.

Reflective Learning Moment

Medications to treat OSA

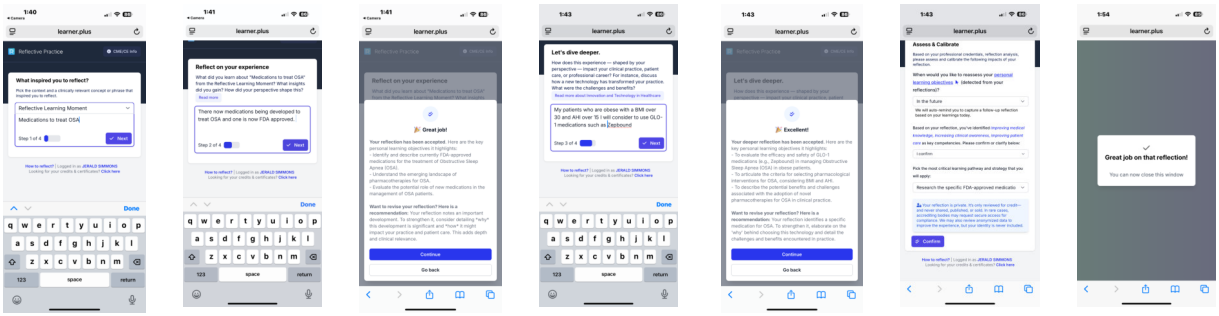
Step 1 of 4

Next

4

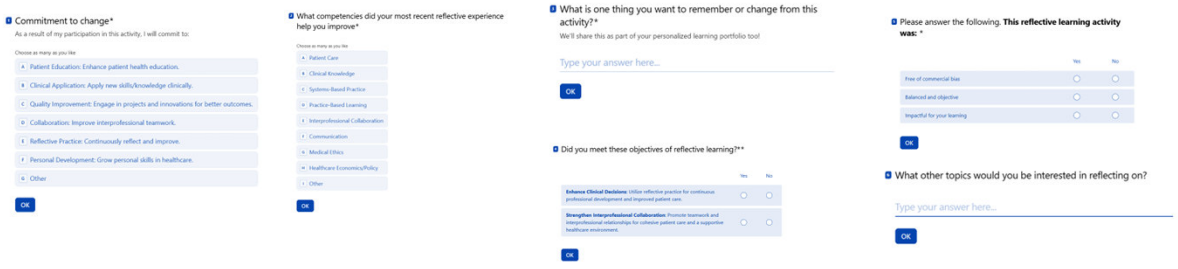
Steps to go through to reflect to get CME / CE credits

1 2 3 4 5 6 7



5

At the end of the conference, you can claim your certificate. You will first be asked a series of questions.



6

Reflective Practice

CERTIFICATE OF COMPLETION

JOINTLY ACCREDITED PROVIDER™ INTERPROFESSIONAL CONTINUING EDUCATION

In support of improving patient care, this activity has been planned and implemented by CME Outfitters.com, LLC and SCAD Group Inc. CME Outfitters, LLC is jointly accredited by the Accreditation Council for Continuing Medical Education (ACCME), the Accreditation Council for Pharmacy Education (ACPE), and the American Nurses Credentialing Center (ANCC), to provide continuing education for the healthcare team.

IPCE CREDIT™

CME Outfitters, LLC, certifies that

JERALD SIMMONS

has participated in the Internet Searching and Learning activity titled **Reflective Practice**

on Mon Apr 20 2026

and is awarded 21 AMA PRA Category 1 Credit(s)™ for completing this activity 21 time(s).

CME Outfitters, LLC designates this Internet Searching and Learning activity for a maximum of 1.0 *AMA PRA Category 1 Credits™*. Physicians should claim only the credit commensurate with the extent of their participation in the activity.

JOINT PROVIDERSHIP

SCOTT J. HERSHMAN, MD, FACEHP, CHCP
Senior Director, Accreditation & Joint Providership
CME Outfitters, LLC.

Mon Apr 20 2026
PRINTED DATE

7

The Physiology of Sleep and its Links to ADD/ADHD, Sleepwalking, Daytime Sleepiness and More

Jerald H. Simmons, M.D.

Director, Comprehensive Sleep Medicine Associates
Director, Sleep Education Consortium

www.CSMA.clinic

8

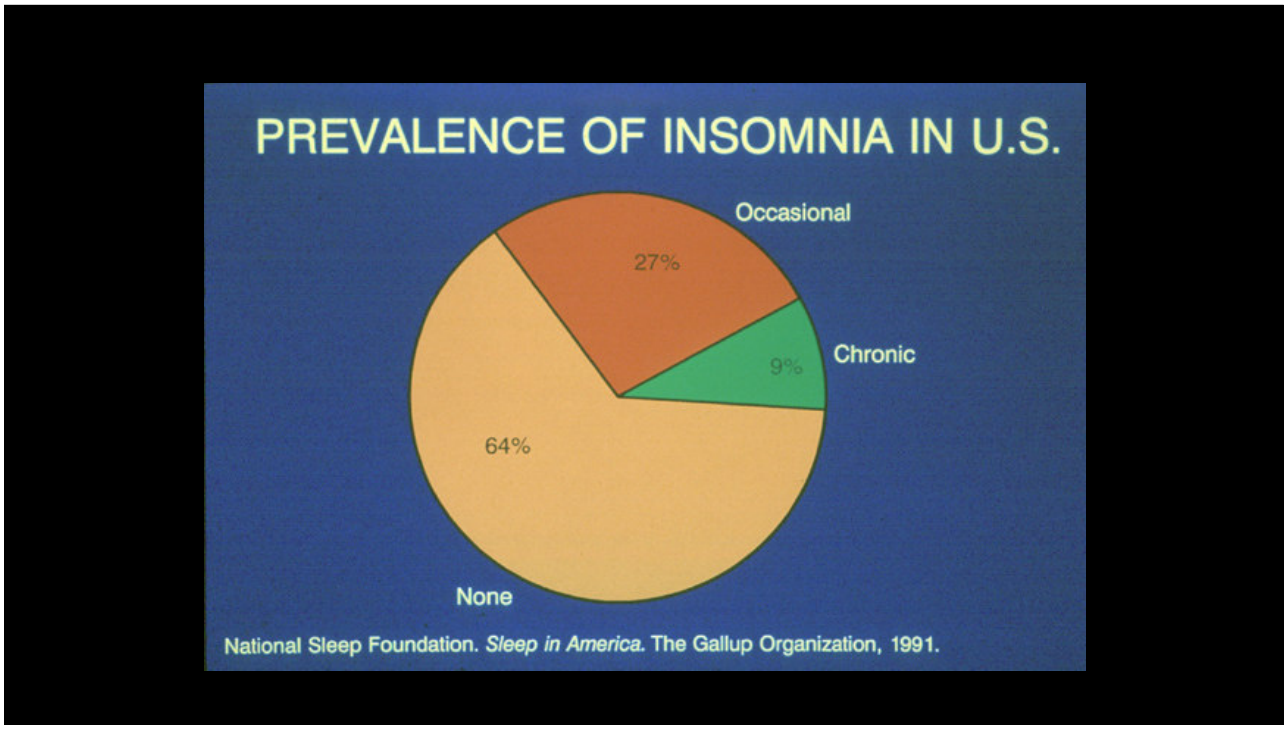
Excessive Daytime Sleepiness

9

A 1997 National Sleep Foundation Gallup Survey found that:

- **32% of the adult American population have Excessive Daytime Sleepiness (EDS).**
- **Only 17% of those with daytime sleepiness have consulted a physician about their daytime sleepiness**

10



11

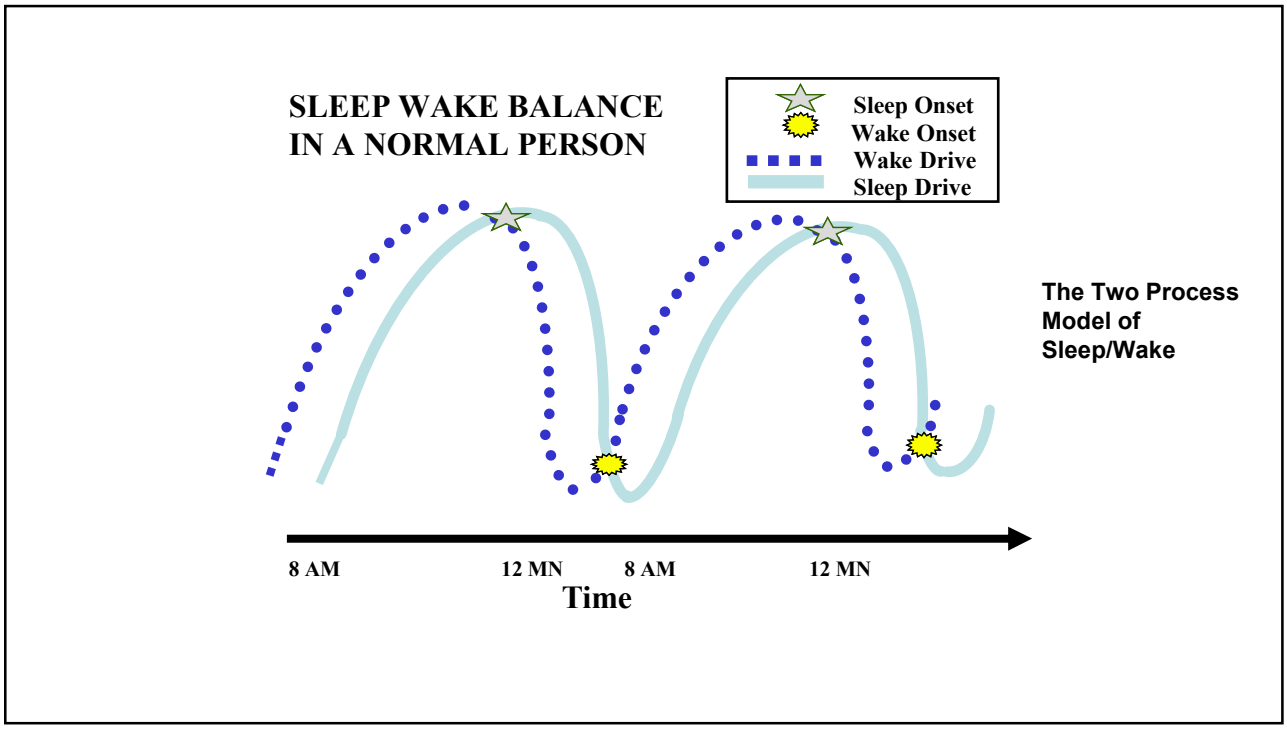
The Result of Drowsy Driving

12

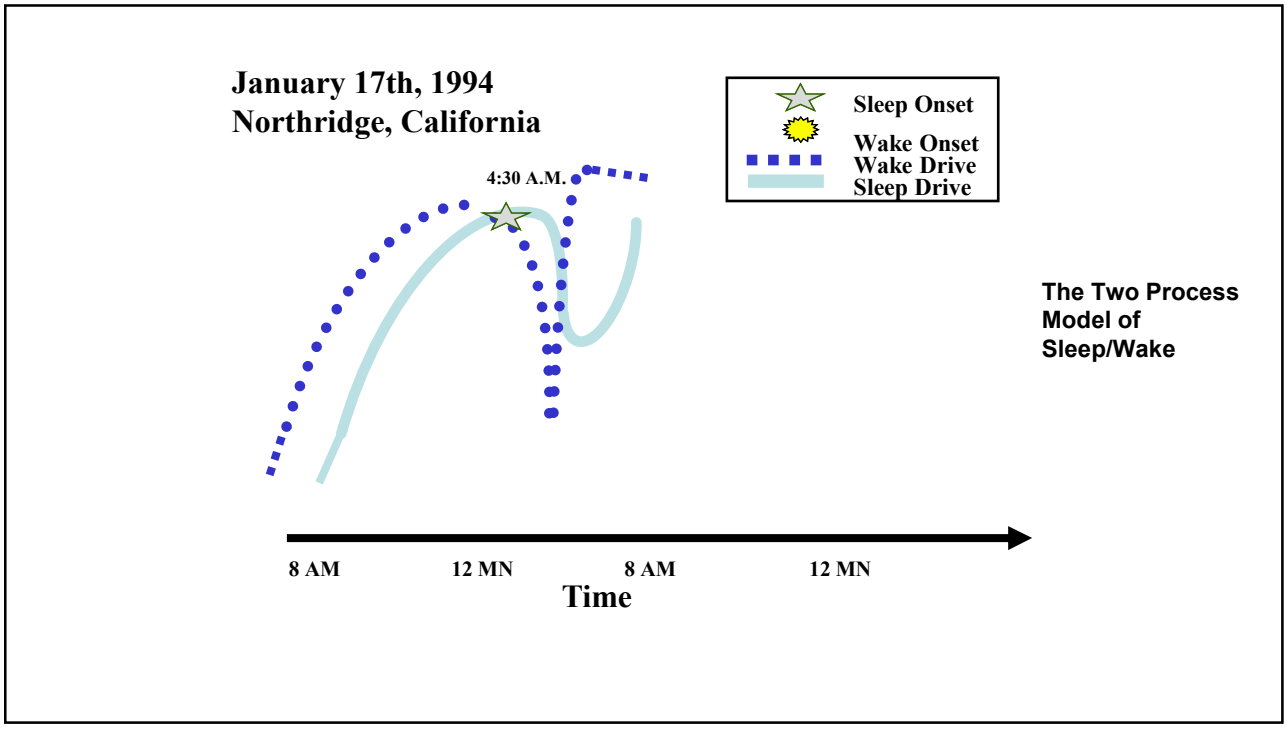
IT IS IMPORTANT TO REALIZE THAT SLEEP AFFECTS DAYTIME FUNCTIONING AND DAYTIME FUNCTIONING CAN AFFECT SLEEP

13

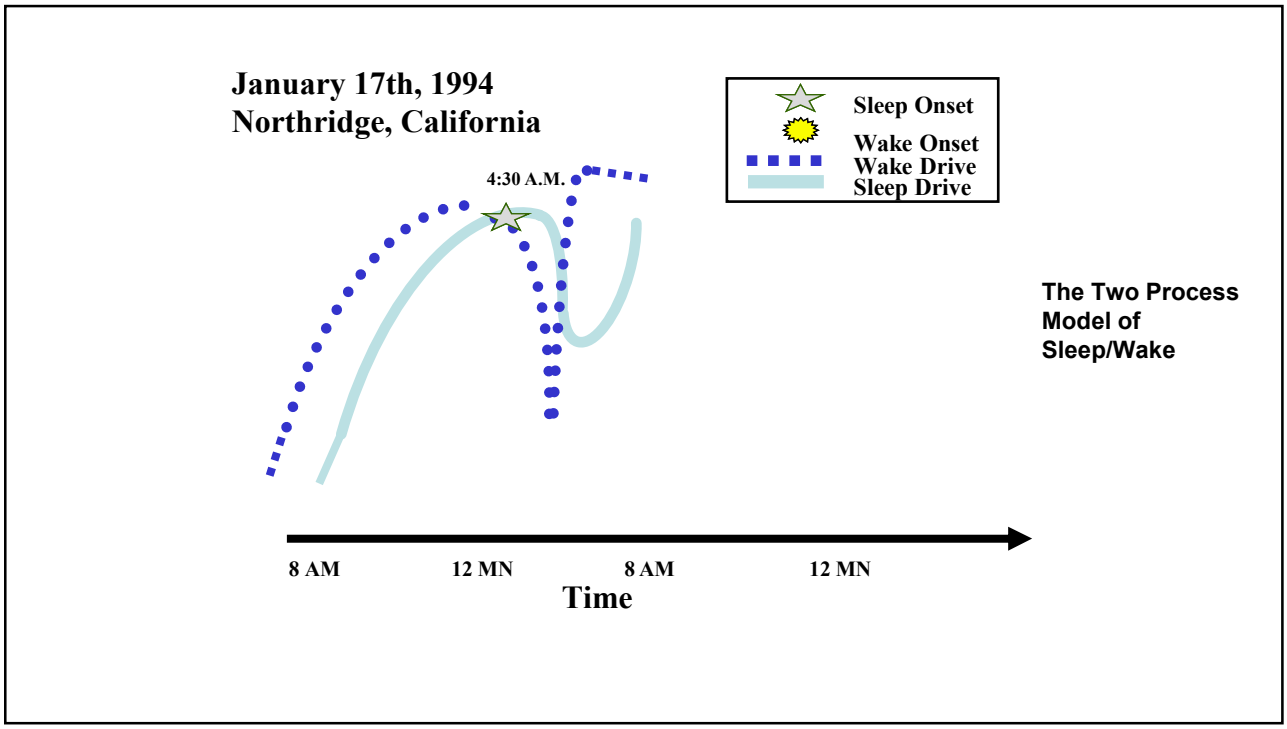
14


SLEEP WAKE BALANCE
IN A NORMAL PERSON
Sleep Onset
Wake Onset
Wake Drive
Sleep Drive
8 AM
12 MN
8 AM
12 MN
Time
The Two Process
Model of
Sleep/Wake

15


January 17th, 1994
Northridge, California
4:30 A.M.
Sleep Onset
Wake Onset
Wake Drive
Sleep Drive
8 AM
12 MN
8 AM
12 MN
Time
The Two Process
Model of
Sleep/Wake

16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12 MN
Time
The Two Process Model of Sleep/Wake

17


The Two Process Model of Sleep/Wake

18

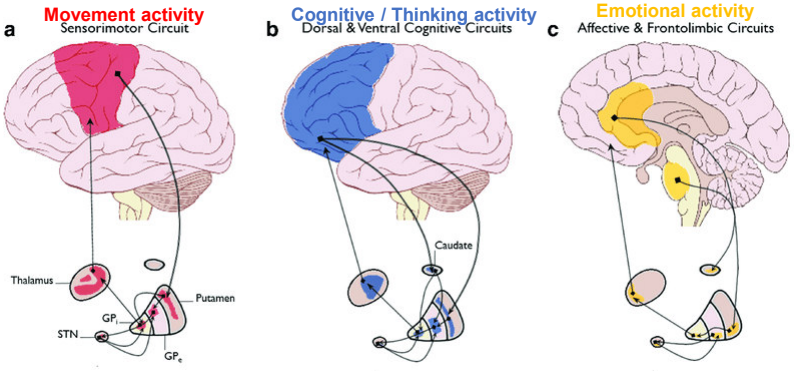
Wakefulness – Enhanced by activating pathways in the brain

During wakefulness, the Cortico – Striato – Thalamo – Cortical (CSTC) circuitry is very active which is part of the wake process and promotes the wake drive.

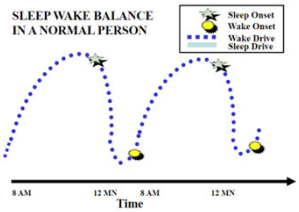
cortico-striato-thalamo-cortical (CSTC) circuitry

CSTC are activation circuits in the brain. Different subtypes involved in different types of processes. a) Motor function b) Cognitive Function c) Emotional Function

Factors that increase Wakefulness
- Movement
- Emotions such as stress
- Actively thinking



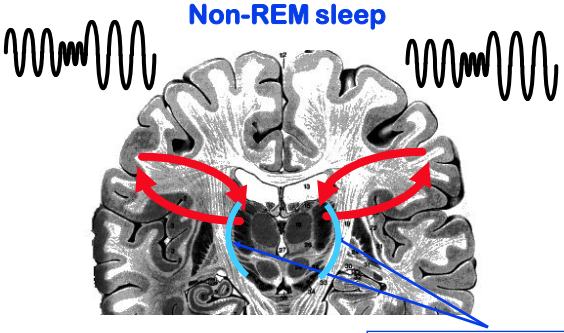
The CSTC is stimulating and enhances the wake state using Dopamine, Norepinephrine, Serotonin Histamine and other pathways.



19

Non-REM Sleep

During Non-REM sleep, the Reticular Thalamic Nucleus inhibits sensory input from the Thalamus that goes to the cortex (Thalamocortical pathways) which produces synchronous EEG activity during Non-REM sleep

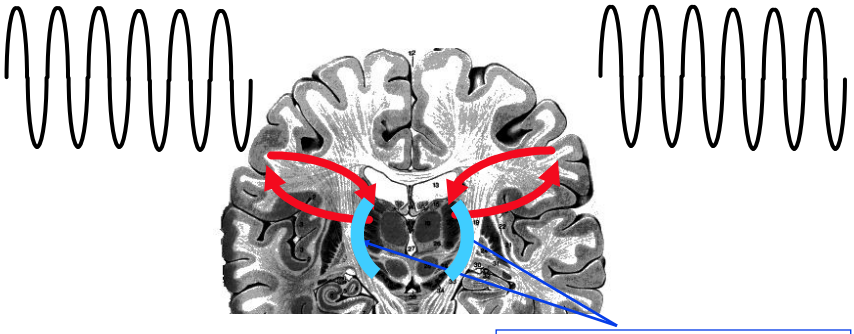


Reticular Thalamic Nucleus (Filter)
GABA inhibitory neurons that induce sleep

20

Non-REM Sleep N3 (Slow Wave Sleep)

The deepest level of sleep is N3 also known as Slow Wave Sleep, named by the high voltage slow waves that are present during this sate of sleep.

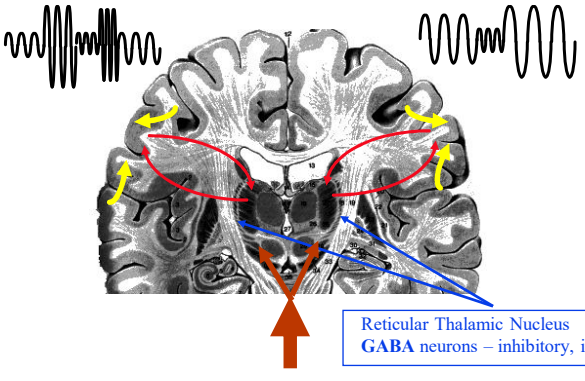


Reticular Thalamic Nucleus
Using GABA inhibition, induces sleep
By "filtering" out the information,
preventing it to get to the cortex

21

Wakefulness

The Ascending Reticular Activating System inhibits the Reticular Thalamic Nucleus allowing the cortex to be active during wakefulness



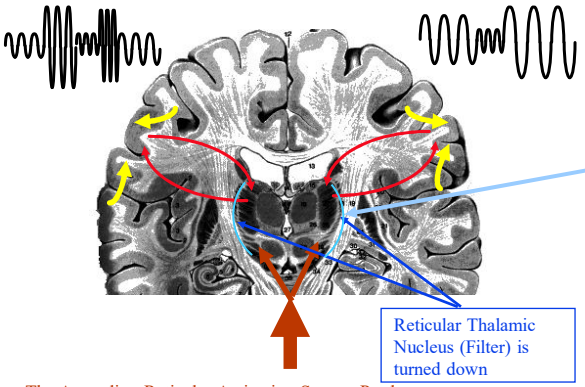
Reticular Thalamic Nucleus
GABA neurons – inhibitory, induce sleep

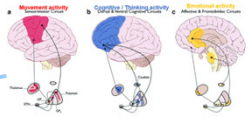
The Ascending Reticular Activating System Produces
Desychronization of the EEG During Wakefullness by
Inhibition of the Reticularthalamic Nucleus. **Acetylcholine and Histamine** – enhance wakefulness

22

Wakefulness

The Ascending Reticular Activating System inhibits the Reticular Thalamic Nucleus allowing the cortex to be active during wakefulness

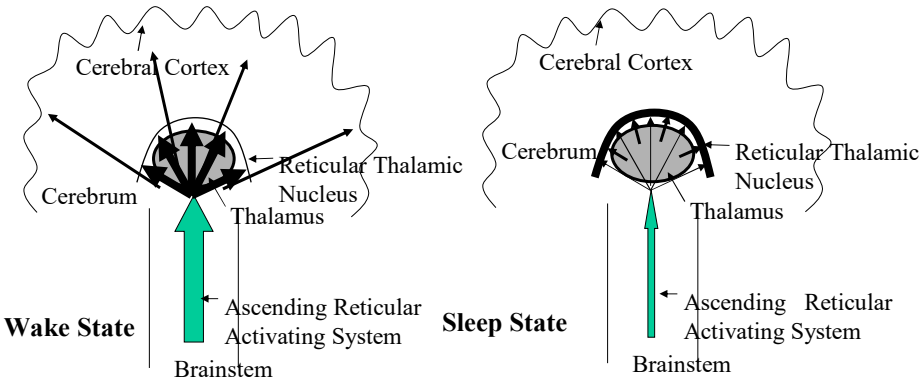




These systems in the brain are able to function because the "Filter" is turned down (weak) and does not prevent information to get to the cortex.

The Ascending Reticular Activating System Produces Desynchronization of the EEG During Wakefulness by Inhibition of the Reticularthalamic Nucleus (turning down the "filter".

23



Physiologic Mechanism of the Sleep Drive / Wake Drive system and the balance between sleep and wake states

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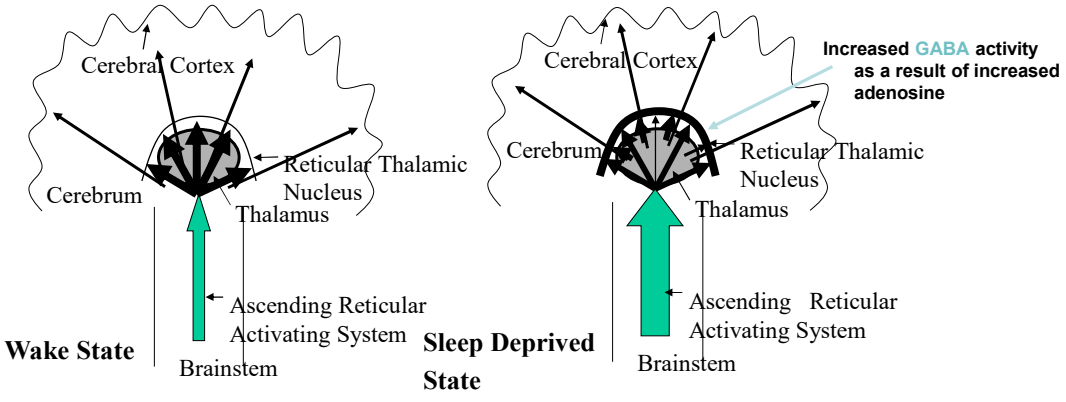
Physiologic Mechanism of the Sleep Drive / Wake Drive system and the changes between Wake and Sleep Deprived states



As a person becomes sleep deprived the reticular thalamic nucleus enhances its activity in attempts to shut down the cortex and induce sleep. Therefore it takes a greater degree of activity from the Ascending Reticular Activating System in order to overcome the Reticular Thalamic Nucleus activity and remain awake.

25

Epworth Sleepiness Scale

Situation	Chance of dozing (0–3)			
Sitting and reading	0	1	2	3
Watching television	0	1	2	3
Sitting inactive in a public place—for example, a theater or meeting	0	1	2	3
As a passenger in a car for an hour without a break	0	1	2	3
Lying down to rest in the afternoon	0	1	2	3
Sitting and talking to someone	0	1	2	3
Sitting quietly after lunch (when you've had no alcohol)	0	1	2	3
In a car, while stopped in traffic	0	1	2	3
			Total Score	

0 = would never doze
1 = slight chance of dozing
2 = moderate chance of dozing
3 = high chance of dozing

ESS total score ≥10 indicates need for further evaluation to determine cause of excessive sleepiness.

Johns MW. *Sleep*. 1991;14:540-545. Reprinted with permission from the American Academy of Sleep Medicine.

All the questions on the Epworth Sleepiness Scale pertain to situations that are not very activating and all are sitting or lying down.

This enhancing the likelihood of unmasking sleepiness, if present. Often patients will fill this out incorrectly. For example, some people do not sit and read during the day, particularly if they avoid it because of sleepiness. So since they never sit and read they would put a 0 instead of 3.

Insurance companies have come to focus on this tool for pre-authorization of services. It may be best to review the responses with patients.

26

The Relationship between ADHD and Sleep

"New" Concepts Connecting Childhood Sleep Disorders and Attention Deficit Hyperactivity Disorder

Not so new. These concepts were being recognized in the 1980's.

27

RLS/PLMD in Children: Daytime Symptoms

- Association with ADHD symptoms: inattention, hyperactivity, fidgetiness, difficulty sitting still
- Daytime "sleepiness" rare

28

PLMS and RLS has been show to be associated with ADHD in children

Treatments have demonstrated improvement in Sx of both ADHD and RLS / PLMS

Recent publications:

Walters AS, Mandelbaum DE, Lewin DS, Kugler S, England SJ, Miller M. Dopaminergic therapy in children with restless legs/periodic limb movements in sleep and ADHD. Pediatr Neurol 2000;22:182-186

Picchietti DL, England SJ, Walters AS, Willis K, Verrico T. Periodic limb movement disorder and restless legs syndrome in children with attention-deficit hyperactivity disorder. J Child Neurol 1998;13:588-594

Picchietti DL, Underwood DJ, Farris WA, Walters AS, Shah MM, Dahl RD, Trubnick LJ, Bertocci MA, Wagner M, Hening WA. Further studies on periodic limb movement disorder and restless legs syndrome in children with attention-deficit hyperactivity disorder. Move Dis 1999;14:1000-1007

29

Sleep Disordered Breathing and ADHD

- Habitual snoring more common in ADHD (33%) than other psych (11%), gen. peds (9%); severity of inattention/hyperactivity correlated with severity SDB symptoms Chervin, 1997

30

PEDIATRICS

Sleep Disorders and Daytime Sleepiness in Children with Attention-Deficit/ Hyperactive Disorder

Natali Golan BSc[1]; Eli Shahar MD[2]; Sarit Ravid MD[2,3]; Giora Pillar MD, DSc[1,3]

[1]Sleep Laboratory, [2]Pediatric Neurology Unit, and [3]Department of Pediatrics A, Rambam Medical Center and Technion-Israel Institute of Technology, Haifa, Israel

Study Objectives: Children with attention-deficit/hyperactive disorder (ADHD), in spite of being hyperactive, still benefit from treatment with stimulant medications. We hypothesized that children with ADHD are in fact sleepy during the day, and we sought to test it objectively.
Design: Single blind comparative study
Setting: University medical center
Participants: Thirty-four children with a previous diagnosis of ADHD (mean age ± SD, 12.4 ± 4.6 years) and 32 matched controls (mean age, 12.0 ± 3.6 years).
Interventions: N/A.
Measurements: All participants underwent a full-night polysomnographic study followed by a multiple sleep latency test (MSLT).
Results: Sleep latency, total sleep time, and sleep efficiency were comparable between the groups, yet children with ADHD were significantly sleepier during the day than those in the control group (mean MSLT score of 21.9 ± 5.5 minutes versus 27.9 ± 2.0 minutes, P < .005). Of the children with ADHD, 17 (50%) had signs of sleep-disordered breathing, compared with 7 of the control group (22%, P < .05). Five of the ADHD group had periodic limb movements during sleep (15%) versus none in the control group. Children without sleep-disordered breathing or periodic limb movements during sleep had the lowest nocturnal sleep efficiency and total sleep time.
Conclusions: We conclude that children with ADHD demonstrate objective daytime somnolence, which may explain the beneficial effects of treatment with stimulant medications. Primary sleep disorders, especially sleep-disordered breathing and periodic limb movement disorder, should be looked for in children with ADHD.

Citation: Golan, N; Shahar E; Ravid S; Pillar G. Sleep disorders and daytime sleepiness in children with attention-deficit/hyperactive disorder. *SLEEP* 2004;27(2):261-6.

31

The key role of daytime sleepiness in cognitive functioning of adults with attention deficit hyperactivity disorder

Published online by Cambridge University Press: **05 March 2020**

Bartosz Helfer, Natali Bozhilova, Ruth E. Cooper, Joanna I. Douzenis, Stefanos Maltezos and Philip Asherson

Show author details

Cureus. 2020 Jun; 12(6): e8436. PMCID: PMC7336577
Published online 2020 Jun 4. doi: 10.7759/cureus.8436 PMID: 32642351

Attention-Deficit/Hyperactivity Disorder Patients May Have Undiagnosed Narcolepsy

Monitoring Editor: Alexander Muacevic and John R Adler

Lukas Wilenius[1] and Markku Partinen[2,3]

▸ Author information ▸ Article notes ▸ Copyright and License information Disclaimer

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Nature and Science of Sleep

Dovepress
open access to scientific and medical research

Open Access Full Text Article

REVIEW

Sleep disorders in patients with ADHD: impact and management challenges

This article was published in the following Dove Press journal:
Nature and Science of Sleep

Dafna Wajszilber[1]
José Arturo Santiseban[1,2]
Reut Gruber[1,2]

[1]Department of Psychiatry, Faculty of Medicine, McGill University, Montréal, Quebec, Canada; [2]Attention Behavior and Sleep Lab, Douglas Research Center, Montréal, Quebec, Canada

Abstract: Attention deficit/hyperactivity disorder (ADHD) is one of the most commonly diagnosed disorders in childhood, enduring through adolescence and adulthood and presenting with symptoms of inattention, hyperactivity, and/or impulsivity and significantly impairing functioning. Primary sleep disorders such as sleep-disordered breathing, restless leg syndrome, circadian rhythm sleep disorder, insomnia, and narcolepsy are commonly comorbid in these individuals but not often assessed and are therefore often left untreated. Sleep disturbances in individuals with ADHD can result in significant functional impairments that affect mood, attention, behavior, and ultimately school/work performance and quality of life. Previous reviews have described findings related to sleep but have neglected to examine potential impacts of these sleep disorders and ADHD on daytime functioning. This review investigates empirical findings pertaining to sleep abnormalities and related cognitive, behavioral, emotional, and physical impairments in individuals with ADHD and comorbid primary sleep disorders across the life span. It discusses implications to management and highlights existing limitations and recommended future directions.

Keywords: ADHD, impairments, sleep-disordered breathing, restless leg syndrome, circadian rhythm sleep disorders, insomnia, narcolepsy

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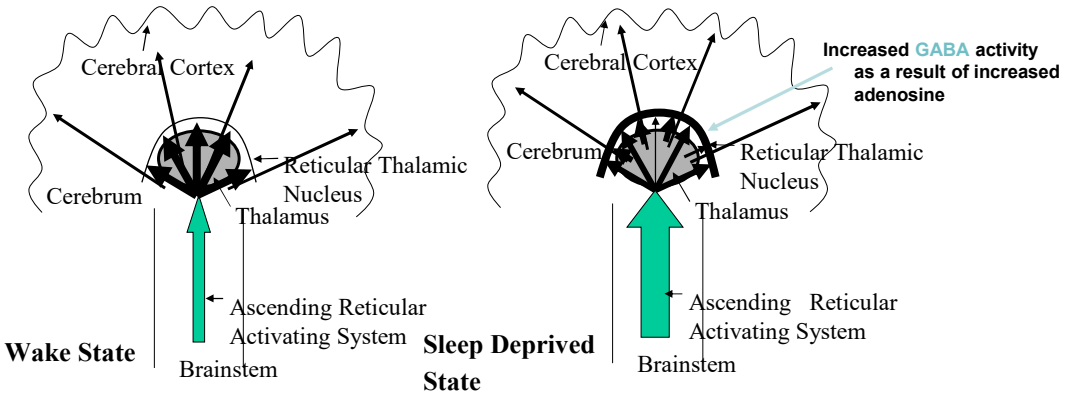
Physiologic Mechanism of the Sleep Drive / Wake Drive system and the changes between Wake and Sleep Deprived states



As a person becomes sleep deprived the reticular thalamic nucleus enhances its activity in attempts to shut down the cortex and induce sleep. Therefore it takes a greater degree of activity from the Ascending Reticular Activating System in order to overcome the Reticular Thalamic Nucleus activity and remain awake.

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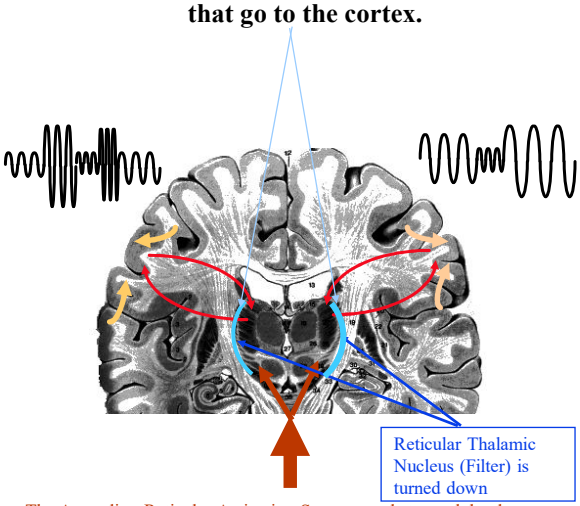
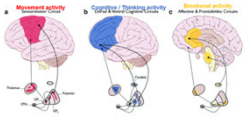
Sleep Deprivation (or suboptimal sleep)

The Reticular Thalamic Nucleus is trying to filter the signal going to the cortex and this disrupts the underlying circuits that go to the cortex.

In many children ADHD symptoms result from the struggle within the brain of trying to function under the circumstances of a sleep deprived state (enhanced filtering from the reticular thalamic nucleus)



The Ascending Reticular Activating System needs to work harder to overcome the increased activity of Reticularthalamic Nucleus (the "filter") in order to keep the person awake.



These systems in the brain do not function normally when the brain is sleep deprived. The "filter" inhibits "information" from optimally getting to the cortex.

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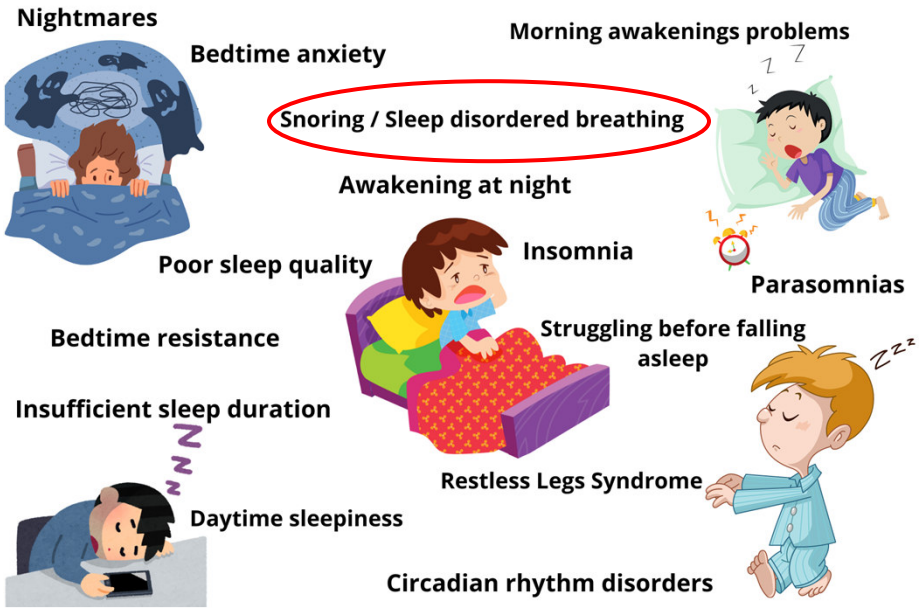
Sleep problems and ADHD



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Taking A Deeper Dive

Understanding The Different Stages Of Sleep

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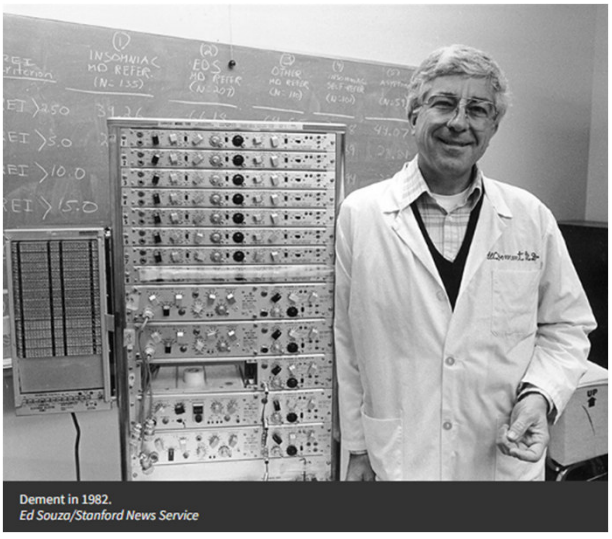
REM Sleep

William Dement, MD, PhD, working with Nathaniel Kleitman, PhD, and Eugene Aserinsky, PhD, Discovering rapid eye movements sleep, coining the term REM, and its association with dreaming. .



Dement in 1982.
Ed Souza/Stanford News Service

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Dr. Dement, one of my mentors, founded the worlds FIRST sleep center in 1970 at Stanford University and the first Sleep Fellowship training program, where I was fortunate to attend and work with Dr. Dement from 1991-1992.
(July 29, 1928 – June 17, 2020)

Stanford University



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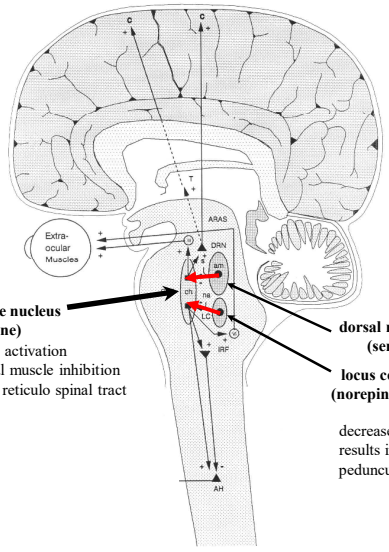
Brainstem regulation and control of REM



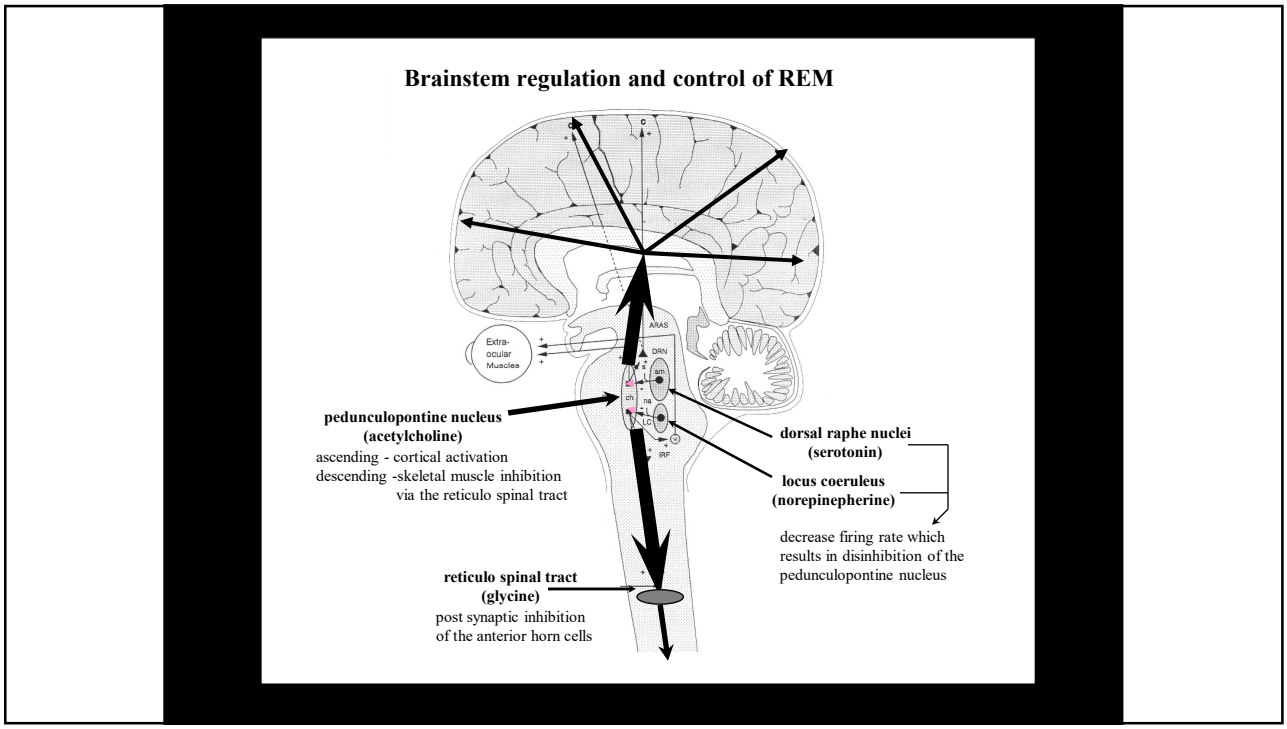
pedunculopontine nucleus (acetylcholine)
ascending - cortical activation
descending -skeletal muscle inhibition via the reticulo spinal tract

dorsal raphe nuclei (serotonin)

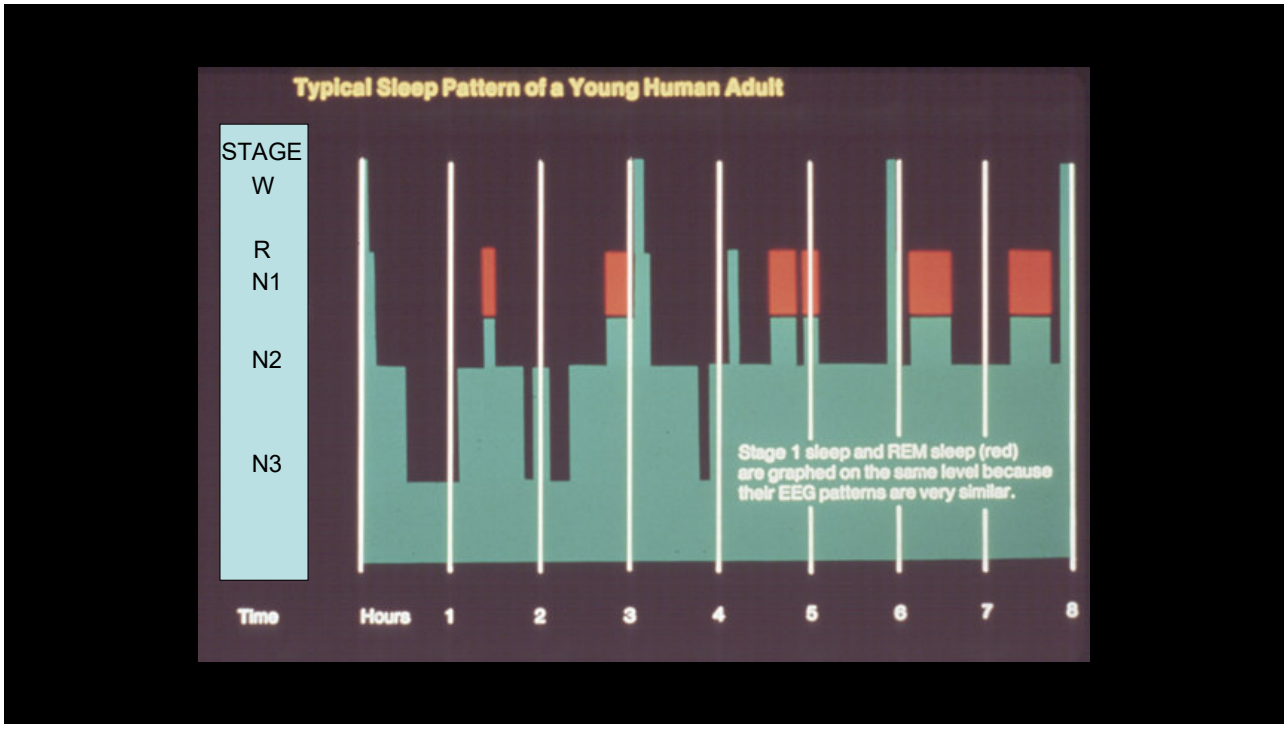
locus coeruleus (norepinepherine)

decrease firing rate which results in disinhibition of the pedunculopontine nucleus

40


Brainstem regulation and control of REM
ARAS
DRN
lm
cn
na
LC
IRF
Extra-ocular Muscles
pedunculopontine nucleus
(acetylcholine)
ascending - cortical activation
descending -skeletal muscle inhibition
via the reticulo spinal tract
dorsal raphe nuclei
(serotonin)
locus coeruleus
(norepinepherine)
decrease firing rate which
results in disinhibition of the
pedunculopontine nucleus
reticulo spinal tract
(glycine)
post synaptic inhibition
of the anterior horn cells

41


Typical Sleep Pattern of a Young Human Adult
STAGE
W
R
N1
N2
N3
Stage 1 sleep and REM sleep (red) are graphed on the same level because their EEG patterns are very similar.
Time
Hours
1
2
3
4
5
6
7
8

42

Stages of Sleep

N Sages – Somatic Restoration
Divided into N1, N2 and N3

N3 is the stage in which there is the highest level of Growth Hormone release

R Stage – (REM) – Limbic Restoration ---
Consolidation of Short Term Memory into Long Term Memory

Active (vivid) Dreaming, Rapid Eye Movements
Muscle Atonia

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Suppressed GH in OSA patients - Resolves with nasal CPAP

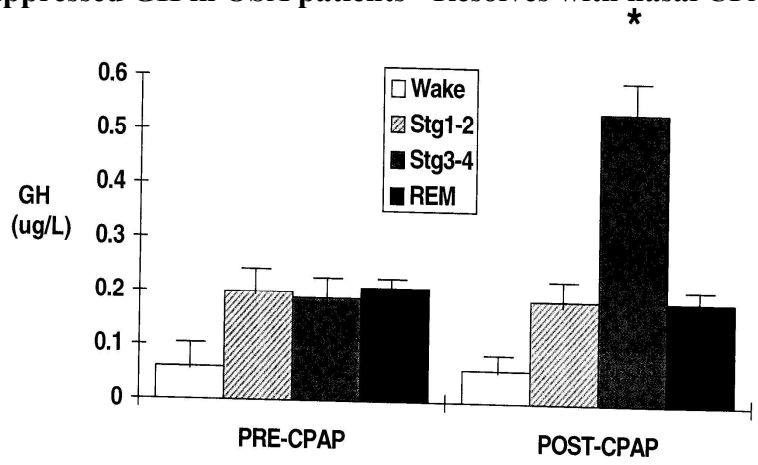

FIG. 1. Plasma growth-hormone (GH) levels in different sleep stages in eight patients with severe sleep apnea before and after CPAP. Note that the relationship between slow-wave sleep and GH concentrations becomes significant on CPAP treatment.

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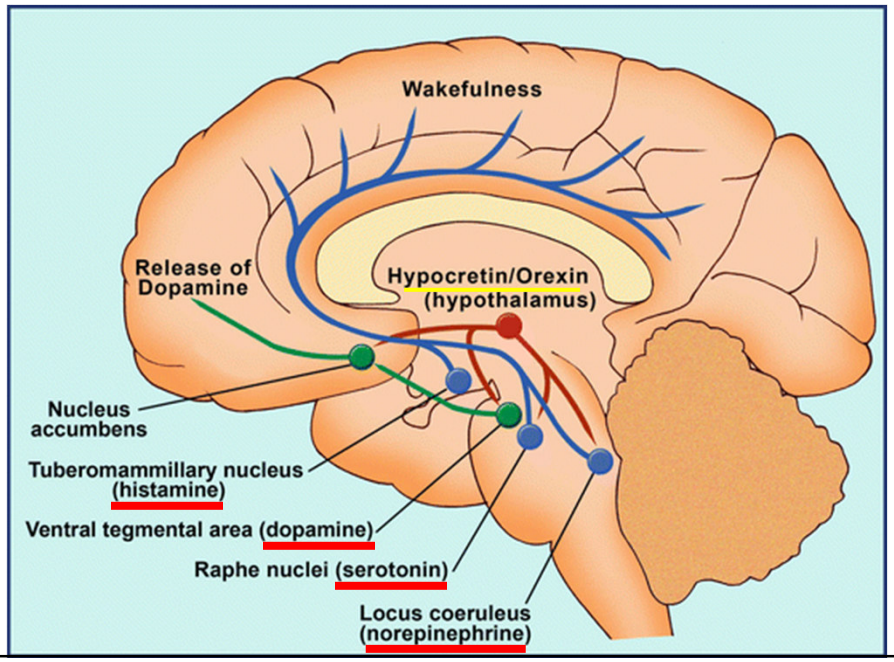
Neurotransmitter systems and the control of wakefulness



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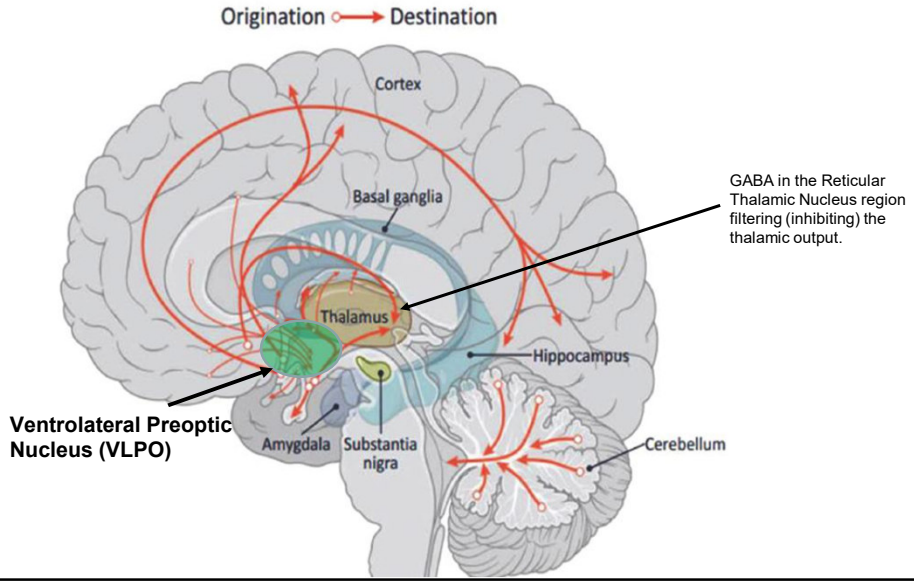
Sleep

Sleep is driven by a gamma-aminobutyric acid (GABA) system originating from the VLPO region

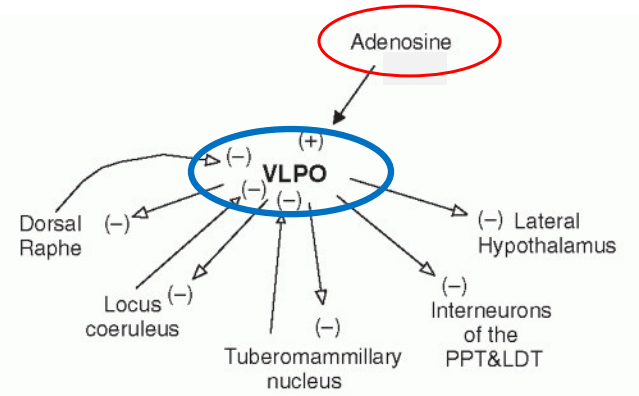
GABA pathways in the brain are shown below



46

Why do we sleep?

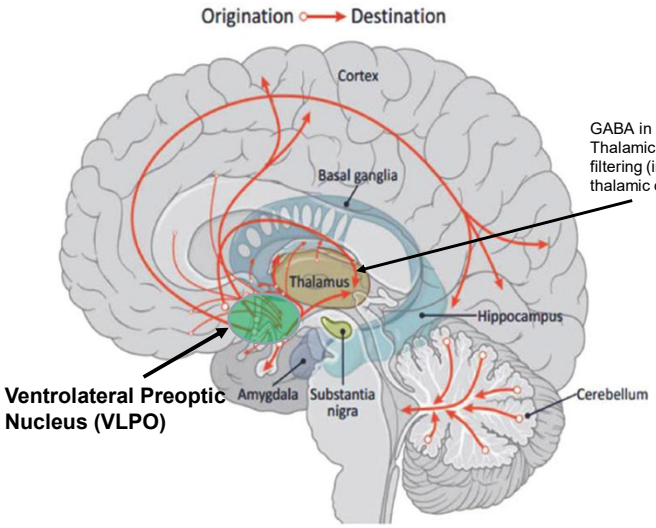
One model hypothesizes that sleep occurs as a result of adenosine accumulation as the brain breaks down ATP for energy production



Ventrolateral Preoptic (VLPO) region – Sleep promotion region of the brain that releases GABA, increasing sleep propensity (Sleep Drive)

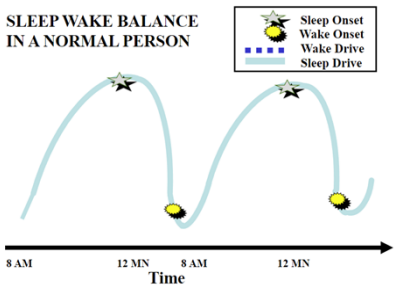
47

Sleep is driven by a GABA system originating from the VLPO region.
GABA pathways in the brain are shown below
GABA is an inhibitory neurotransmitter on thalamic outputs to the cortex



GABA in the Reticular Thalamic Nucleus region filtering (inhibiting) the thalamic output.

The VLPO GABA system governs the Sleep drive possibly in response to increasing Adenosine levels



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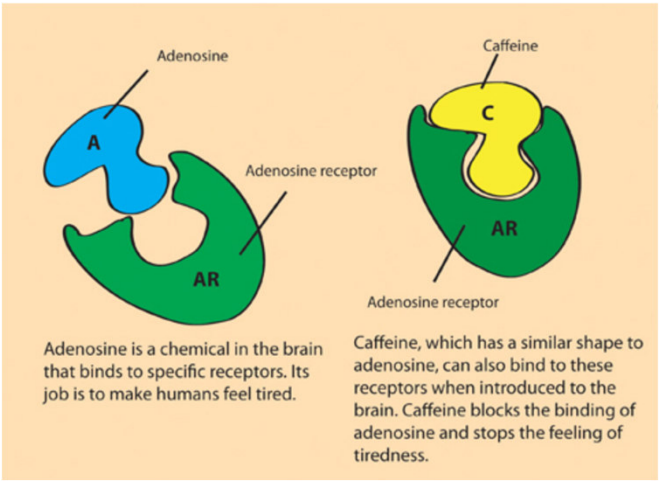
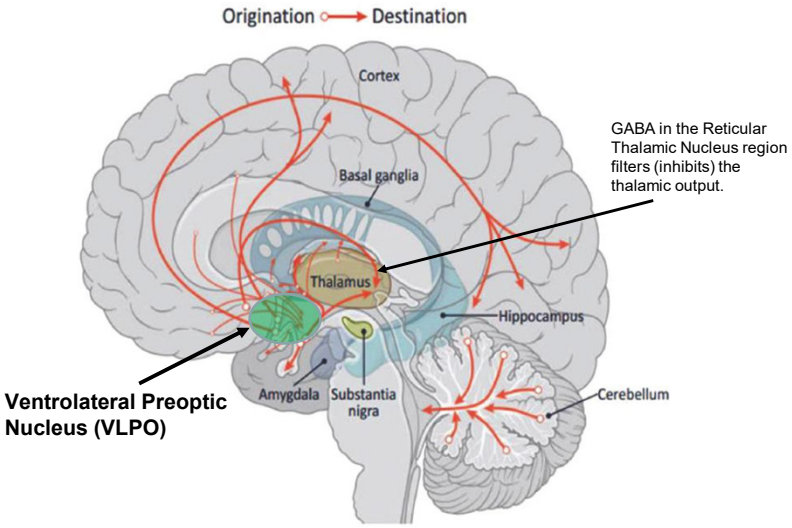
Caffeine is by far the #1 drug used to enhance wakefulness



49

Caffeine has several potential mechanism on enhancing wakefulness. One main factor is by inhibiting adenosine's enhancement on GABA activity from the VLPO region. This reduces the sleepiness caused by increased adenosine in the brain.



Adenosine increases the VLPO GABA activity (sleep drive)

Caffeine inhibits adenosine receptors, lowering GABA from the VLPO nucleus and decreases of the Sleep Drive.

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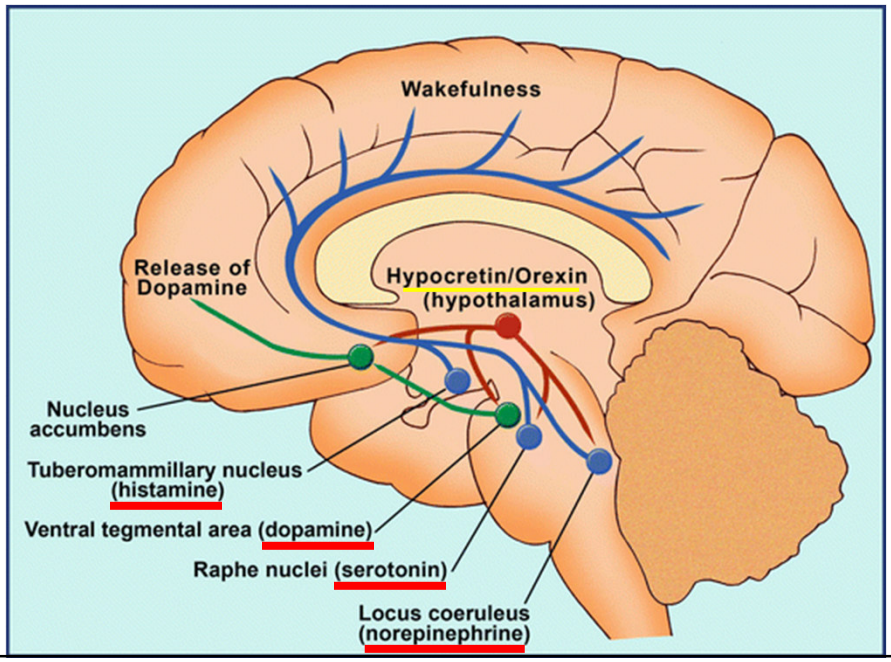
Neurotransmitter systems and the control of wakefulness



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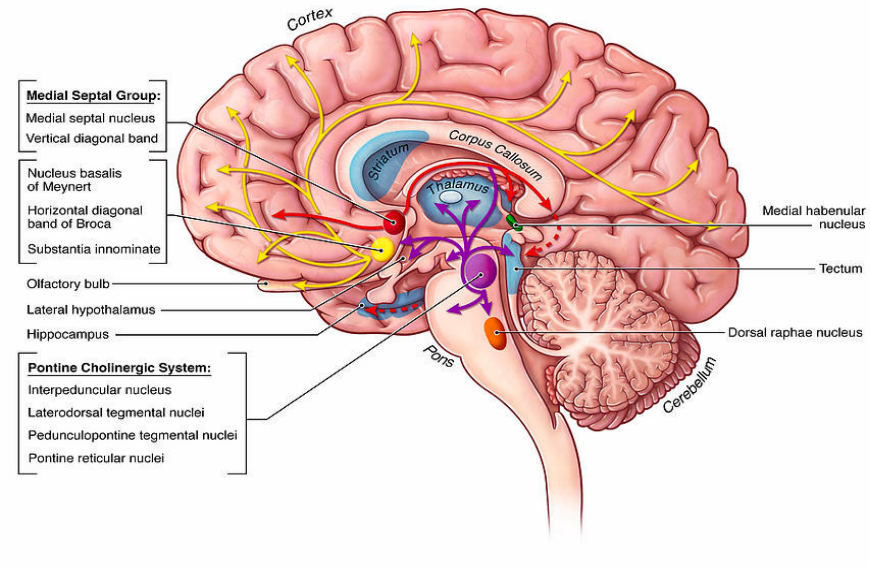
Cholinergic Pathways



Cholinergic Pathways, Illustration **is a photograph by Evan Oto, February 6th, 2017.**

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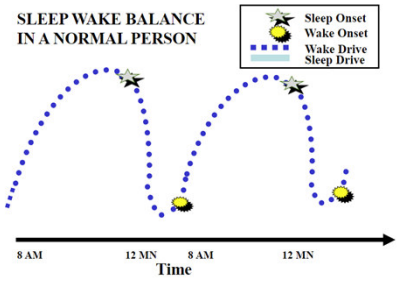
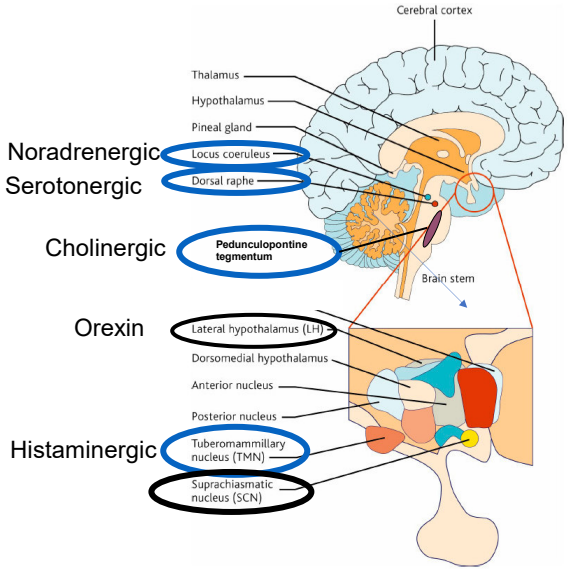
These regions and their Neurotransmitter / Neuropeptide enhance the wake state





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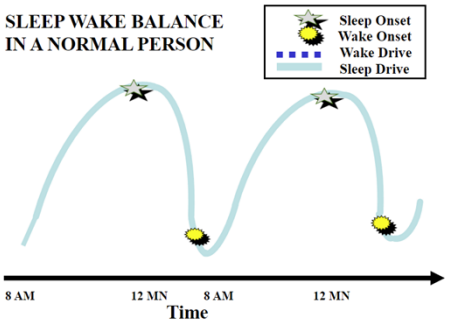
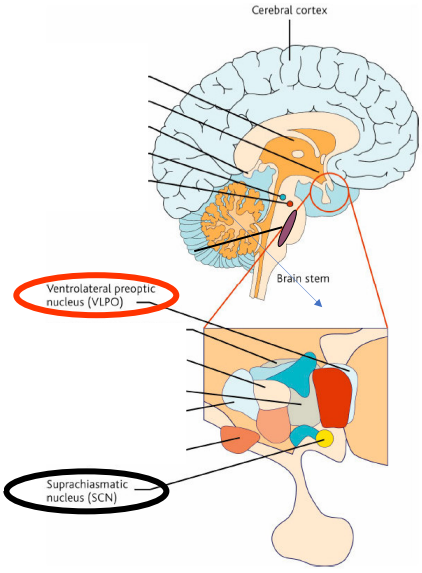
VLPO (GABA) enhance the **Sleep drive**, and possibly modulated by Adenosine levels and the SCN (? Melatonin)





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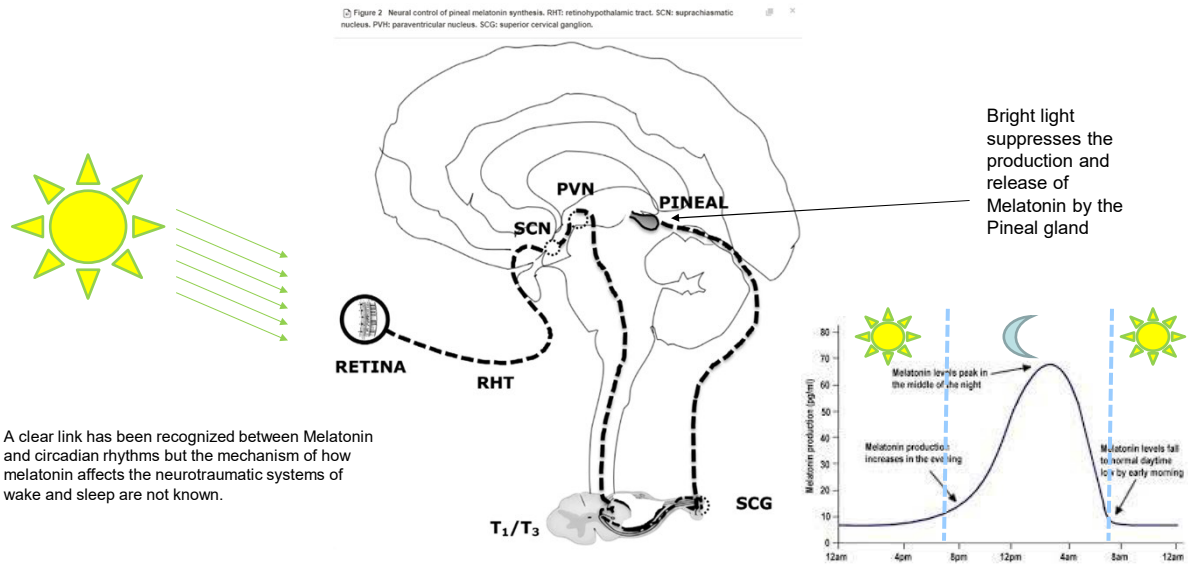
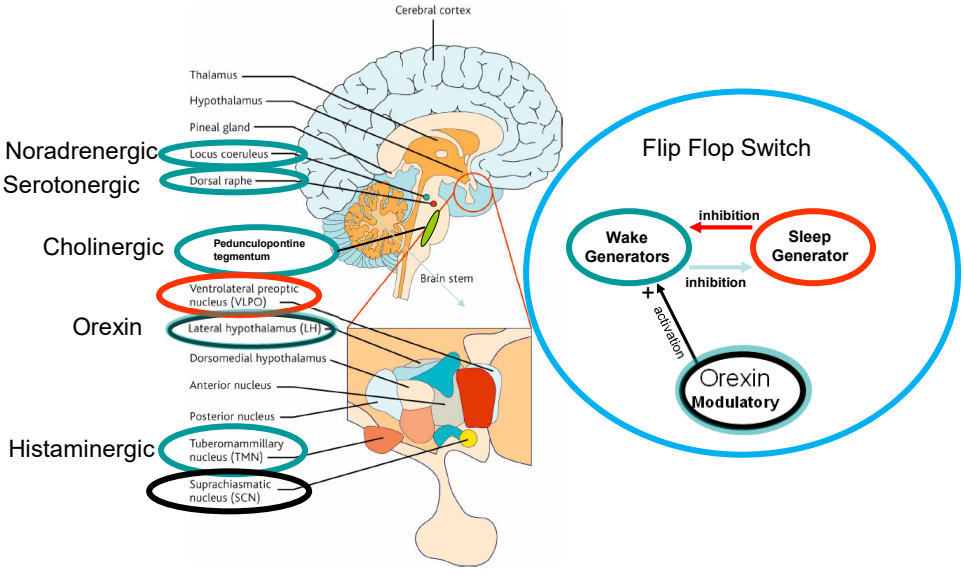
Melatonin and the Sleep / Wake cycle



A clear link has been recognized between Melatonin and circadian rhythms but the mechanism of how melatonin affects the neurotraumatic systems of wake and sleep are not known.

55

Control Regions in the Brain Producing the **Sleep Wake Balance** and Shifting levels of Cortical Activation



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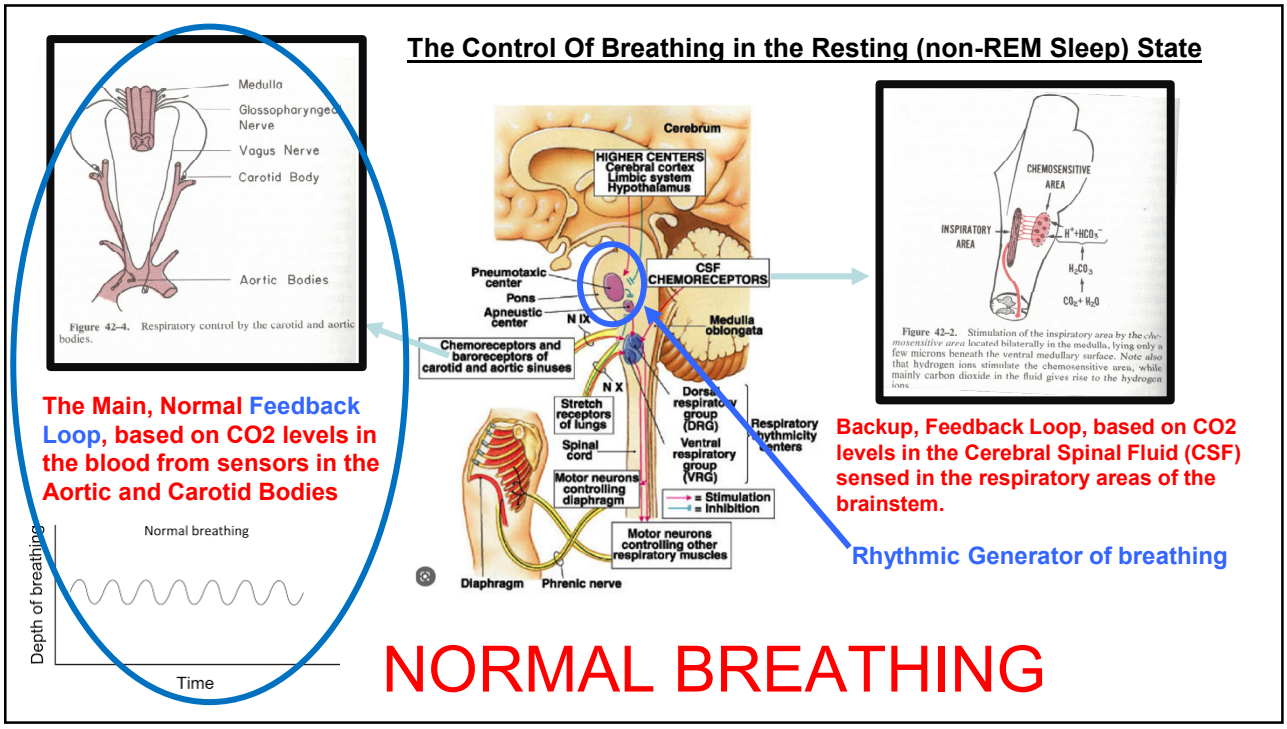
What is the main stimulus that drives our breathing?

CO_2

Our breathing is mainly trigger by the buildup of CO2 in our blood. We breath to release this by product of metabolism (exhaust product) from our body.

57



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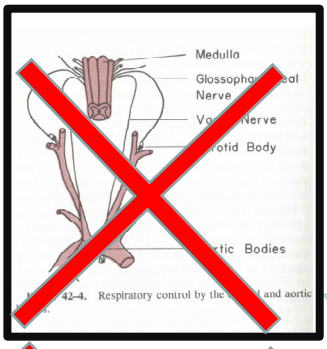
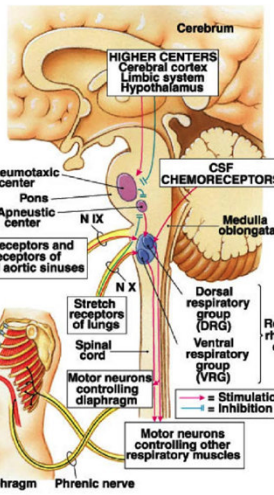
When the Main Feedback System does not work, then the Backup Feedback System kicks in





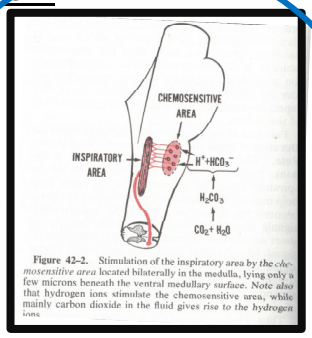
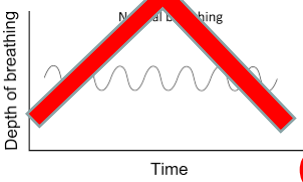


Figure 42–2. Stimulation of the inspiratory area by the chemosensitive area located bilaterally in the medulla, lying only a few microns beneath the ventral medullary surface. Note also that hydrogen ions stimulate the chemosensitive area, while mainly carbon dioxide in the fluid gives rise to the hydrogen ions.

The Main, Normal Feedback Loop, based on CO_2 levels in the blood from sensors in the Aortic and Carotid Bodies



Backup, Feedback Loop, based on CO_2 levels in the Cerebral Spinal Fluid (CSF) sensed in the respiratory areas of the brainstem.

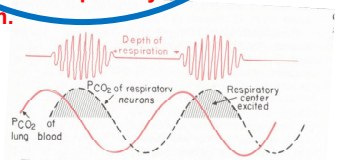


Figure 42–13. Cheyne-Stokes breathing, showing the changing P_{CO_2} in the pulmonary blood (*solid line*) and the delayed changes in P_{CO_2} of the fluids of the respiratory center (dashed line).

Cheyne Stokes Breathing

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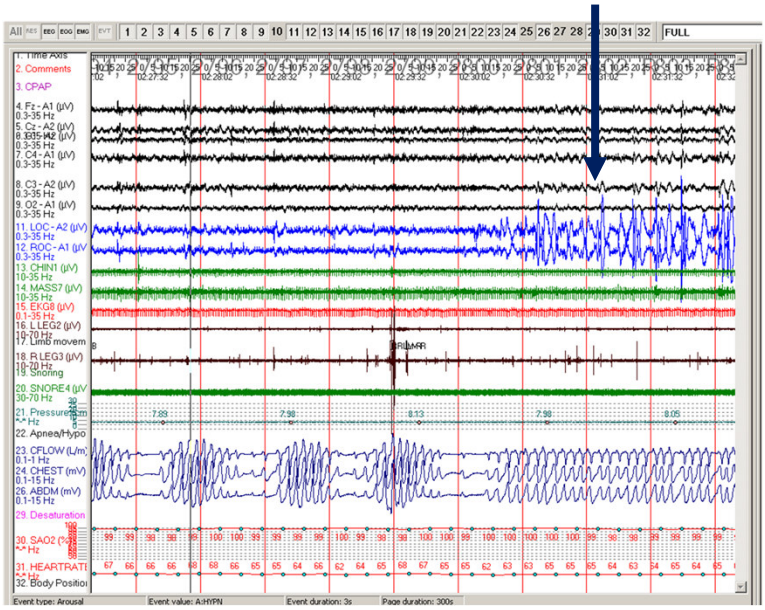
Cheyne Stokes Respirations during non-REM Sleep in a patient with CHF that terminates when the patient goes into REM sleep.



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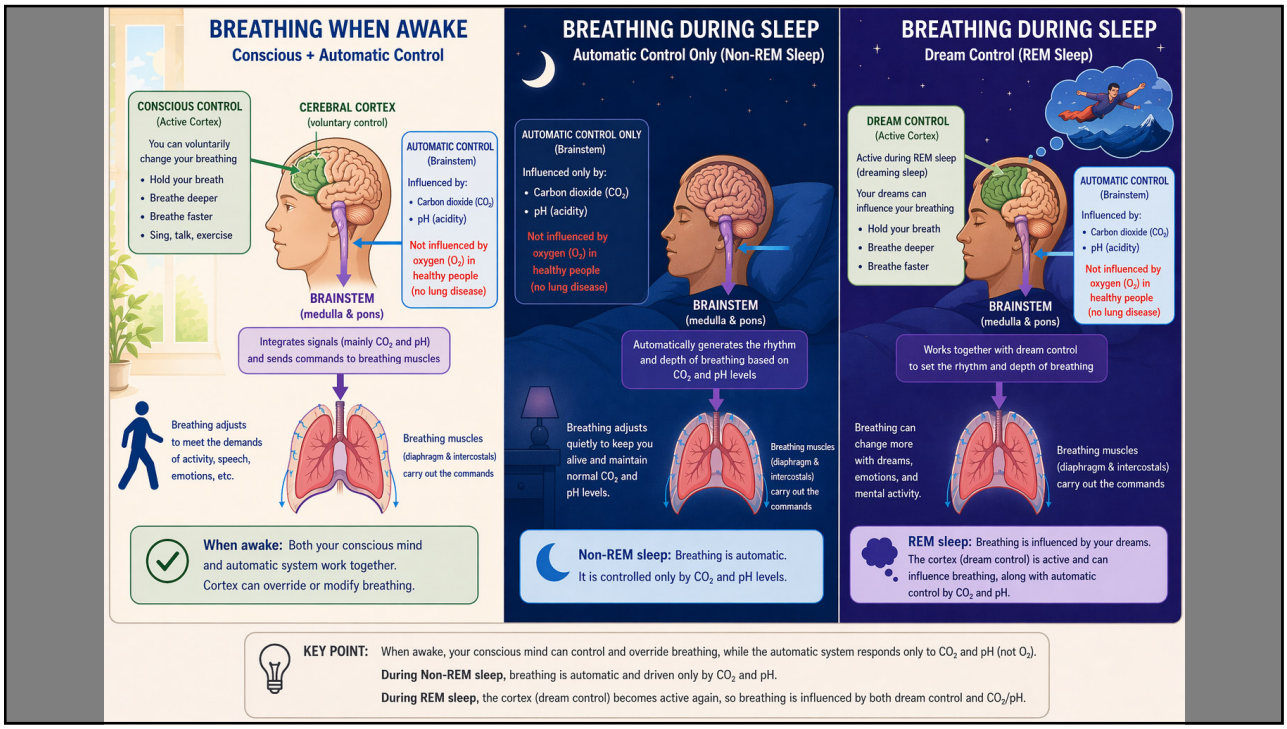

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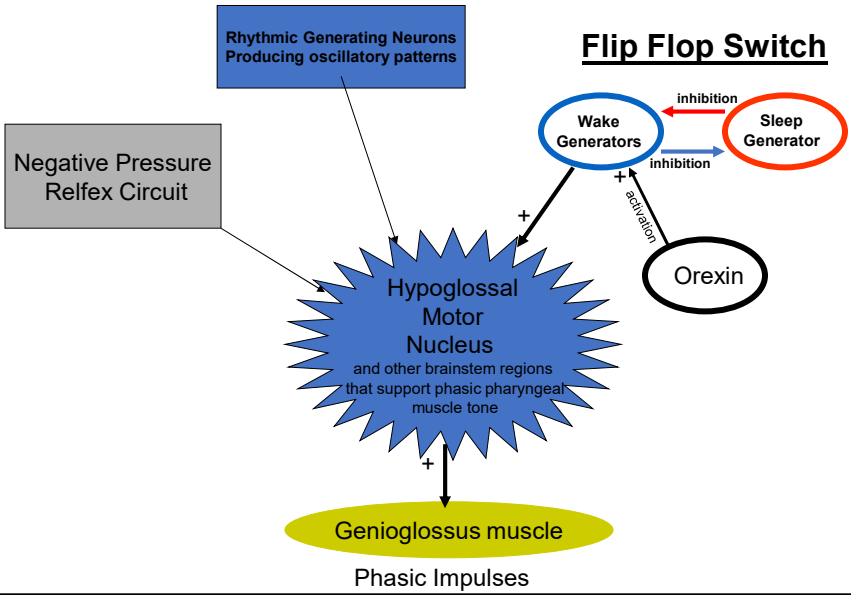
Degrees of sleepiness and wakefulness can influence many systems, including upper airway motor systems



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Changes in genioglossus muscle tone in response to negative pressures of the upper airway. This reflex is altered by sleep, wake and sleep deprivations. These factors alter the activity level of the Hypoglossal Motor Nucleus

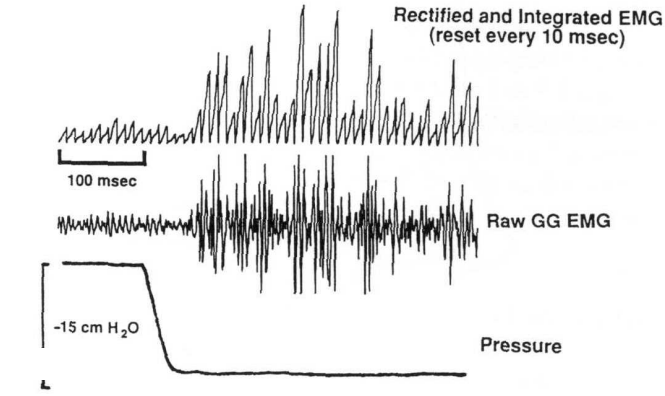

FIG. 3. Reflex activation of genioglossus muscle following a −15 cm H_2O pressure change applied to the upper airway via a face mask in a normal subject. Note the short latency of genioglossus activation from the onset of the pressure change (≈40 mseconds).

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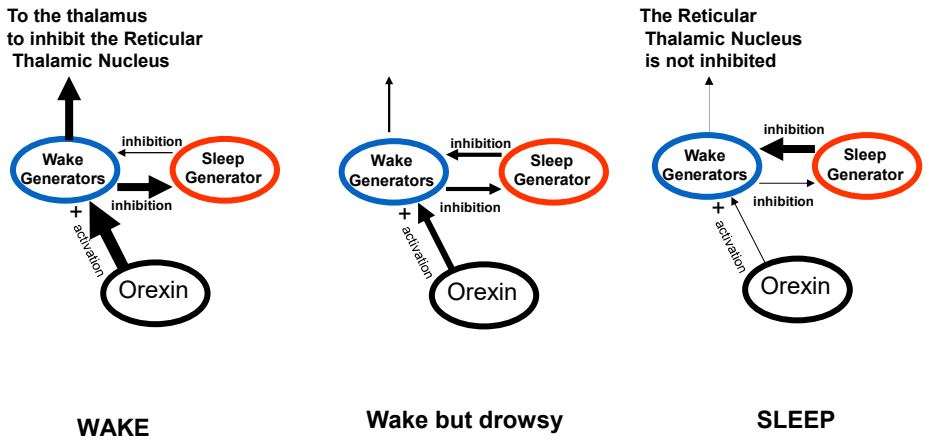
Flip Flop Switch



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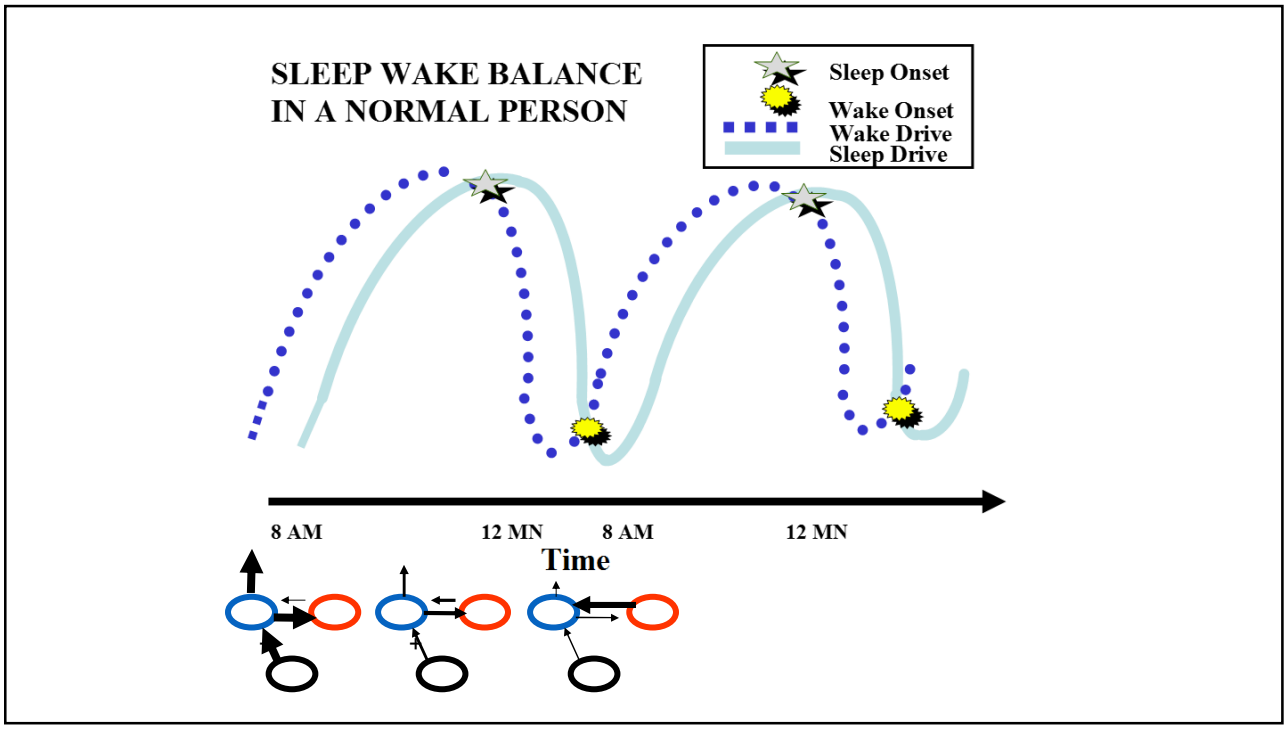

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Conditions Causing Excessive Daytime Sleepiness

Central Nervous System cause

Narcolepsy – Type 1 (with Cataplexy and loss of Orexin)
Type 2 (without Cataplexy but with other REM features)
Kleine - Levin Syndrome – periodic hypersomnolence with characteristic features.
Idiopathic Hypersomnolence – Not related to REM sleep but no known pathology

Will be discussed tomorrow by Maggie Lavender, RN, FNP

Fragmentation of Sleep as the cause

Obstructive Sleep Apnea / Upper Airway Resistance Syndrome (Will discuss later today)
Periodic Leg Movements Disorder (PLMD) – either with or without Restless Leg Syndrome Sx
Environmental causes of sleep fragmentation
Chronic pain causing sleep fragmentation

Will be discussed later today by William Ondo, MD

Lack of Total Sleep Time

Insufficient Sleep Syndrome – not allowing enough time for sleep
Insomnia – inability to sleep during the normal sleep opportunities (i.e. at night)

Other secondary causes

Medication side effects
EDS associated with Shift Work Disorder –
Other medical conditions – Stoke, CHF, Metabolic conditions etc.
Substance abuse

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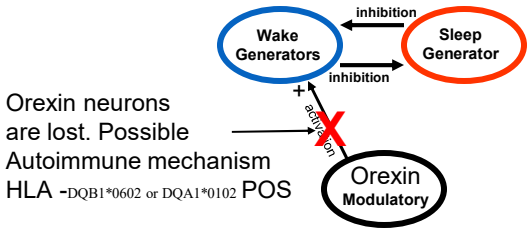
Narcolepsy is associated with a deficiency of Orexin levels, more tightly in Type 1 than in Type 2.

Flip Flop Switch



Orexin neurons are lost. Possible Autoimmune mechanism HLA -DQB1*0602 or DQA1*0102 POS

Orexin = Hypocretin

Narcolepsy is a disorder is sleep state disassociation, mainly between wake and sleep

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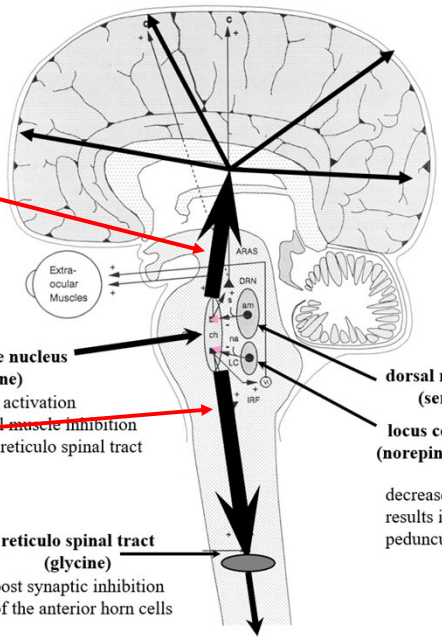
Features of narcolepsy are associated with components of REM sleep

Hypnagogic (on falling asleep) or Hypnopompic (on waking up) hallucinations

Sleep paralysis
Cataplexy



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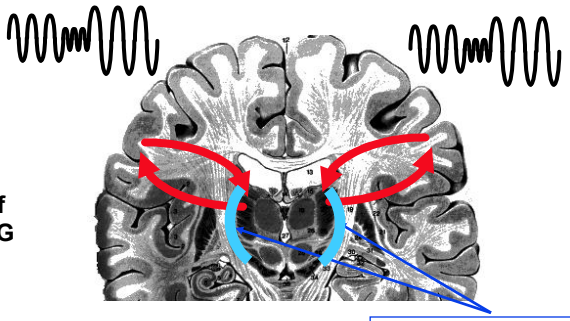
Possible Mechanism of Idiopathic Hypersomnolence

The Reticular Thalamic Nucleus, as it inhibits Thalamocortical pathways which produces synchronous EEG activity during Non-REM sleep. Sleep Spindles are a signature in the EEG that the Reticular Thalamic Nucleus is actively functioning.

Patients with Idiopathic Hypersomnolence may have a higher frequency of Sleep Spindles in their EEG



Reticular Thalamic Nucleus
GABA neurons – inhibitory, induce sleep

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> Sleep. 1994 Aug;17(5):449-55. doi: 10.1093/sleep/17.5.449.

Relationship between sleep spindles and hypersomnia

A Bové [1], A Culebras, J T Moore, R E Westlake

Affiliations + expand

PMID: 7991957 DOI: 10.1093/sleep/17.5.449

Abstract

Sleep spindles (SS) and K complexes constitute the physiological markers of stage 2 sleep. Because sleep allows a spontaneous thalamic manifestation in the form of SS, one could hypothesize that there is some kind of relationship between SS and the complaint of hypersomnia. To investigate this possible relationship we compared nonhypersomnolent subjects with hypersomnolent patients who carried a diagnosis of narcolepsy or idiopathic hypersomnia. SS were counted in well-defined nocturnal stage 2 sleep segments, and the average SS density (number of SS in stage 2/minute stage 2) was tabulated for the entire night. Agreement between two independent scores was higher than 95%. The results show that the average SS density is higher in both cerebral hemispheres in the hypersomnolent group, especially in the idiopathic hypersomnia patients. At the beginning and at the end of the nocturnal sleep time, SS density is increased in this group compared with the normal one. These findings support the complaint of hypersomnia, mainly in idiopathic hypersomnia patients. This is in agreement with the notion that SS are generated by thalamic structures that serve a gatekeeping function during nonrapid eye movement sleep, and further suggests that their relative abundance expresses the power of that control.

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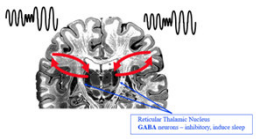
The Spectrum of the strength of the Underlying Sleep Drive Mechanism

Stronger than normal



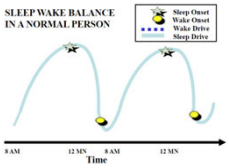
Weaker than normal

Idiopathic Hypersomnia

Narcolepsy

Primary Idiopathic Insomnia

Psychophysiologic Insomnia



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Measuring Sleepiness

Subjective

Objective

The Stanford Sleepiness Scale (SSS)

Degree of Sleepiness	Scale Rating
Feeling active, vital, alert, or wide awake	1
Functioning at high levels, but not at peak; able to concentrate	2
Awake, but relaxed; responsive but not fully alert	3
Somewhat foggy, let down	4
Foggy; losing interest in remaining awake; slowed down	5
Sleepy, woozy, fighting sleep; prefer to lie down	6
No longer fighting sleep, sleep onset soon; having dream-like thoughts	7

Multiple Sleep Latency Test (MSLT)

1. **Consists of 4 or 5 napping sessions**
2. **These are spaced about 2 hours apart throughout the day**
3. **Allow 20 minutes for Sleep Onset**
4. **If asleep then allow 15 minutes for REM.**
5. **MUST have an in-lab overnight NPSG the night before.**
6. **Used to diagnose Narcolepsy and Idiopathic Hypersomnolence**

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Multiple Sleep Latency Test (MSLT)

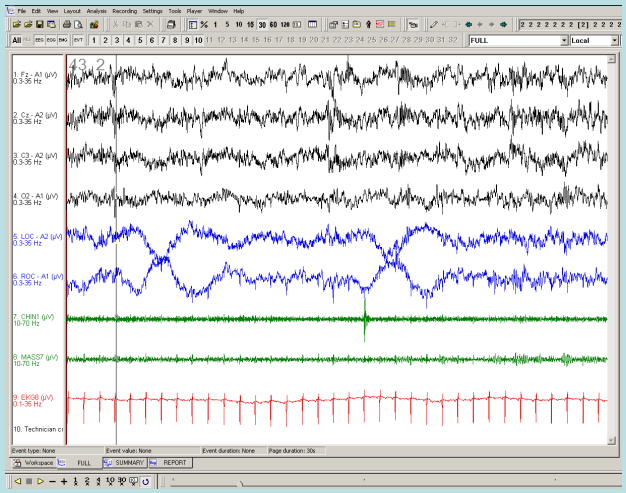
Consists of 4 or 5 napping sessions at two-hour intervals during the day
Each lasting 20 to 35 min, to assess for sleep tendency and onset to REM sleep.

Parameters needed to stage sleep

EEG

EOG

EMG



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Sleepiness Severity Criteria

Severity of Sleepiness	Description and Levels of Impairment	MSLT Criteria*
Mild	Sleep episodes are present only during times of rest or when little attention is required (eg, reading while lying down, watching television, or as a passenger in a motor vehicle). Mild sleepiness produces a minor impairment of social or occupational function.	10–15 min
Moderate	Sleep episodes are present daily and occur during mild physical activities requiring a moderate degree of attention (eg, watching a concert, movie, or theater performance, attending a meeting, or driving). This usually produces a moderate degree of impairment of social or occupational function.	5–10 min
Severe	Sleep episodes are present daily and at times of activity that require moderate attention (eg, eating, direct conversation, driving, walking or other physical activities). These symptoms produce marked impairment of social or occupational function.	<5 min

MSLT=Multiple Sleep Latency Test.
*MSLT criteria are a guide, and should be considered in conjunction with the patient's overall clinical status.
American Sleep Disorders Association, 1997.

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Sleep and ADHD
What inspired you to reflect?
Pick the context and a clinically relevant concept or phrase that inspired you to reflect.
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Sleep and ADHD
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Next

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