Basics of Sleep Medicine: From A to ZZZZ's

Joyce K. Lee-Iannotti, MD
Chief and Medical Director,
Sleep Disorders Center
BUMC-P

IM Sleep Lecture
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Objectives

- Normal Sleep
- Physiology of sleep (just briefly)
- Sleep stages
- Introduction to the PSG
- Sleep Disorders
  - Insomnia
  - OSA

Basic Sleep Concepts

- Drive for sleep exceeds the drive for food and water, and freedom from pain
- Sleep deprivation, total or chronic partial, may have serious consequences
  - death in experimental animals
  - impaired perception and microsleeps in humans
- Sleep debt must eventually be repaid

Sleep-Wake Cycle Regulation

- Two related key processes promote sleepiness or mental arousal at different times
  - Homeostatic drive
  - Circadian rhythm
- Together, these determine when sleep can occur under both normal and abnormal circumstances

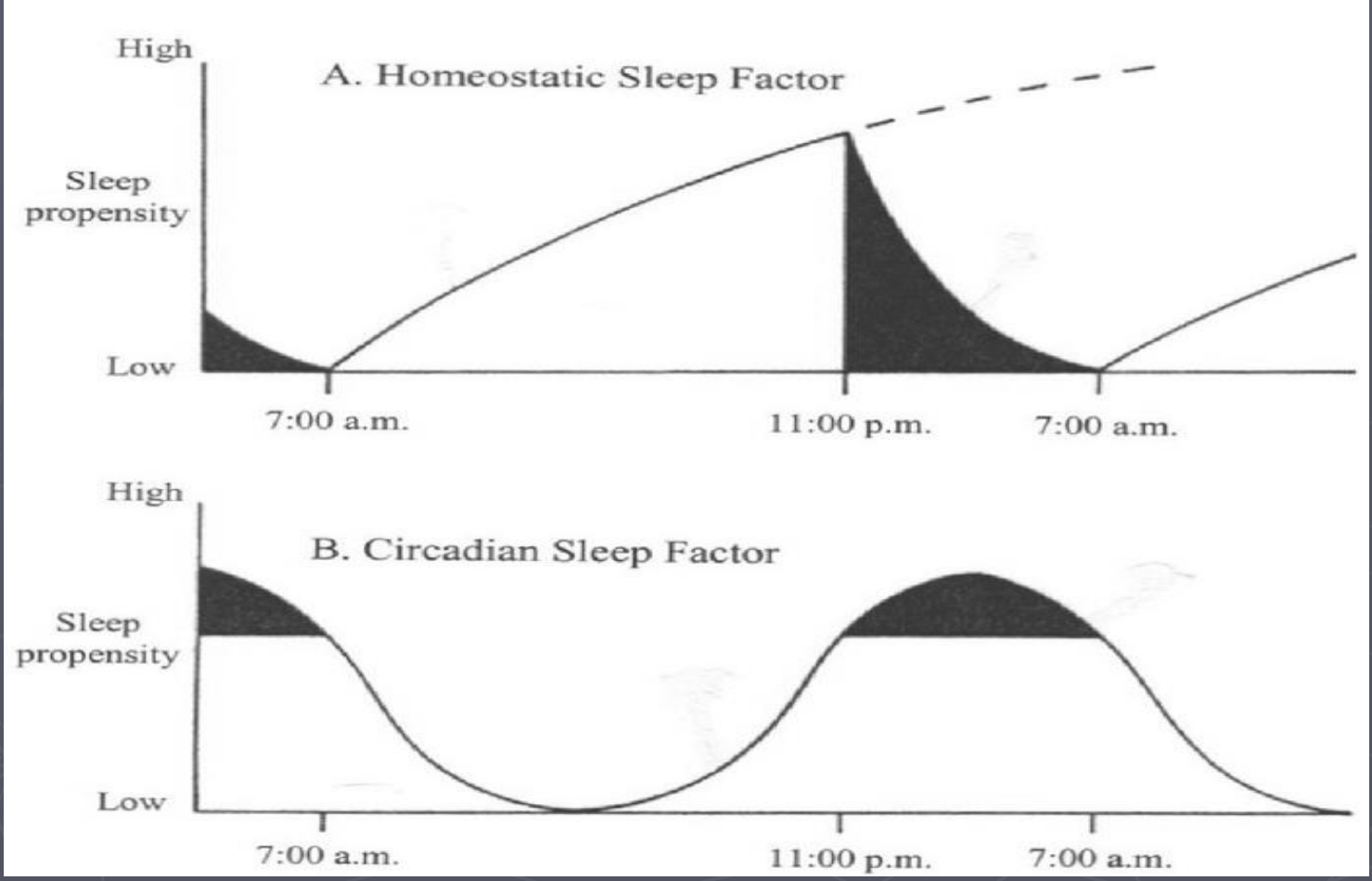
Homeostatic drive

- Has a ratio of approximately 1/3 sleep and 2/3 waking
- Sleep deprivation, acute or chronic, increases the homeostatic sleep drive and therefore sleepiness
- Hypothetically, the homeostatic sleep drive could be satisfied by sleep at any hour

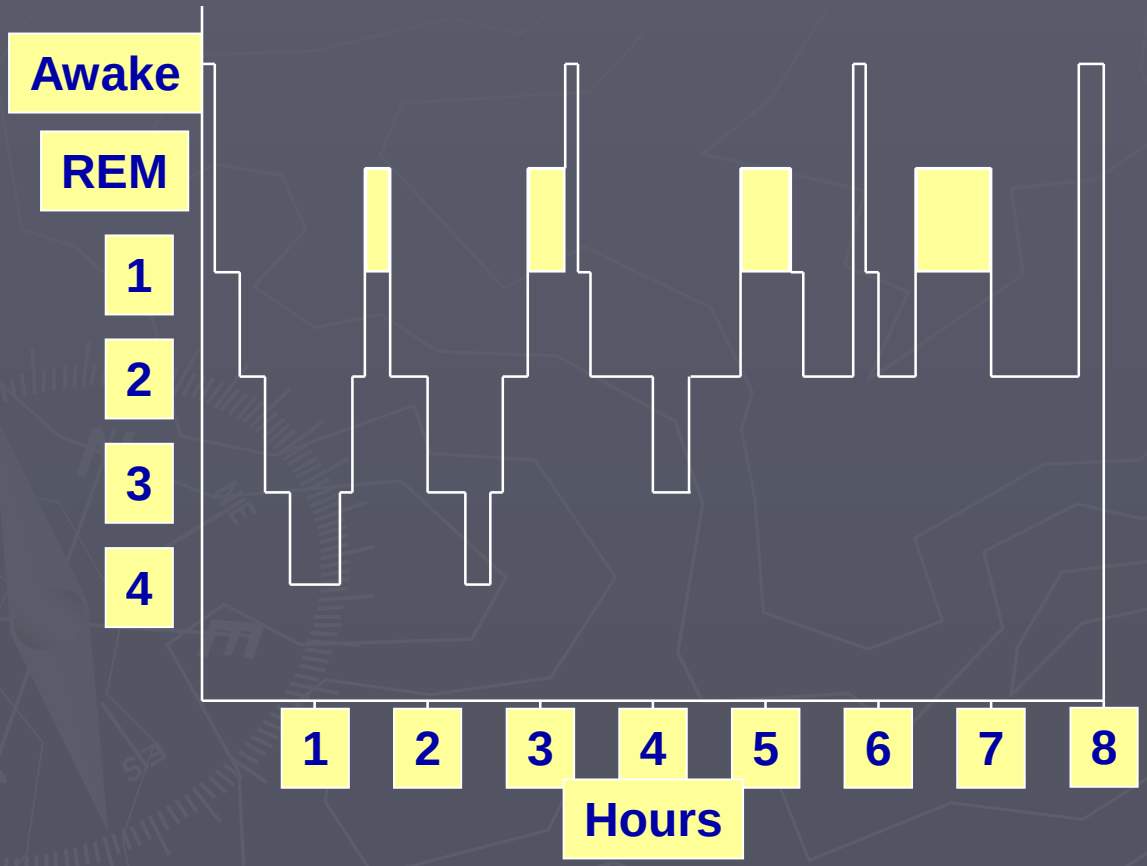
Circadian rhythm

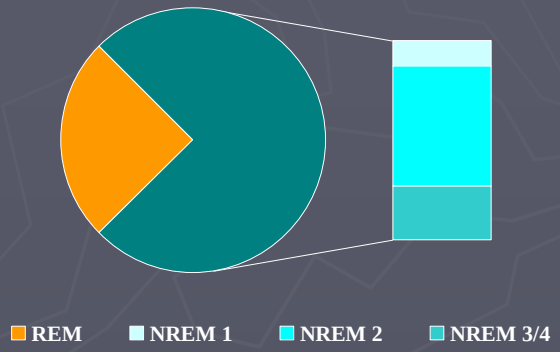
- Entrained and synchronized
- Timing of sleepiness promoted by the endogenous circadian clock
- Facilitates the rhythmic cycle of sleep at the same approximate nighttime hours (each day)
- Reinforced by the daily photoperiod, and possibly influenced by other light exposure

Sleep/Wake Cycle



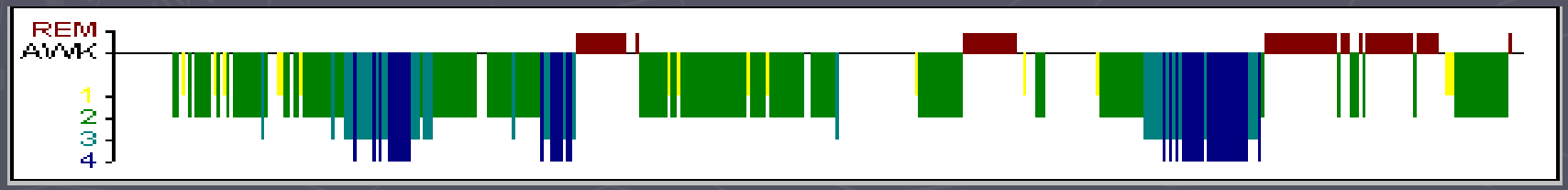
Normal Sleep





Normal Sleep Architecture

- Approximately 90 minute cycle including NREM and REM
- Slow wave dominates first third of night
- REM sleep dominates last third of night (early morning hours)
- REM sleep: 20-25% total sleep time
  - Can see REM-rebound with sleep deprivation, abrupt withdrawal of REM suppressants



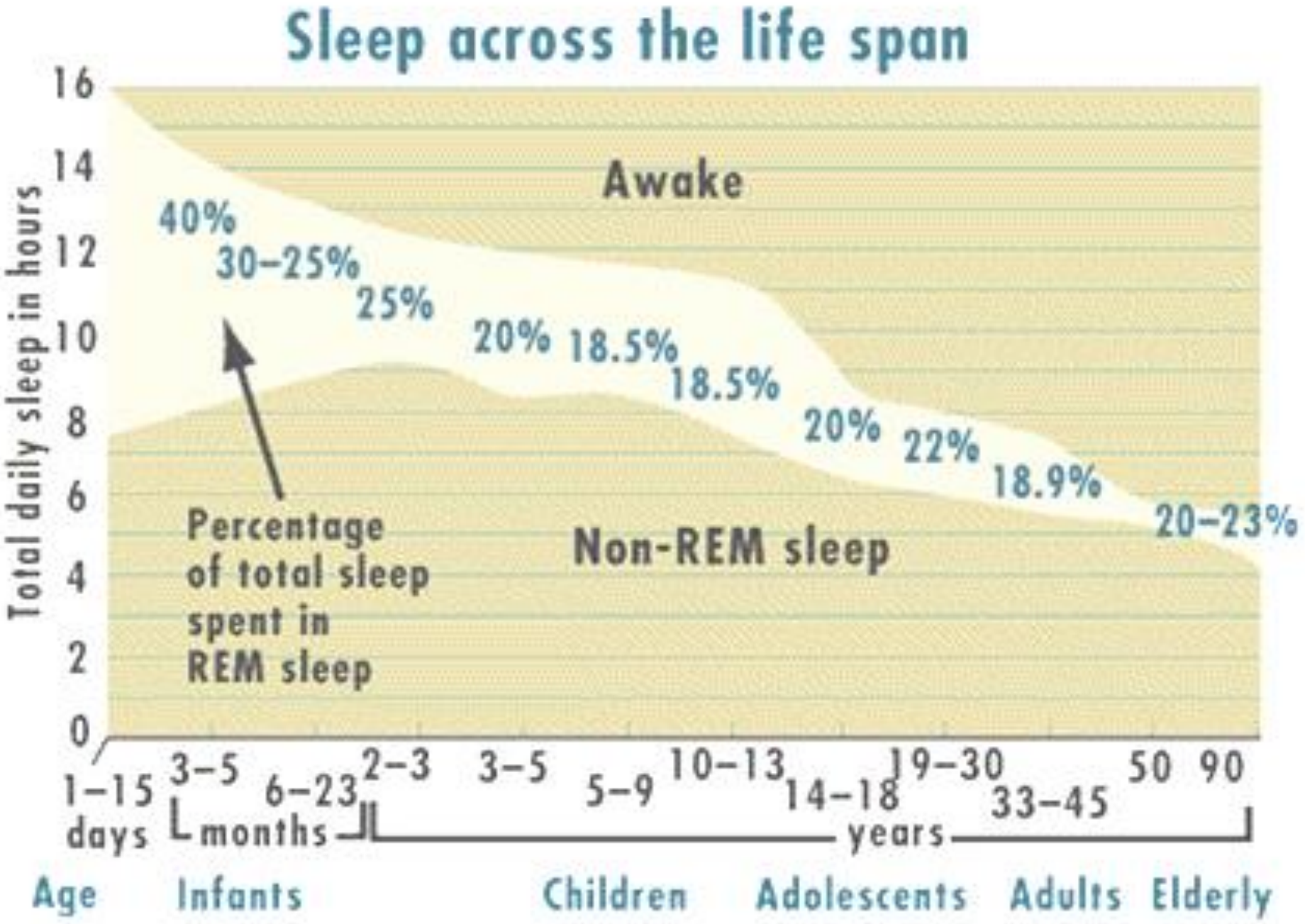

Sleep across the life span
Total daily sleep in hours
16
14
12
10
8
6
4
2
0
Awake
Non-REM sleep
40%
30–25%
25%
20%
18.5%
18.5%
20%
22%
18.9%
20–23%
Percentage of total sleep spent in REM sleep
1–15 days
3–5 months
6–23 months
2–3
3–5
5–9
10–13
14–18
19–30
33–45
50
90
years
Age
Infants
Children
Adolescents
Adults
Elderly

Table 9.1 Brain Structures for Arousal and Sleep

Structure	Neurotransmitter(s) It Releases	Effects on Behavior
Pontomesencephalon	Acetylcholine, glutamate	Increases cortical arousal
Locus coeruleus	Norepinephrine	Increases information storage during wakefulness; suppresses REM sleep
Basal forebrain		
Excitatory cells	Acetylcholine	Excites thalamus and cortex; increases learning, attention; shifts sleep from NREM to REM
Inhibitory cells	GABA	Inhibits thalamus and cortex
Hypothalamus (parts)	Histamine	Increases arousal
	Orexin	Maintains wakefulness
Dorsal raphe and pons	Serotonin	Interrupts REM sleep

Introduction to the PSG

Types of sleep studies

- **Diagnostic** – overnight study
  - In-lab (OSA, CSA, PLMD/RLS, RBD, parasomnias, seizure)
  - Home sleep study (just for OSA)
- **PAP titration** - Once a patient is identified as having sleep apnea another study is performed in which the technician adjusts the CPAP level during the test/mask fitting [CPAP/Bilevel PAP +/- ST/ASV]
- **Split Night** - Combines a diagnostic study and a CPAP titration study into one night. The patient is diagnosed during the first half of the night (AHI >30); CPAP applied the second half if required by protocol
- **MSLT** - Multiple Sleep Latency Test
- **MWT** – Maintenance of Wakefulness Test

Indications for PSG

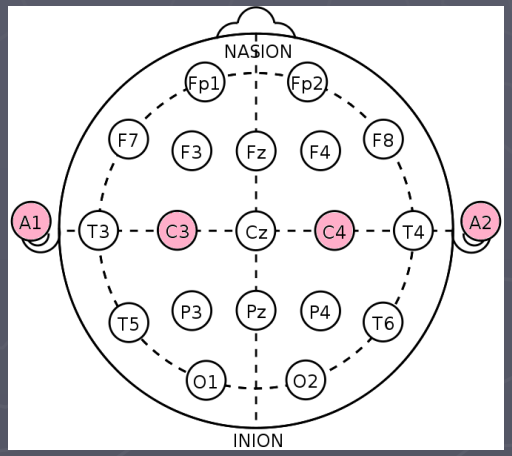
- Excessive daytime sleepiness (EDS)
- Unexplained behavioral events in sleep
- Insomnia or unexplained awakenings
- Sleep-related breathing disturbances
- Effect of treatment for sleep disorders

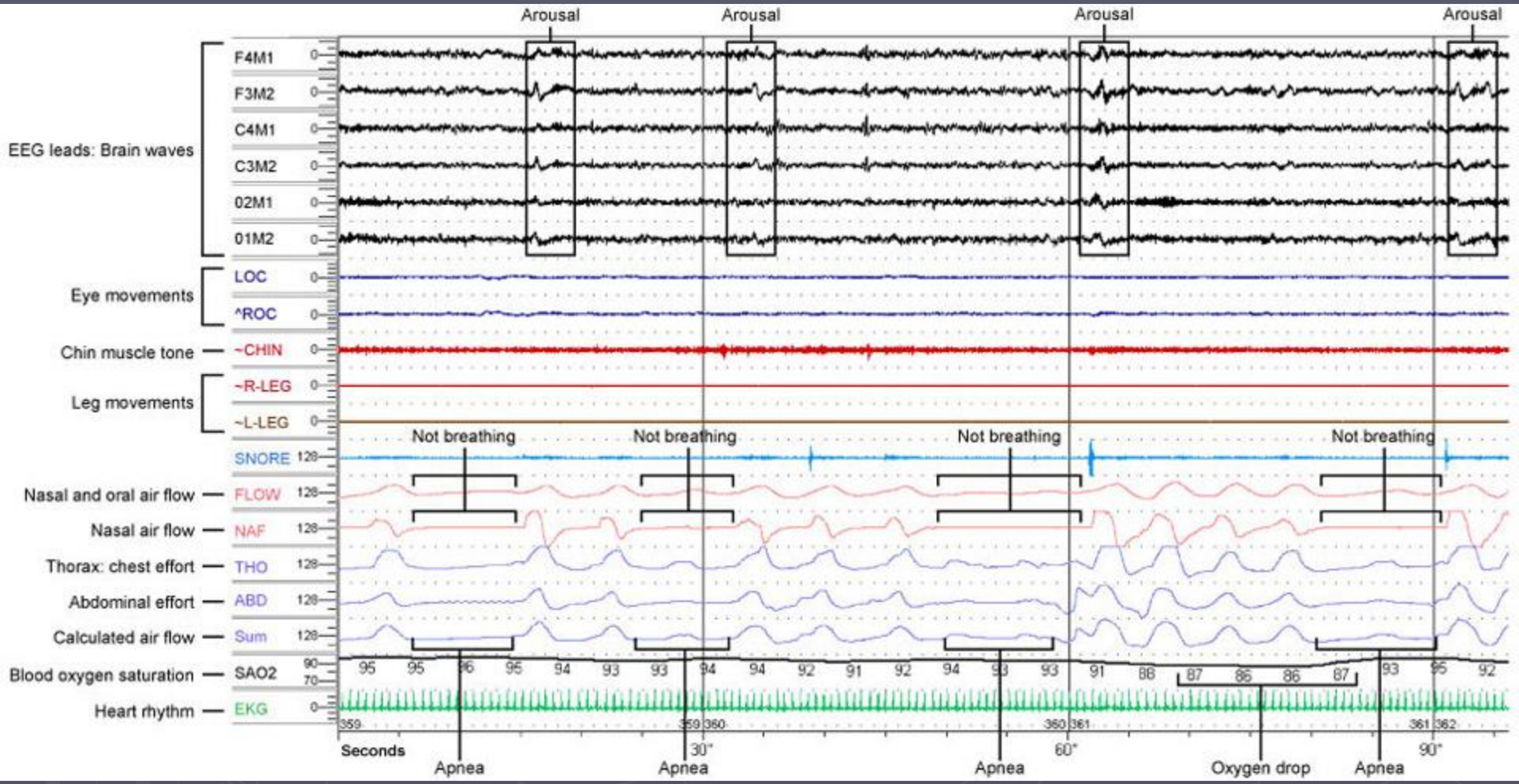
PSG Parameters

- EEG
- EOG (electro-oculogram)
- Chin EMG
- Leg EMG
- ECG
- Airflow
- Effort
- Oxygen
- Body position

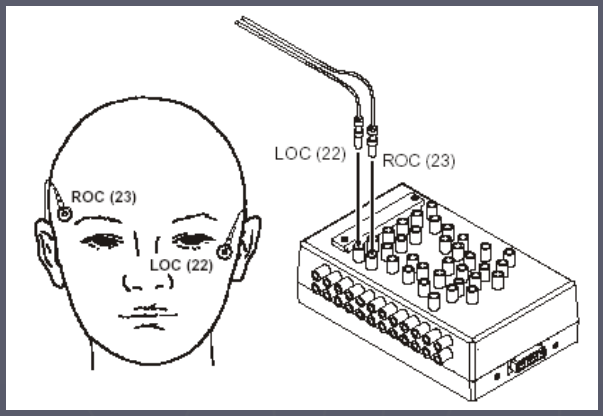
EEG

- Minimum of 3 EEG derivations required to sample from frontal, central and occipital regions
- Recommended derivations
  - F4-M1
  - C4-M1
  - O2-M1
  - F3, C3, O1 and M2 placed for backup
- Alternative derivations
  - Fz-Cz
  - Cz-Oz
  - C4-M1
  - Fpz, C3, O1 and M2 placed for backup
- Additional derivations required for evaluation of seizures
  - International 10-20 electrode placement (seizure protocol)
- Paper speed: 10 mm/sec (30 sec epochs)

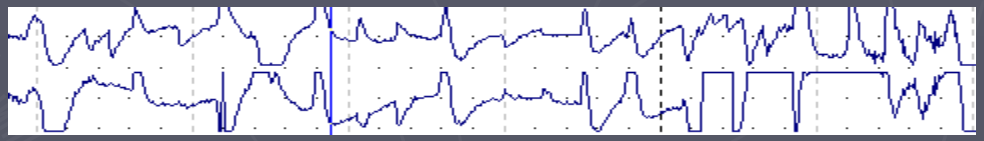



Arousal
Arousal
Arousal
Arousal
EEG leads: Brain waves
F4M1
F3M2
C4M1
C3M2
02M1
01M2
Eye movements
LOC
^ROC
Chin muscle tone
~CHIN
Leg movements
~R-LEG
~L-LEG
Not breathing
Not breathing
Not breathing
Not breathing
SNORE
Nasal and oral air flow
FLOW
Nasal air flow
NAF
Thorax: chest effort
THO
Abdominal effort
ABD
Calculated air flow
Sum
Blood oxygen saturation
SAO2
Heart rhythm
EKG
Seconds
30"
60"
90"
Apnea
Apnea
Apnea
Oxygen drop
Apnea

EOG

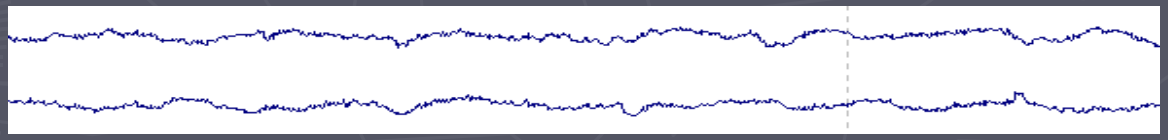


- EOG records voltage changes caused by EM
- Recommended derivations:
  - E1-M2 (E1 placed 1 cm *below* LOC)
  - E2-M2 (E2 placed 1 cm *above* ROC)
- Alternative derivations:
  - E1-FPz (E1 placed 1 cm below/lateral to LOC)
  - E2-FPz (E2 placed 1 cm above/lateral to ROC)
- Wake:



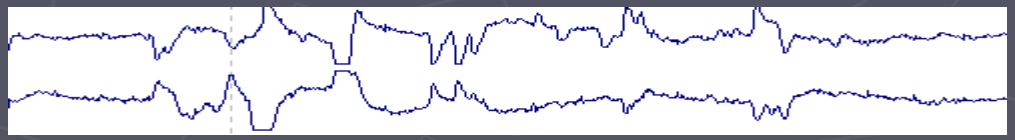
random, high amplitude

- Stage 1:



slow rolling, conjugate, regular

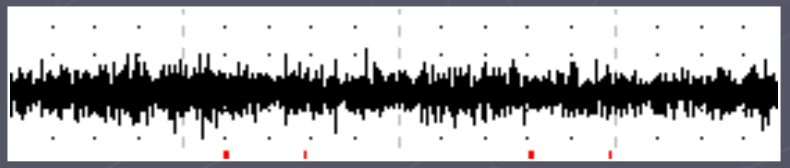
- REM:



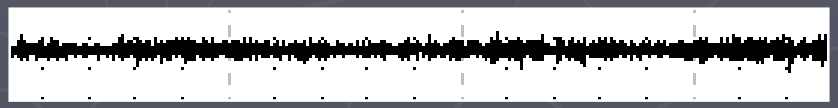
conjugate, irregular, sharply peaked EM

EMG

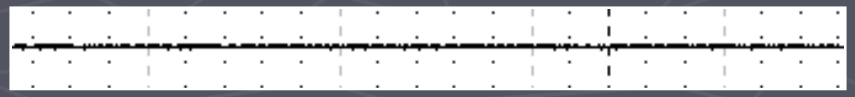
- Recorded as the potential between two surface electrodes placed several centimeters apart
- Typically, the chin (submental) muscle is used because it exhibits large differences during sleep, aiding in the identification of stages
- Wake - high activity



- Sleep - lower activity

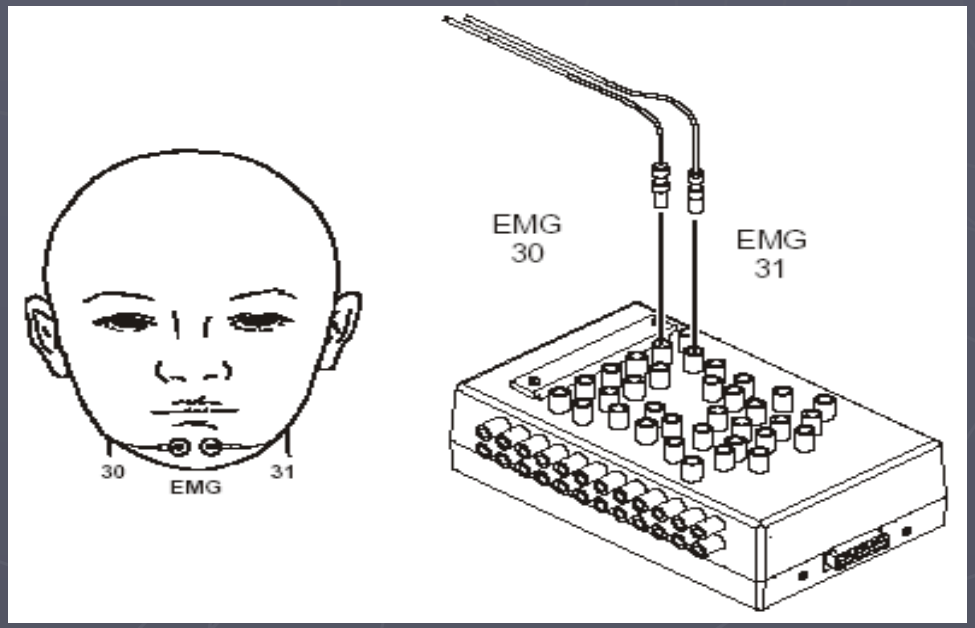


- REM sleep - paralysis of skeletal muscles



EMG Placement

- Chin Electrode placement (2 required)
  - Midline 1 cm above inferior edge of mandible (optional)
  - 2 cm below inferior edge of mandible to right of midline
  - 2 cm below inferior edge of mandible to left of midline

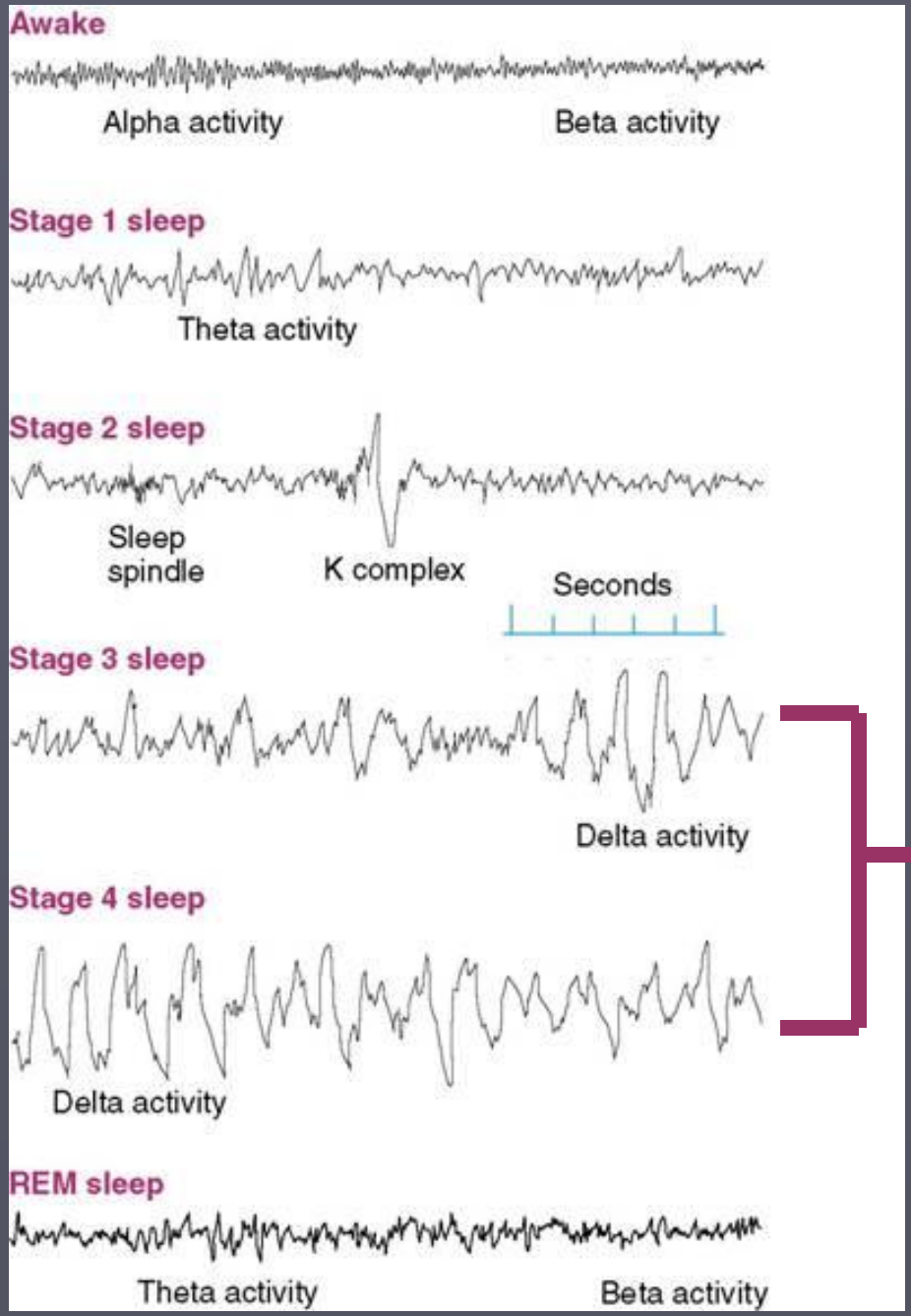


REM vs. NREM Sleep

- Non-REM
  - Physical restoration
  - Driven by homeostatic drive
  - Quiet brain, active body
- REM
  - Mental restoration/memory
  - Driven by circadian rhythm
  - Active brain, quiet body

REM vs. NREM Sleep

Physiologic Variable	NREM	REM
Heart rate	Regular	Irregular
Respiratory rate	Regular	Irregular
Blood pressure	Regular	Variable
Skeletal muscle tone	Preserved	Absent
Brain 0_2 consumption	Reduced	Increased
Ventilatory response	Normal	Reduced
Temperature	Normal	Poikilothermic



Overview of sleep stages

Combined to become N3

2007 AASM scoring guidelines

Respiratory Variables

- Respiratory effort (thoracic and abdominal belts)
- Airflow (thermistor, thermocouple, nasal pressure)
- SpO2 (pulse oximetry)
- Snoring microphone
- Optional signals
  - ETCO2
  - tcCO2

Airflow methods

- **Qualitative**
  - Thermal sensors
    - Measure temperature changes
    - Breathe in cool air, breathe out warm air; measures the difference in temperature, but can underestimate
    - Measures apneas
  - CO_2 monitors
    - End tidal CO_2 monitor, Transcutaneous
  - Nasal pressure
    - More sensitive, detects hypopneas

- **Quantitative**
  - Pneumotachography
    - Gold standard
    - Place a face mask over pt' s face and measure tidal volume, uncomfortable so not commonly used

Effort methods

- Qualitative
  - Piezo-electric belts (crystals embedded in belt that sense movement)
  - Intercostal EMG
- Semi-quantitative
  - Respiratory inductive plethysmography (RIP): can give tidal volume, but not very accurately
- Gold standard: Esophageal pressure (balloon inserted into lower esophagus)

Other Variables Typically Recorded

- ECG
- Leg movement: EMG
- Video
- Body position

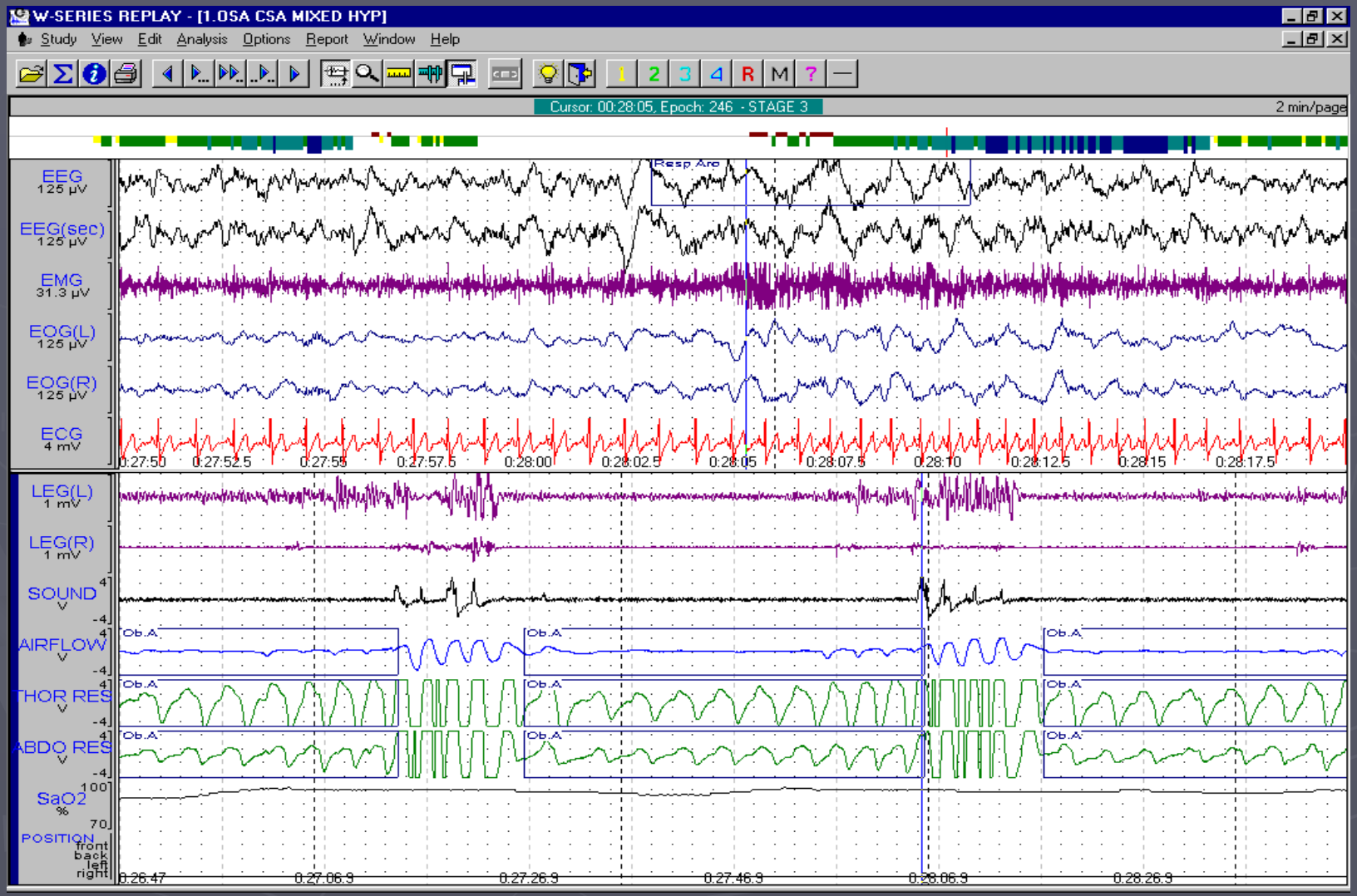
Respiratory Events

- Apneas – absence of airflow
  - Drop in peak thermal sensor excursion by >90% of baseline
  - Duration of events lasts at least 10 seconds
  - At least 90% of event's duration meets the amplitude reduction criteria for apnea
- Hypopneas – reduced airflow
  - Nasal pressure excursions drop by at least 30% from baseline
  - Duration at least 10 seconds
  - There is a ≥3% desaturation or an arousal (4% for Medicare)

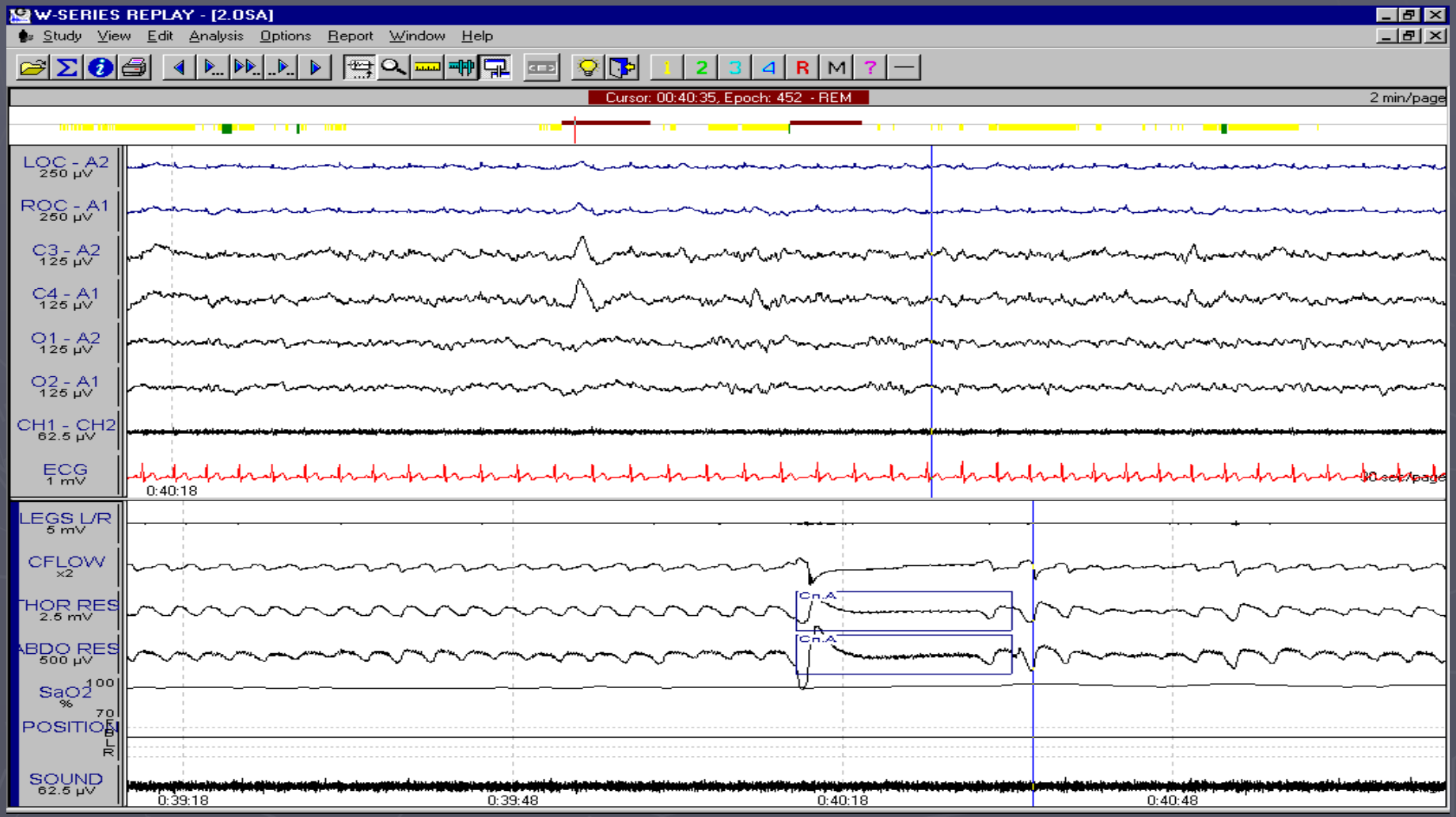
Types of Apnea

- Obstructive:
  - Associated with continued or increased inspiratory effort, but absent airflow
- Central:
  - Absent inspiratory effort and airflow
- Mixed:
  - Absent inspiratory effort initially, followed by resumption of effort in the second portion of the event

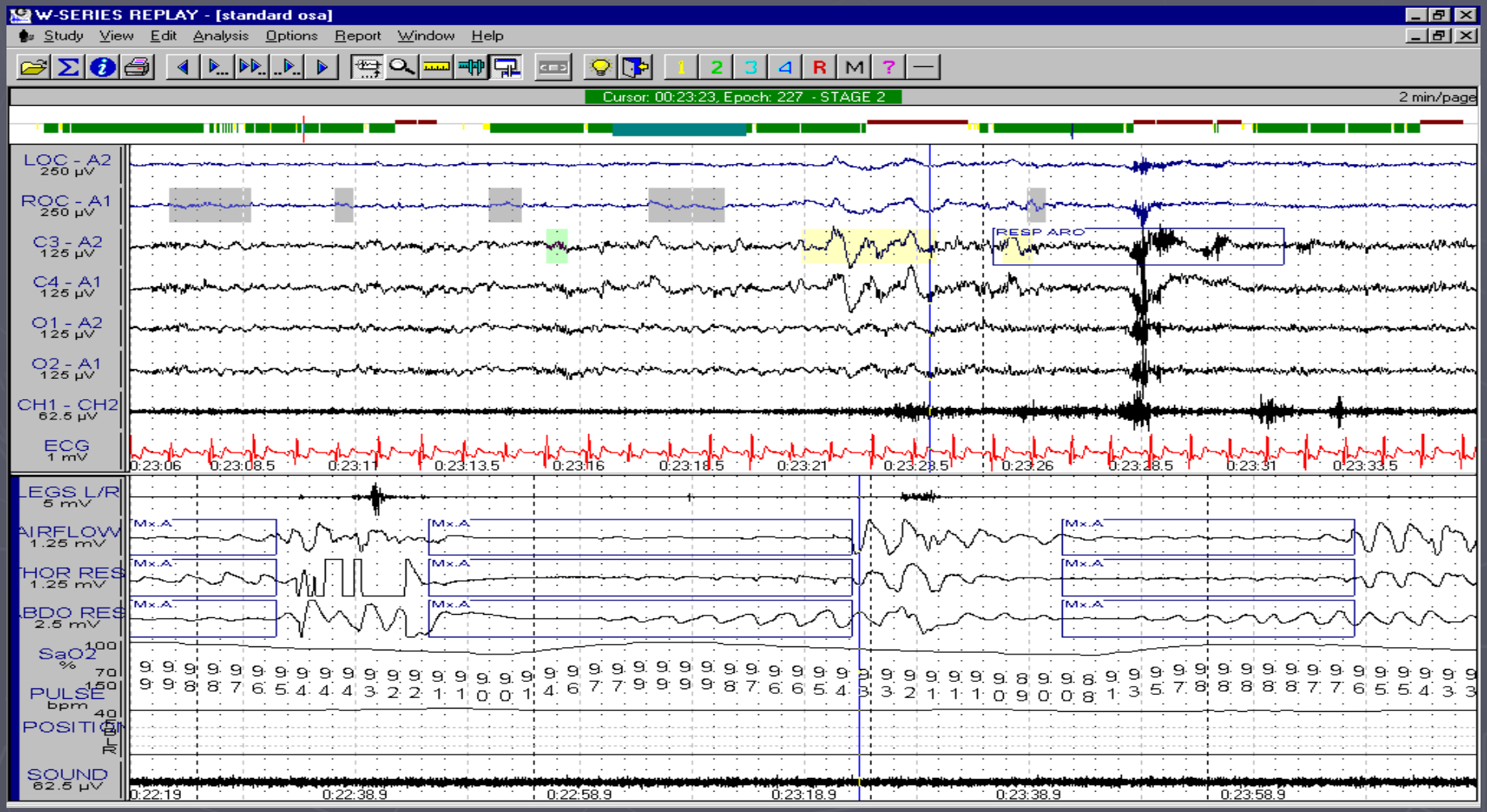
Example - Obstructive Apnea



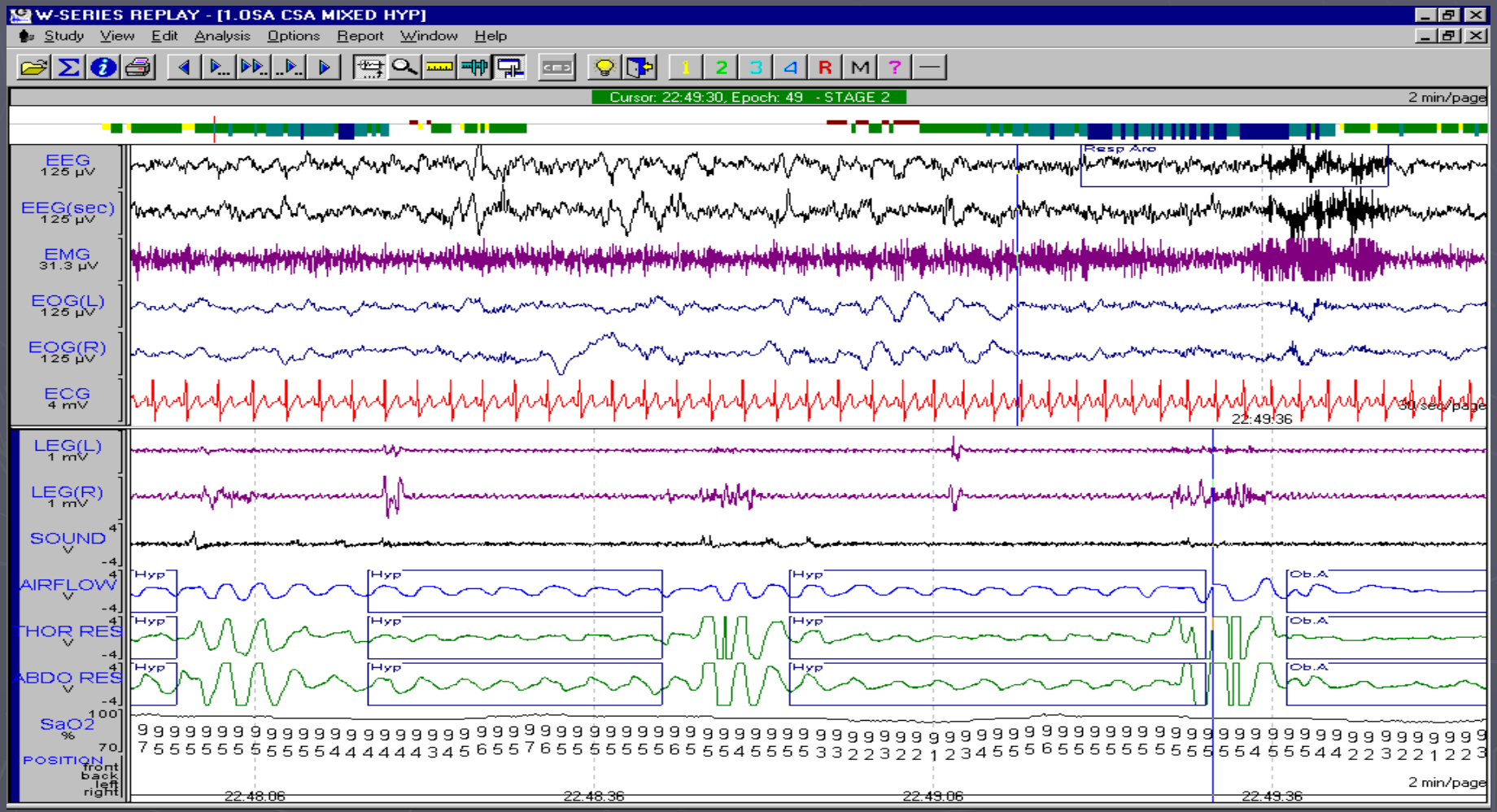
Example - Central Apnea



Example - Mixed Apnea

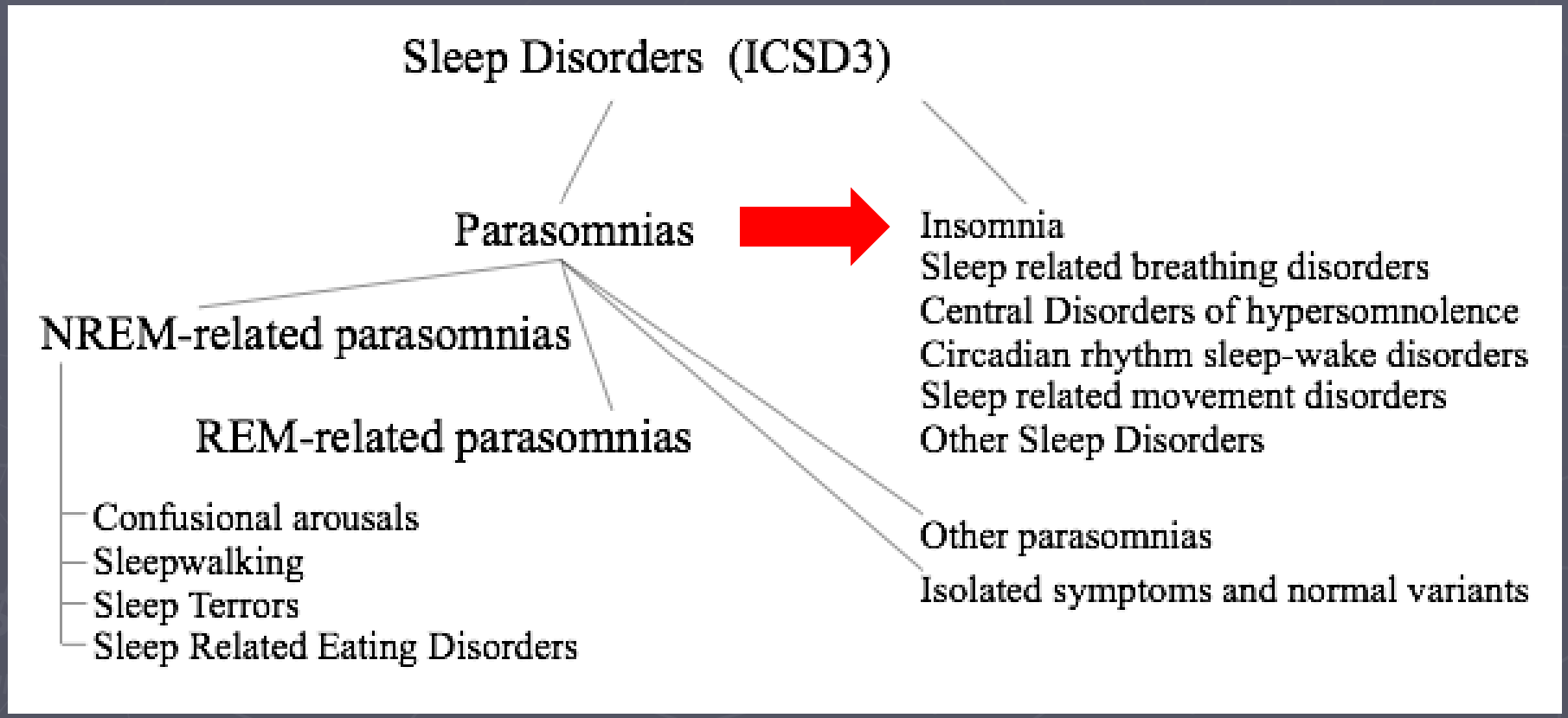


Example - Hypopnea



Common Sleep disorders: An overview via Cases

ICSD-3 (2014)


Sleep Disorders (ICSD3)
Parasomnias
NREM-related parasomnias
REM-related parasomnias
Confusional arousals
Sleepwalking
Sleep Terrors
Sleep Related Eating Disorders
Insomnia
Sleep related breathing disorders
Central Disorders of hypersomnolence
Circadian rhythm sleep-wake disorders
Sleep related movement disorders
Other Sleep Disorders
Other parasomnias
Isolated symptoms and normal variants

Case 1

- 69 yo F, travel agent presents with insomnia x 15+ years
- PMH: hypothyroidism, OA, MVP, irritable bowel syndrome, migraines headaches
- Rx: levothyroxine, sumatriptan
- Currently rx'd temazepam 30 mg qhs for insomnia but c/o morning grogginess
- Other tried rx:
  - lorazepam 1-2 mg, diazepam 2 mg – initially worked, lost effectiveness
  - zolpidem 10-20 mg – nocturnal eating, sleep walking
  - Trazodone, imipramine, paroxetine, seroquel – "like a zombie"

Case 1

- Sleep routine
  - BT: 22:30 (admits to reading but in lounge chair next to bed)
  - SL: 45-60 min
  - Awakenings: 1-3 x with variable SL after each (10-60 min), admits to rumination (stressors: finances, parents)
  - WT: 7 AM
  - Estimated TST: 5-6 hours (desires 7 hours)
- No symptoms to suggest OSA, RLS/PLMD, parasomnias, REM behavior disorder
- No psychiatric co-morbidities but family label her a "worry-wart"
- No drug or excessive caffeine/ETOH use, non-smoker

Case 1

- Exam
  - BMI 23.5
  - BP 126/78, pulse 72, RR 13, O2 sat 97% RA
  - Friedman tongue position 1 (Mallampati 1), no nasal obstruction
  - Rest of exam nl (cardio/lungs/neuro/affect/etc)
- Questionnaires
  - Epworth sleepiness score: 6
- Lab work: TSH, CBC, Vit D, B12, Fe all wnl
- PSG 1 year ago, showed no OSA
  - Sleep latency 66 min, TST 246 min, SE 73%, no N3 sleep, 15% REM

Differential Diagnosis?

Case 1

- CHRONIC INSOMNIA
  - Sleep onset and sleep maintenance
  - Treatment:
    - Both behavioral + pharmacological treatments are reasonable
    - Behavioral:
      - Sleep restriction in bed
      - Delaying bedtime until sleepy
      - Stimulus control (getting out of bed when unable to sleep)
      - Regular BT/WT (even on weekends)
    - Pharmacological:
      - Benzodiazepines can be used for <3months (with co-morbid anxiety) but recommended as short-term therapy; >6 months → develop tolerance and dependence
      - Other anxiolytics with SE of sedation: TCA's
      - GBP (concomitant tx for migraines/OA pain), "Vitamin G"
      - Other sedative-hypnotics (next slide)

Prescription Sedative-Hypnotics

Drug	Duration	Onset of action	Hypnotic dose	Half life
Zaleplon (Sonata)	Short	15-30 min	10-20 mg	1 hr
Zolpidem (Ambien)	Short	30 min	5-10 mg	2.5 hrs
Ramelteon (Rozerem)	Short	30-45 min	8mg	1-2.6 hours
Triazolam (Halcion)	Short	15-30 min	0.125-0.25 mg	2.9 hrs
Suvorexant(Belsomnra)	**Intermediate**	**30-60 min**	**10-20 mg**	**12 hours**
Eszopiclone (Lunesta)	Intermediate	30 min	1-3 mg	6 hours
Oxazepam (Serax)	Intermediate	45-60 min	15-30 mg	8.0 hrs
Estazolam	Intermediate	15-60 min	1-2 mg	10-24 hrs
Lorazepam (Ativan)	Intermediate	30-60 min	1-2 mg	14 hrs
Temazepam (Restoril)	Intermediate	45-60 min	15-30 mg	11 hrs
Clonazepam (Klonopin)	Long	30-60 min	0.5 mg-1 mg	23 hrs
Diazepam (Valium)	Long	15-30 min	5-10 mg	43 hrs*
Flurazepam (Dalmane)	Long	30-60 min	15-30 mg	74 hrs*

Insomnia

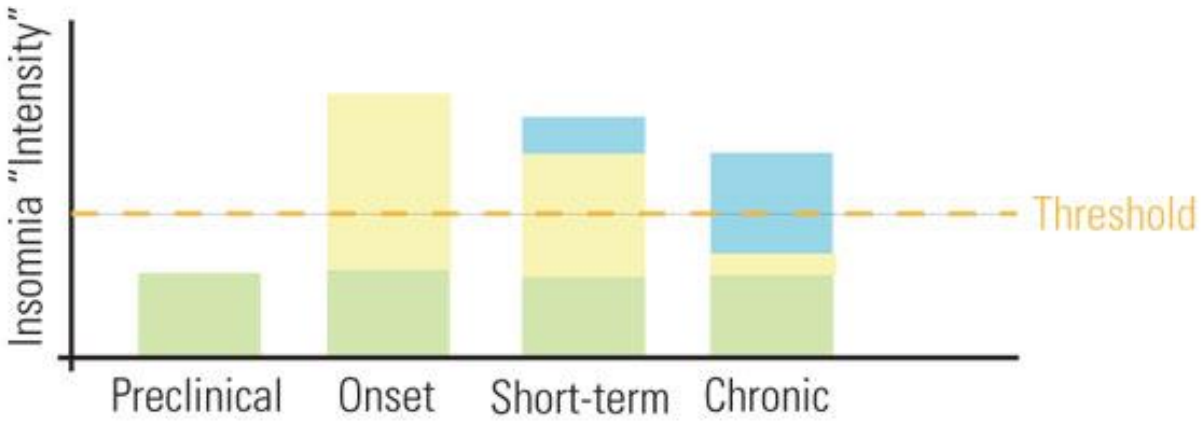
- 2012 Sleep In America Poll by NSF – 58% of American Adults experience insomnia a few nights a week or more
- Insomnia definition: sleep latency >30 min + dysfunction
- ICSD-3 recognizes 3 types:
  - Short-term – "adjustment" or "transient", <3 mos
  - Chronic – at least 3x/week for >3 mos
  - Other – catch-all group
- 3 patterns
  - Sleep Onset Insomnia
  - Sleep Maintenance Insomnia
  - Terminal Insomnia (Early Morning Awakening)

Spielman's model for insomnia

FIGURE

A MODEL OF CHRONIC INSOMNIA[2-4]

Predisposing Factors	Precipitating Factors	Perpetuating Factors
• Biologic traits	• Medical illness	• Excessive time in bed
• Psychological traits	• Psychiatric illness	• Napping
• Social factors	• Stressful life events	• Conditioning



Erman MK. *Primary Psychiatry.* Vol 14, No 7. 2007.

Treatment of Insomnia

Depends on the Stage of Insomnia

- Treatment of Pre-Morbid Conditions
  - Sleep Hygiene
- Treatment of Precipitating Conditions
  - Psychiatric Counseling
- Treatment of Perpetuating Conditions
  - Cognitive Behavioral Therapy
    - Relaxation Techniques
    - Breathing Techniques
  - Medications – ok but SHORT TERM ONLY

Good sleep hygiene tips

Sleep Hygiene *Do's* and *Don'ts*

Do:

- Establish a regular bedtime and rise time
- Exercise in the late afternoon or early evening
- Take a hot bath a couple of hours before bedtime
- Establish a comfortable sleep environment (e.g., bed, and bedding)
- Sleep in a dark, quiet area that is temperature and humidity controlled
- Establish a relaxing pre-sleep routine that you use every night before sleep, such as washing your face, getting into pajamas, reading or listening to soft music before turning the lights out.

Don't:

- Take daytime naps
- Use stimulants such as caffeine and nicotine
- Drink alcohol before bedtime
- Go to bed too hungry or too full
- Eat offensive foods, such as spicy or acidic foods (e.g., orange juice) before bed
- Try too hard to fall asleep
- "Watch the clock"
- Take prescription and over-the-counter medications that might be stimulating (check with your doctor)

Insomnia inducing Rx

Table 2. Medications and Substances That May Contribute to Insomnia	
Analgesics	Opioids, NSAIDs
Antidepressants	SSRIs, venlafaxine, duloxetine, MAOIs
Stimulants	Caffeine, methylphenidate, amphetamines, cocaine
Decongestants	Phenylephrine, pseudoephedrine
Cardiovascular	β-blockers, diuretics
Pulmonary	Albuterol, theophylline

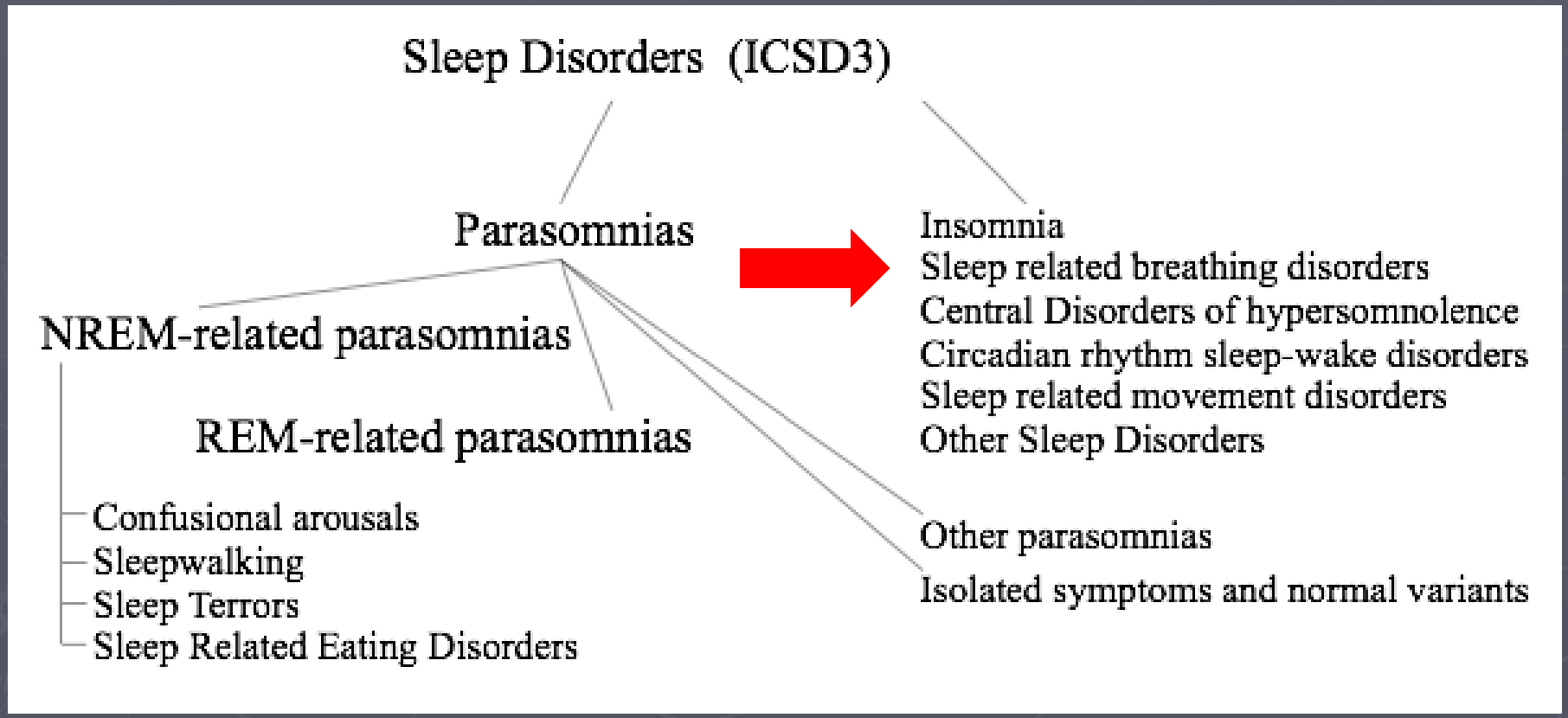
MAOI, monoamine oxidase inhibitor; **NSAID**, nonsteroidal anti-inflammatory drug; **SSRI**, selective serotonin reuptake inhibitor

Based on references 2, 7, 8, and 10.

Table 1. Sleep Disorder Differential Diagnosis of Insomnia

Disease	*Characteristics*	*Notes*
Sleep–related breathing disorders		
The obstructive sleep apnea syndrome	Upper airway obstruction during inspiration in sleep.	History of snoring, witnessed pauses in respiration, and daytime sleepiness. Patients may report non-restful sleep or insomnia. Polysomnography is necessary for diagnosis.
The central sleep apnea syndrome	Repetitive pauses in breathing during sleep without upper airway occlusion.	History of congestive heart failure or central nervous system disease. Polysomnography is necessary for diagnosis.
Sleep–related movement disorders		
The restless legs syndrome	Uncomfortable or restless feeling in legs most prominent at night and at rest; alleviated by movement.	Occurs in up to 10% of the general population. Approximately 80% of patients with this syndrome also have periodic leg movement disorder on polysomnography, although polysomnography is not necessary for diagnosis.
Periodic limb movement disorder	Repetitive stereotypic leg movement in sleep and during quiet wakefulness.	Strongly associated with the restless legs syndrome. Polysomnography is necessary for diagnosis.
Nocturnal leg cramps	Pain in calf or foot resulting in awakening from sleep.	Painful cramp awaken the patient from sleep. Predisposing factors include diabetes, exercise, pregnancy, and metabolic and endocrine abnormalities.
Circadian rhythm sleep–wake disorders		
Time zone change syndrome (jet lag)	Travel leads to reports of poor sleep, daytime sleepiness, or both.	History of recent travel across multiple time zones.
Shiftwork sleep disorder	Insomnia as a consequence of shiftwork. Sleep occurs at times counter to normal circadian rhythm and social and environmental factors.	History of insomnia associated with shiftwork; this disorder also affects persons who permanently work the night shift.
The delayed sleep-phase syndrome	Delay of the major sleep phase relative to clock time.	History of sleep-onset insomnia and difficulty awakening at the desired time. Patients have no difficulty maintaining sleep once asleep. Sleep log and actigraphy can aid diagnosis.
The advanced sleep-phase syndrome	The major sleep phase is advanced relative to clock time.	Inability to stay awake until desired bedtime and early-morning awakening. Occurs most commonly in elderly. Sleep log and actigraphy can aid diagnosis.
Parasomnias related to non–rapid eye movement	Include confusional arousals, sleepwalking, sleep terrors, and sleep-related eating disorders.	Disorders of arousal that may be a cause of disrupted sleep. Sleep history and input from bed partner or family may aid in identification.

ICSD-3 (2014)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Parasomnias
NREM-related parasomnias
REM-related parasomnias
Confusional arousals
Sleepwalking
Sleep Terrors
Sleep Related Eating Disorders
Insomnia
Sleep related breathing disorders
Central Disorders of hypersomnolence
Circadian rhythm sleep-wake disorders
Sleep related movement disorders
Other Sleep Disorders
Other parasomnias
Isolated symptoms and normal variants

Case 2

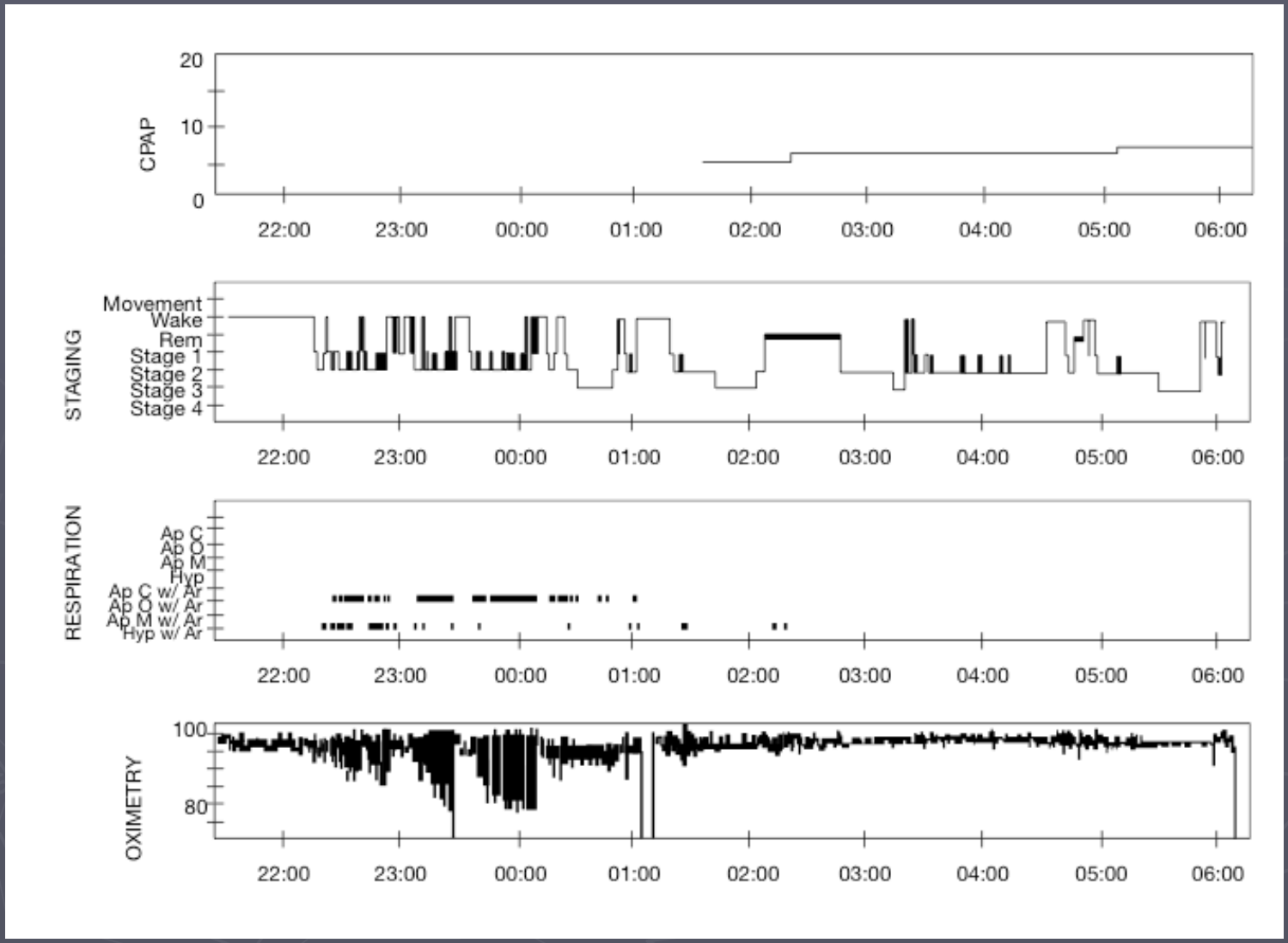
- 73 yo RH German man
- PMH: HTN, HPL, CAD, CHF (EF 30%), V-fib s/p ICD, paroxysmal AF
- Recent left MCA stroke secondary to AF-related cardiac emboli with residual right HP and expressive aphasia

Case 2

- Noted to have abnormal overnight oximetry while in stroke rehab

  Evidence of periodic desaturations in saw-tooth pattern with lowest O2 saturation of 82%, a pattern suggestive of sleep apnea.

Split study


CPAP
20
10
0
22:00
23:00
00:00
01:00
02:00
03:00
04:00
05:00
06:00
STAGING
Movement
Wake
Rem
Stage 1
Stage 2
Stage 3
Stage 4
RESPIRATION
Ap C
Ap O
Ap M
Hyp
Ap C w/ Ar
Ap O w/ Ar
Ap M w/ Ar
Hyp w/ Ar
OXIMETRY
100
80

Case 2

- Pt used CPAP 8 cm H2O
- No stroke recurrence
  - Patient symptomatically improved, able to cooperate with rehabilitation
  - NIHSS 12→5
- Downloaded data showed good compliance (88%) and efficacy (AHI 45→3)

Sleep Related Breathing Disorders

- Obstructive Sleep Apnea
  - Most common cause of EDS and sleep disruption
- Central Sleep Apnea
- Hypoventilation Syndromes

What is OSA?

"… characterized by repetitive episodes of upper airway obstruction that occur during sleep, usually associated with a reduction in blood oxygen saturation…" with associated features of daytime sleepiness and snoring.

What is OSA Syndrome?

- Apnea – Hypopnea Index (AHI) ≥5 events/hour in conjunction with symptoms
- What is a relevant AHI?
  - Consensus Statement 1999: "RDI of 5 (or greater) accompanied by symptoms..."

    Loube et al, Chest 1999
  - Medicare 2014: AHI ≥ 5 with symptoms, or HTN, CAD or CVA

AHI grading

Measures of Sleep Apnea Frequency

- Apnea Index
  - # apneas per hour of sleep
- Apnea / Hypopnea Index (AHI)
  - # apneas + hypopneas per hour of sleep

Chart 2. Classification of the severity level of OSAHS according to the American Academy of Sleeping Medicine -1997.

APNEA/HYPOPNEA INDEX	LEVEL
<5	NORMAL
5-15	MILD
15-30	MODERATE
>30	SEVERE

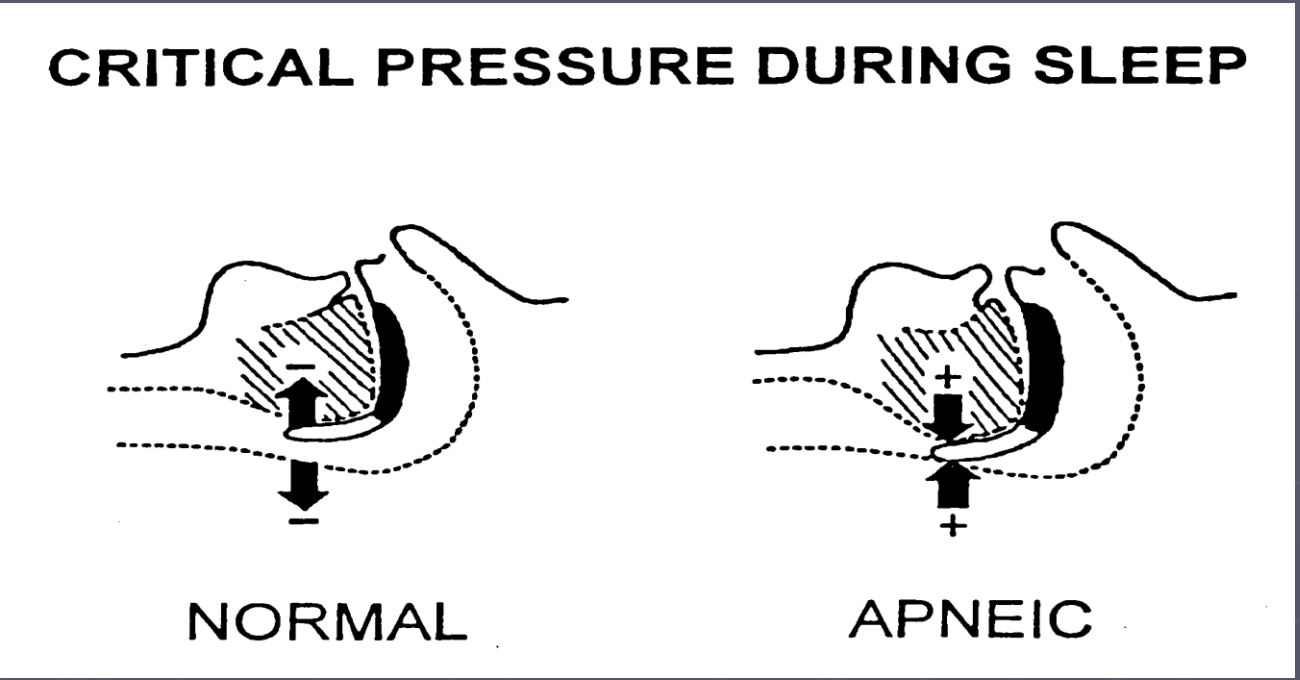
Prevalence of OSA

- Wisconsin Sleep Cohort Study
  - Population based study: 602 working subjects, aged 30-60 years studied with PSG
  - Definition OSAS: AHI ≥5 and hypersomnolence

	F	M
OSA	9%	24%
OSAS	2%	4%

Young et al, NEJM 1993

Pathophysiology of OSA



- Narrowing or collapse of the upper airway
- Decreased tidal volume → hypercapnia and hypoxia
- Increased respiratory effort
- Arousal opens airway
- Ensuing hyperpnea with hypocapnia and adequate oxygenation

Demographics of OSAS

- In younger, but not middle aged groups, OSAS has been reported to be more prevalent in AA's compared to Caucasians
- Despite lower BMI, Asians have a predisposition of OSA thought to be due to cranio-facial features
- Prevalence of OSA increases with age

Risk Factors for OSA

- Sleep Heart Health Study: male, age, BMI, neck girth, snoring, and witnessed apnea predict AHI >15

  Young et al. Arch IM.2002
- Craniofacial abnormalities - nasal obstruction, enlarged uvula/tongue/tonsils, long soft palate, retrognathia, micrognathia, brachycephaly (flat posterior head)
- Family History (increases risk of OSAS 2-4 fold)
- Co-morbid illness
  - cardiopulmonary disease (CHF, OHV)
  - metabolic disorders (hypothyroidism, acromegaly)
  - neurologic disorders (CVA, neuromuscular disorders e.g. MD)
  - Down's syndrome (macroglossia)
- Environmental Factors - tobacco use, ETOH, sedatives

Symptoms/Signs of OSA

- Snoring
- Witnessed apneas
- Daytime sleepiness
- Sleep fragmentation
- Night sweats
- Nocturia
- Dry mouth/sore throat
- Leg kicking while sleeping
- Morning headaches
- Mood changes
- Decreased libido
- Memory problems

- Obesity
- Associated diseases
  - Hypertension
  - Cardiac disease
  - Stroke
  - Glucose intolerance
  - Hypothyroidism
  - Acromegaly

STOP-BANG Questionnaire

TABLE 2. STOP-BANG Questionnaire for identifying patients with obstructive sleep apnea (OSA)

SNORE	Do you snore loudly? (Snoring can be heard through closed door)
TIRED	Do you feel tired, sleepy, fatigued, during daytime?
OBSERVED	Has anyone seen you stop breathing during sleep?
BLOOD PRESSURE	Do you have or are you being treated for high blood pressure?
BMI	Is your BMI > 35kg/m2?
AGE	Are you older than 50?
NECK CIRCUMFERENCE	Is your neck circumference greater than 40 cm?
GENDER	Are you a male?

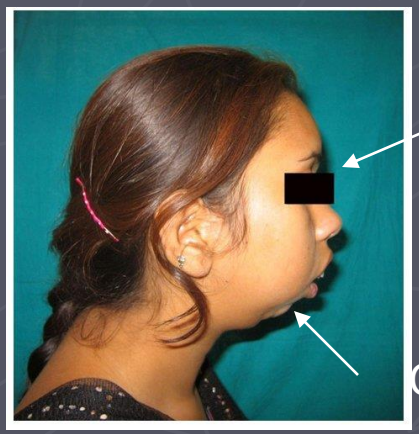
If the answer to three or more of these questions is "yes," a presumptive diagnosis of OSA can be made.

Modified from: Chung F, Elsaid H. Screening for obstructive sleep apnea before surgery: why is it important? *Current Op Anaesthesiol.* 2009;22: 405–411.

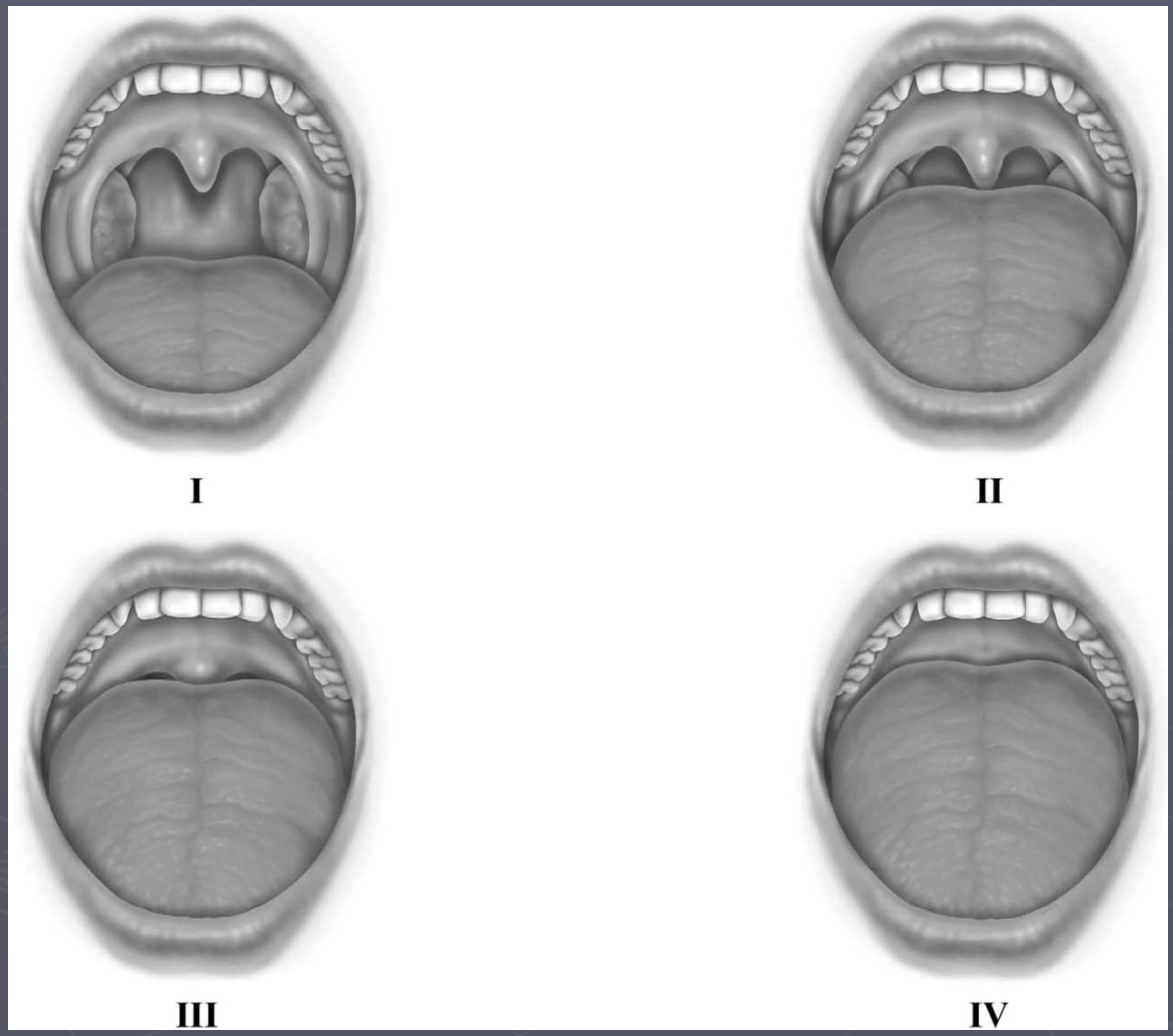
Clinical Examination

- Vital signs (hypertensive, arrhythmias)
- Obese (BMI >30)
  - 40% of those with BMI >40 have OSAS and 50% of those with BMI >50 have OSAS

    Kripke et al. Sleep 1997.
- Neck circumference
  - ≥40 cm associated with sensitivity of 61% and specificity of 93% for OSAS
  - Men >17 inches, women >16 inches
- Oral airway
  - Retrognathia (narrows the upper airway behind the base of the tongue)
  - Dental malocclusion and overlapping teeth (indicated small oral cavity)
  - Macroglossia
  - Edema and erythema of the uvula
  - Elongated soft palate
  - Narrow high arched palate
  - Tonsillar hypertrophy
  - Lateral airway narrowing
- Nasal airway
  - Nasal valve collapse with sniff test
  - Nare size and asymmetry
  - Septal deviation
  - Enlarged inferior turbinates

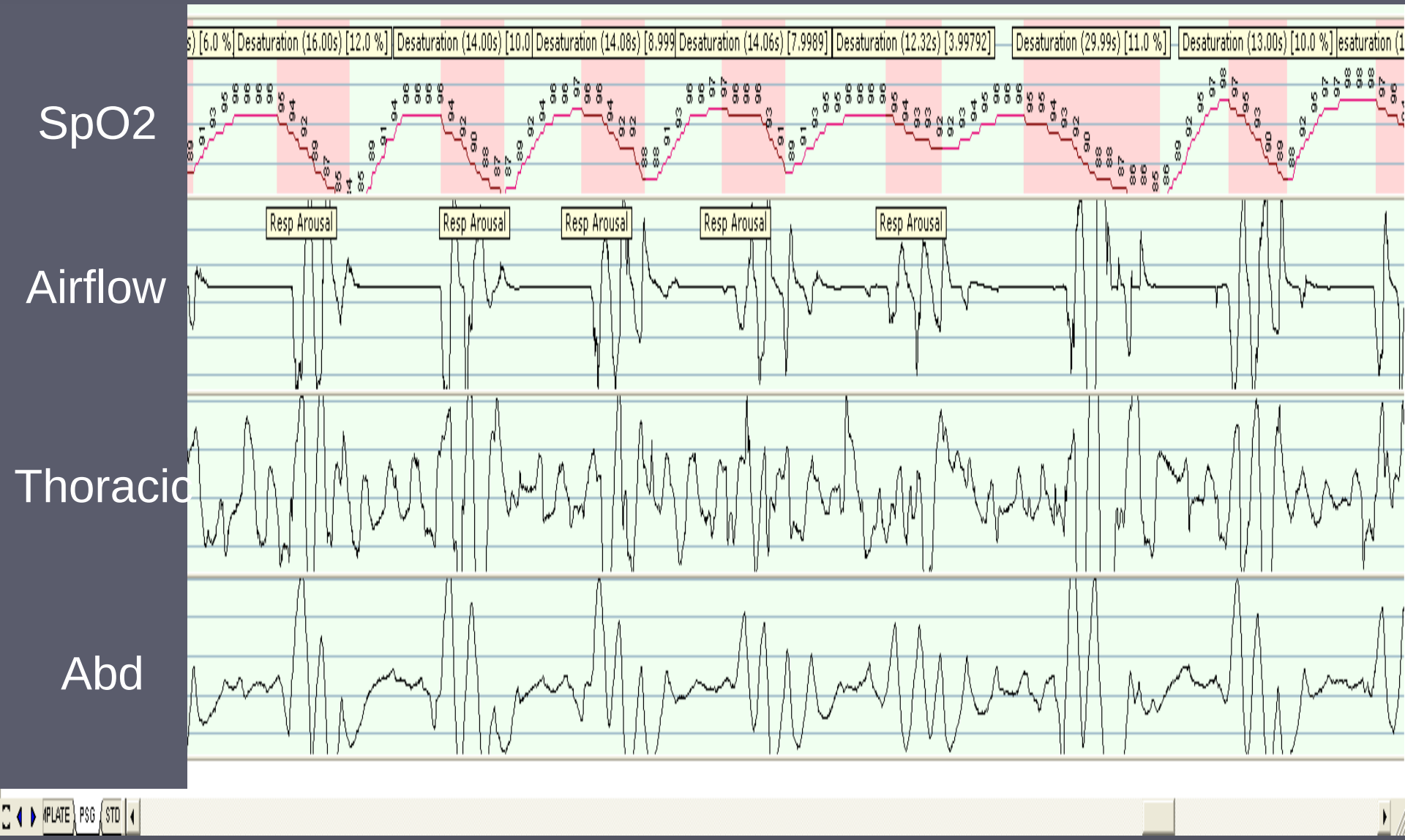


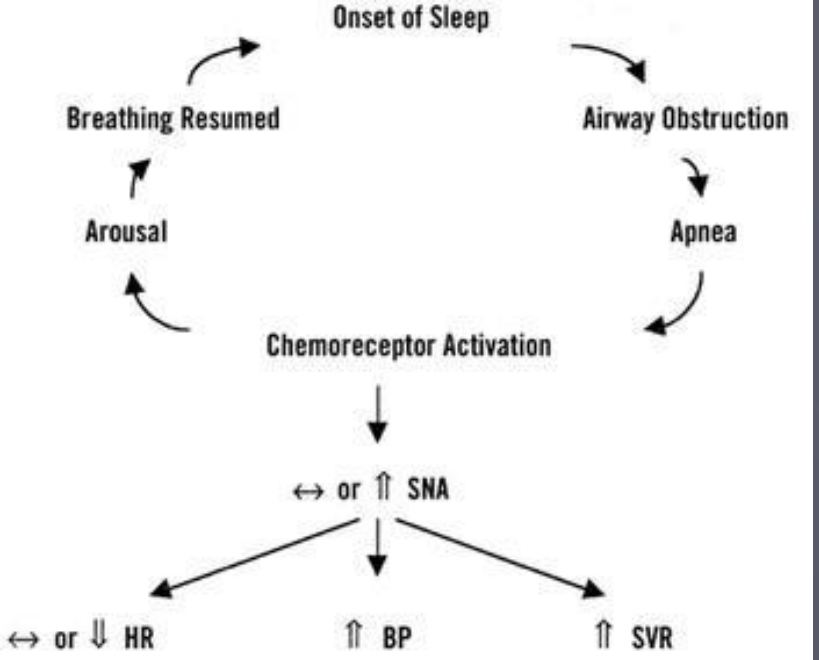
Friedman tongue position (FTP) is based on visualization of structures with the mouth opened widely without protruding the tongue.

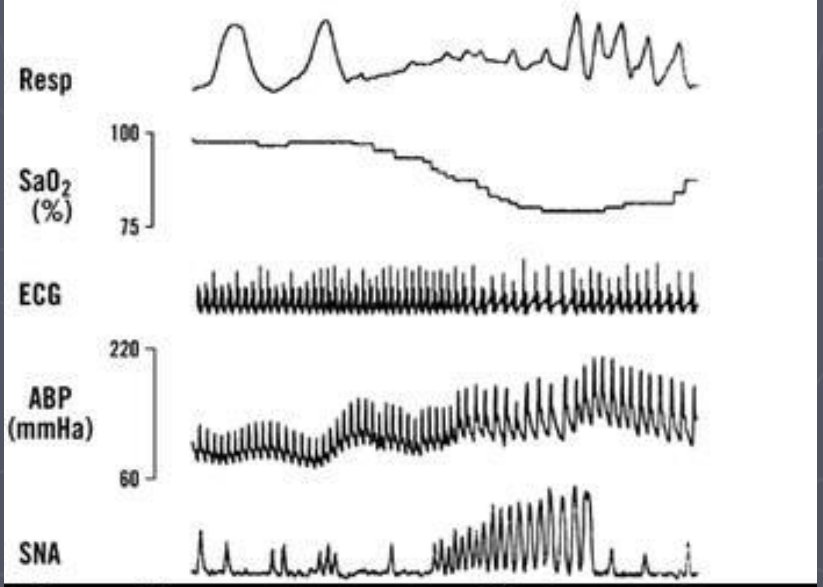


Friedman M et al. Otolaryngology -- Head and Neck Surgery 2006;134:187-196

OSA – Example of a PSG







Consequences of OSA

- Coronary artery disease
- Heart failure
- Stroke
- Sleep Heart Health Study: cross-sectional association between OSA and self-reported CVD:

	CAD	CHF	CVA
AR	1.27	2.38	1.58

Shahar et al, AJRCCM 2001

OSA related to stroke and death

- Conducted at Yale Medical Center
- 1022 participants enrolled but only 898 completed
  - 573 (68%) with OSA (AHI >5, mean AHI 35±29)
  - 325 w/o OSA (AHI<5, mean AHI 2±1.5)
- Mean age 60 yrs
- Follow up of 2-4yrs
- Adjusted for age/sex/race, smoking, alcohol intake, BMI, DM, HTN, AF, high cholesterol

Yaggi et al, NEJM 2005; 353: 2034-2041.

Results

- OSA group - 22 strokes, 50 deaths
  [3.48 events per 100 person-years]
- Control group – 2 strokes, 16 deaths
  [1.60 events per 100 person-years]
- After adjustment for age, sex, race, tobacco use, ETOH, BMI, DM, HTN, AF, HPL, OSA retained a statistically significant association with stroke or death
  [Hazard ratio 1.97; 95% CI 1.12-3.48, P=0.01]

Yaggi et al, NEJM 2005; 353: 2034-2041.

Table 3. Trend Analysis for the Relationship between Increased Severity of the Obstructive Sleep Apnea Syndrome and the Composite Outcome of Stroke or Death from Any Cause (N=1022).*

Severity of Syndrome	Stroke or Death		Mean Follow-up Period	Hazard Ratio (95% CI)
	No. of Events	No. of Patients		
			yr	
AHI ≤3 (reference score)	13	271	3.08	1.00
AHI 4–12	21	258	3.06	1.75 (0.88–3.49)
AHI 13–36	20	243	3.09	1.74 (0.87–3.51)
AHI >36	34	250	2.78	3.30 (1.74–6.26)

- Trend analysis showed a step-wise increase in the risk of stroke/death as a function of increased severity of OSA (p=0.005)
- The risk of stroke/death in pts in the most severe quartile of OSA was 3 x that in the controls

Yaggi et al, NEJM 2005; 353: 2034-2041.

Other Consequences of OSA

- Pulmonary HTN
- Cor Pulmonale
- Cardiac Arrhythmias (atrial fibrillation)
- GERD
- Increased frequency of seizures in epileptics
- Increased headache syndromes (migraines)

Consequences of OSA

- Psychiatric/mood - depression, anxiety, irritability
- Social and sexual dysfunction
- Neurocognitive impairment – general intellectual ability, learning and memory, attention, information processing efficiency, visual and psychomotor performance

Consequences of OSA

- Increased traffic accidents - case-controlled study found those with AHI > 10 had OR of 6.3 for MVA

  Teran-Santos et al, NEJM 1999

- Increased utilization of Health Care Services
- Increased mortality - relative risk 2.7-3.3
- All of these adverse outcomes can be improved by treatment

Treatment of OSA: Conservative Measures

- Weight loss
  - 10% weight loss leads to 26-50% decrease in AHI
  - pharyngeal function improves as weight decreases
  - extensive weight loss (i.e. following gastric bypass surgery) may resolve OSA
  - almost always should be combined with other therapies

Treatment of OSA: Conservative Measures

- Lateral positioning
- Elevating the head of the bed
- Avoiding upper airway irritants - tobacco
- Minimizing sedating agents - alcohol, sedatives

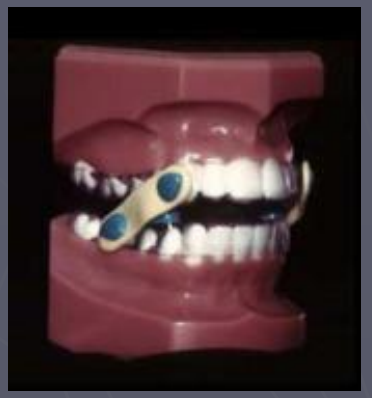
Treatment of OSA: CPAP

- First-line therapy for OSA
- Can eliminate sleep disordered breathing in most patients
- Produces a “pressurized” upper airway to maintain airway patency
- PAP titration study vs autoPAP

Treatment of OSA: CPAP

- Benefits
  - decreases sleep-disordered breathing and EDS
  - improves oxygenation, exercise function
  - improves neuropsychiatric measures
  - decreases MVAs and hospitalizations
  - appears to decrease mortality
- Problems
  - acceptance suboptimal
  - compliance poor at times but can overcome

Alternative treatment for OSA: Oral Appliances



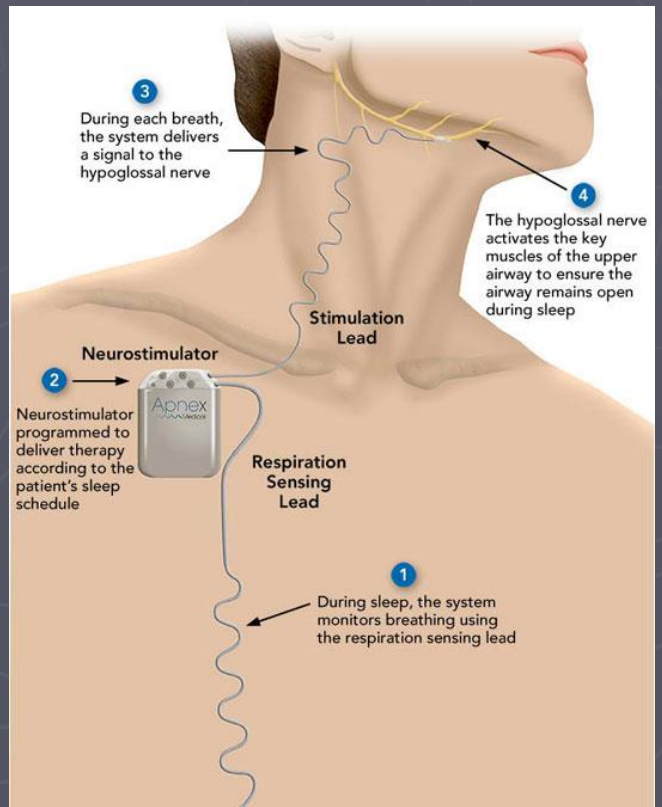
- Relatively new therapy for OSA
- Two categories:
  - Mandibular Advancing Devices
  - Tongue Retaining Devices
- Work by enlarging the pharyngeal cross-sectional area
- Consider in patients with mild/moderate OSA
- RCT suggest about equal efficacy to CPAP with better tolerance

Alternative treatment of OSA

- Provent nasal strips

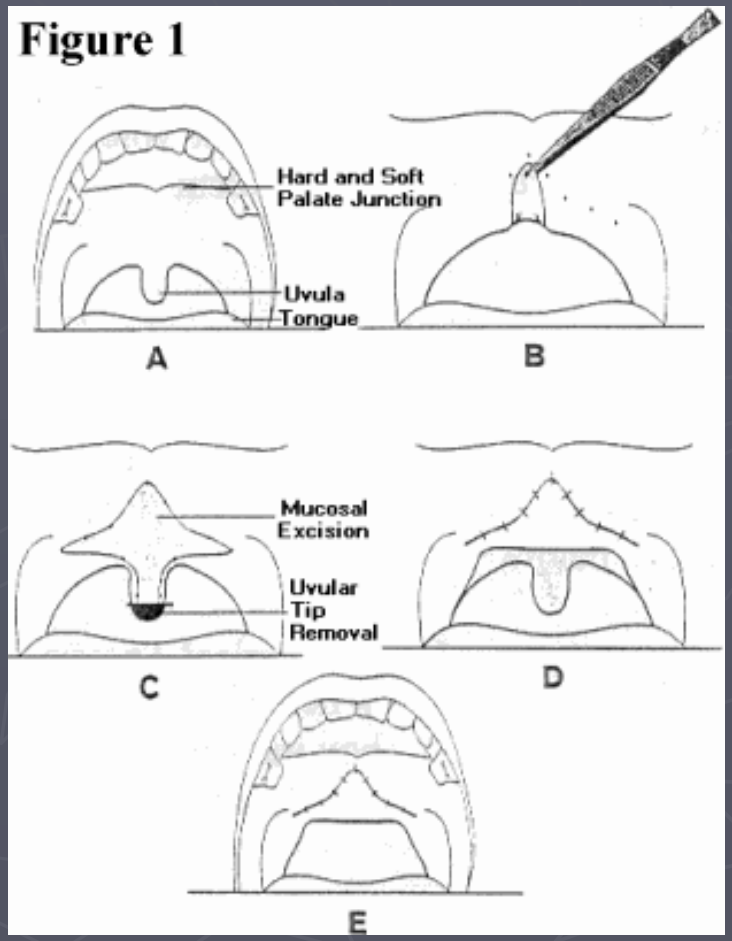
- Positional therapy

- Hypoglossal nerve stimulator



Treatment of OSA: Surgery

- Numerous approaches have been tried
- Surgical data limited
- Procedures in general use:
  - Nasal surgery
  - Tonsillectomy +/- adenoidectomy
  - UPPP
  - Genioglossus advancement
  - Maxillomandibular Advancement (MMA)
  - Tracheotomy



Treatment of OSA: Pharmacotherapy

- Little successes at this point in time
- "Some" efficacy may be present in the following situations:

Condition	Medication
OHV	Medroxyprogesterone
REM OSA	SSRIs, TCAs
CHF	Theophylline

BUMC-P Sleep

- Clinic –Advanced Lung Institute
- Sleep lab – West tower, 1st floor
- We see everything – insomnia, OSA, CSA, narcolepsy, RBD, nocturnal epilepsy, etc
- Office number: 602-351-3400
- 2 physicians, RRT, RPSGT (5)
  - Najma Usmani, MD (sleep pulmonologist)
  - Stacy Thacker, RRT
- Cyrus Guevarra (neurodiagnostics sr mgr)
- Email me: joyce.lee-iannotti@bannerhealth.com


Banner
University Medical Center
Phoenix

Summary

- Sleep Medicine is a relatively new field
- Normal Sleep is dictated by homeostatic pressure and circadian rhythms
- PSG is the gold standard for diagnosis of most sleep disorders
- Insomnia is the most common sleep disorder, but OSA is the most common cause of EDS

- Sleep is cool, dude~
- We welcome rotators all the time ☺
- Sleep Medicine Fellowship debuting 2019

QUESTIONS??


GLASBERGEN
7-5
"It was the most dynamic presentation I ever gave. One person stayed awake for almost seven minutes!"