

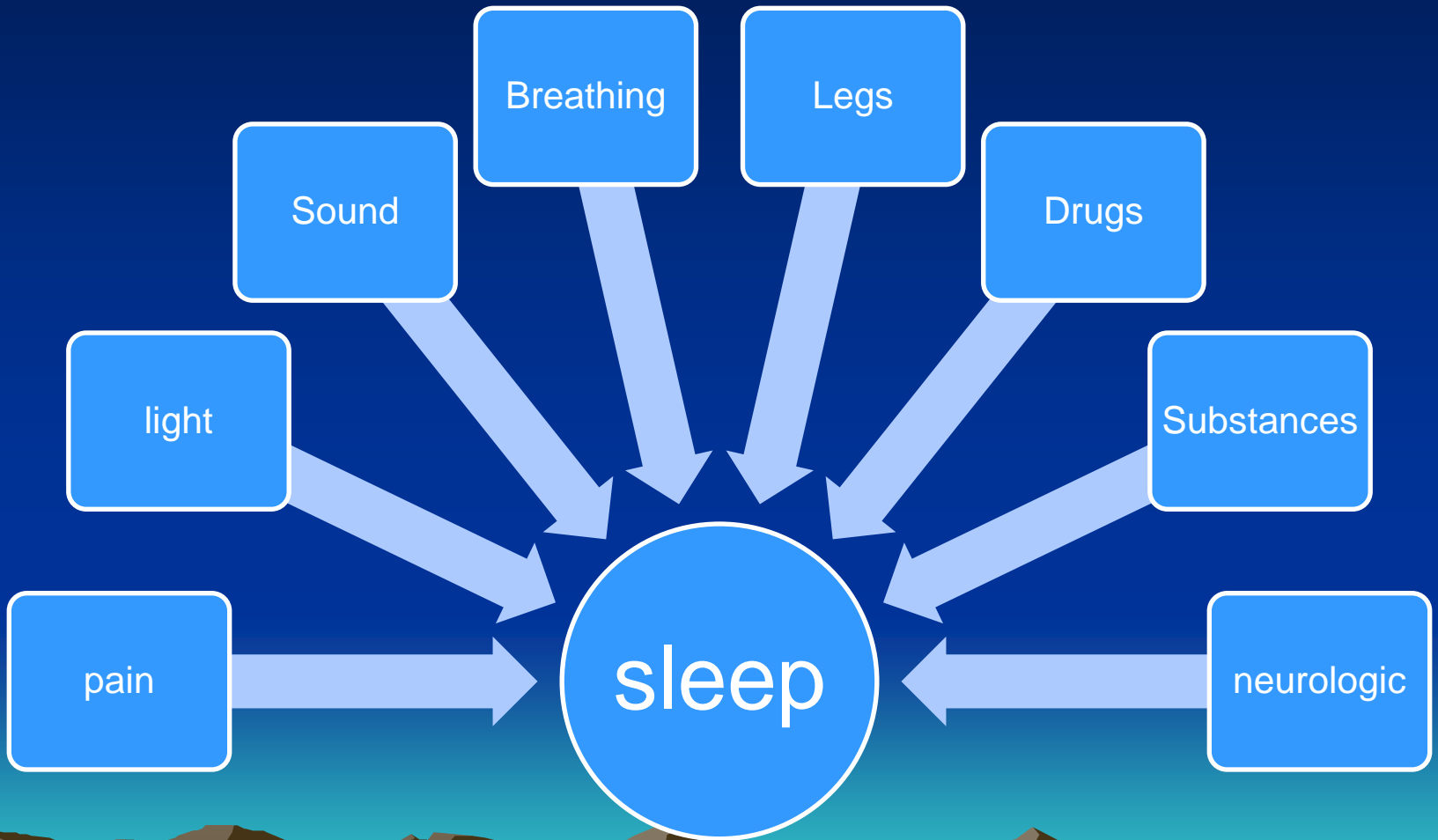
Pain and sleep

June 2011

Raymond Gottschalk



Reversible unconsciousness



Sleep impact on pain

- Sleep deprivation increases IL-6
- IL-6 sensitizes nociceptors
- Descending pathway is opioid and serotonergic in brainstem
- DNIC- diffuse noxious inhibitory control is upper brainstem function reducing pain perception by opioid activation
- DNIC dysfunctional in fibromyalgia



Chronic pain

- 70% report poor sleep
- Pain fragments sleep
- 1960's studies showed disturbed sleep homeostasis
- EEG instability was seen as increase in alpha sleep pattern



Linear and circular relationship

- Acute pain- sleep complaints resolve with pain resolution
- But this can transform into a vicious pain cycle where intense pain causes sleep disturbance and causes pain enhancement
- Linear relationship in 53-89% with chronic pain



Pain and sleep reciprocal cycle

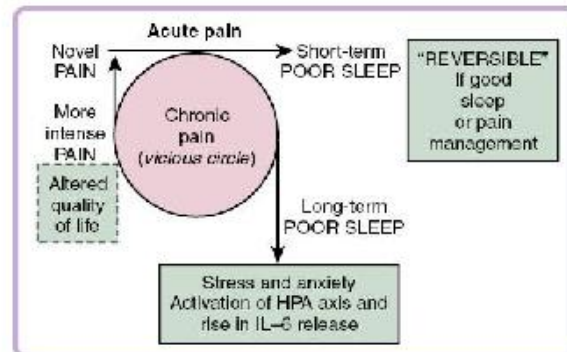


Figure 126-1 Linear (acute pain) and vicious-cycle (chronic pain) relationships. HPA, hypothalamic-pituitary-adrenal; IL-6, Interleukin-6.

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Pain activation of the brain

- Pain is protective response integrating limbic structures, hypothalamus, amygdala, cingulate and frontal cortex



Somatic pain

- 1. Activation of nerve endings
- 2. Transmission - sensory A delta and C
- 3. Spinal relay (1st)
- 4. Thalamic relay (2nd)
- 5. Cortical neuronal activation
- 6. Final reaction to pain



Visceral pain

- Diffuse pain and poorly localized
- Visceral nociceptors are less discriminating
- Tissue sensory ending and brain localization are oversolicited – allodynia
- Activation of descending influences may allow better tolerance



Epidemiology and Risk factors

- Comorbidities are important
- Sleep disruption → daytime sleepiness
- Sleep duration and pain is U shaped
- 6-9 hours sleep seems best and worse function beyond those times and health risks increase



Prevalance

- 11-29% have pain
- 50-70% will have poor sleep
- 44% have insomnia complaints
- Concomitant mood disturbance (depression and anxiety)



Sleep features of chronic pain

- Sleep fragmentation
- Lower percentage SWS (deep sleep)
- Daytime fatigue and sleepiness
- Cognitive impairment



Neurobiological basis

- Spinoreticular and trigeminothalamic tracts behave as gated control mechanisms
- To arouse or not to arouse that is the question?
- Different stages are more vulnerable to arousal with REM and SWS most resistant
- No clear circadian distribution of pain except stiffness more in am



Cognitive impairment

- Memory and attention ↓ in chronic pain
- Sleep deprivation will enhance pain appreciation
- Any stage reduction is hyperalgesic



Neurochemistry and pain

- ACTH and cortisol as well as IL-6 and TNF α increase with chronic pain
- Not sure if due to sleep reduction vs pain influence



Diagnosis

- Review sleep pattern
- Sleep diary
- Mood profile
- Alcohol and medication and drug use
- Associated sleep disorders
- Pain scales VAS
- Sleep study



Sleep study findings

- Primary common disorders
- OSA
- PLMS
- Parasomnia
- Epilepsy
- Insomnia
- Hypoxemia



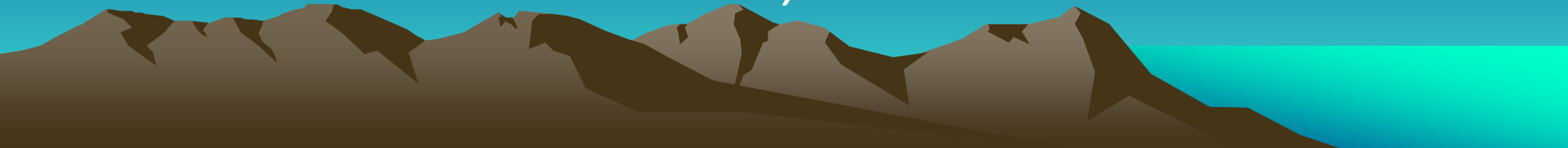
Measures in sleep

- Sleep efficiency
- Sleep stages
- Stage shifts
- Sleep fragmentation
- Alpha sleep
- Autonomic changes and RR interval



Treatment options

- Medications and impact on sleep
- Carry over effects in day
- Differential metabolism of medications
- Incorrect assumptions from the PSG
- Opioids impact on sleep (↓ SWS and REM and breathing)
- SSRI effects on sleep (↓ REM and sleep fracture is increased)







All \$3.75

Scoops

Ice Cream

Bucconer's Beauty
 Vanilla ice cream topped with chocolate sauce and drizzled with Hot Fudge or Hot Chocolate Sauce, and hot or a dash of salted caramel sauce. (Sauce is optional.)

Death by Chocolate
 A mouth-buckling trio: Chocolate Luscious Knight, Chocolate Ice Cream, Chocolate Fudge Brownie, Chocolate Fudge Sauce and Chocolate Sauce. Oh yes, you know you want it!

Strawberry Landslide
 Strawberry & Vanilla ice cream topped with Raspberry and topped with a generous dollop of Raspberry Sauce & Whipped Cream.

Blackboard's Drunken Sundae
 Show us your love. This is a little something for the grown-ups!
 Original Rum Cake and Cream. Vanilla ice cream topped together with Two Caribbean Rum Fudge.

Bananamama
 Vanilla Ice Cream, Chocolate, Big Caramel, Fudge Sauce and lots of whipped cream. (Sauce is optional.)

Dietary Beverage
 Whipping the Cream. Start with the whole way: Vanilla Ice Cream, Hot Fudge Sauce, Chocolate Sauce and Three Creams. (Sauce is optional.)

2015
 ALL DAY
 ALL DAY BAR
 OPEN
 FROM 11:00 AM



Hot Drinks
Iceda Float
Cola Float
Sprite Float
Power Float
1.75

Your creation to life.





08
CAUTION
DECK SLIPPERY
WHEN WET



08
CAUTION
DECK SLIPPERY
WHEN WET



CAUTION
DECK SLIPPERY
WHEN WET







E4











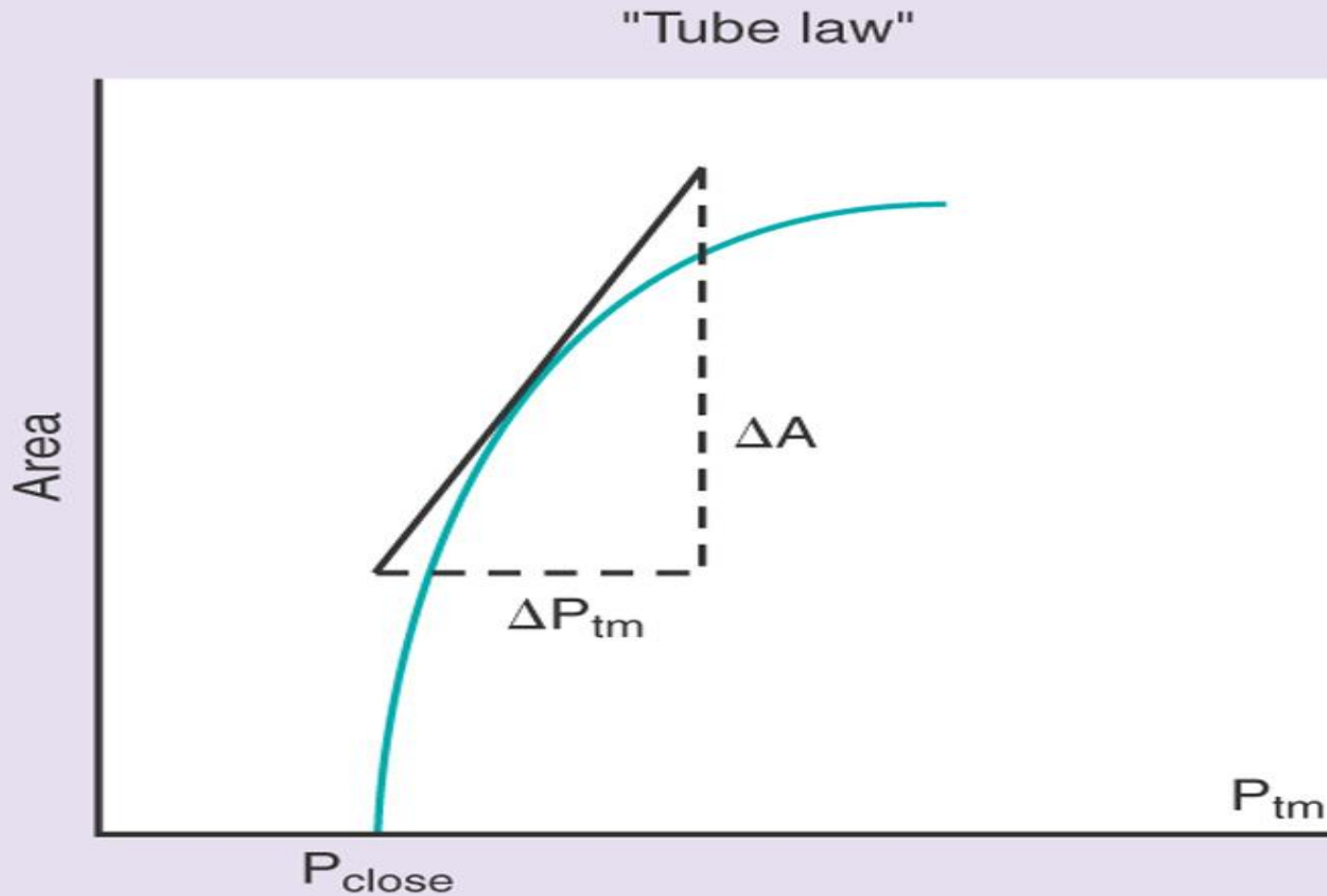




Sleep medicine pearls

- **NHANES 2003-4**
 - Overweight >25
 - Obese >30
 - Morbid >40
- | | men | women |
|----------------|-----|-------|
| Overweight >25 | 71% | 62% |
| Obese >30 | 31% | 33% |
| Morbid >40 | 3% | 7% |
-
- Airway closure in those with sleep apnea
 - normal -4.4cm/H₂O
 - Mild +0.6
 - Moderate +2.2

Tube Law

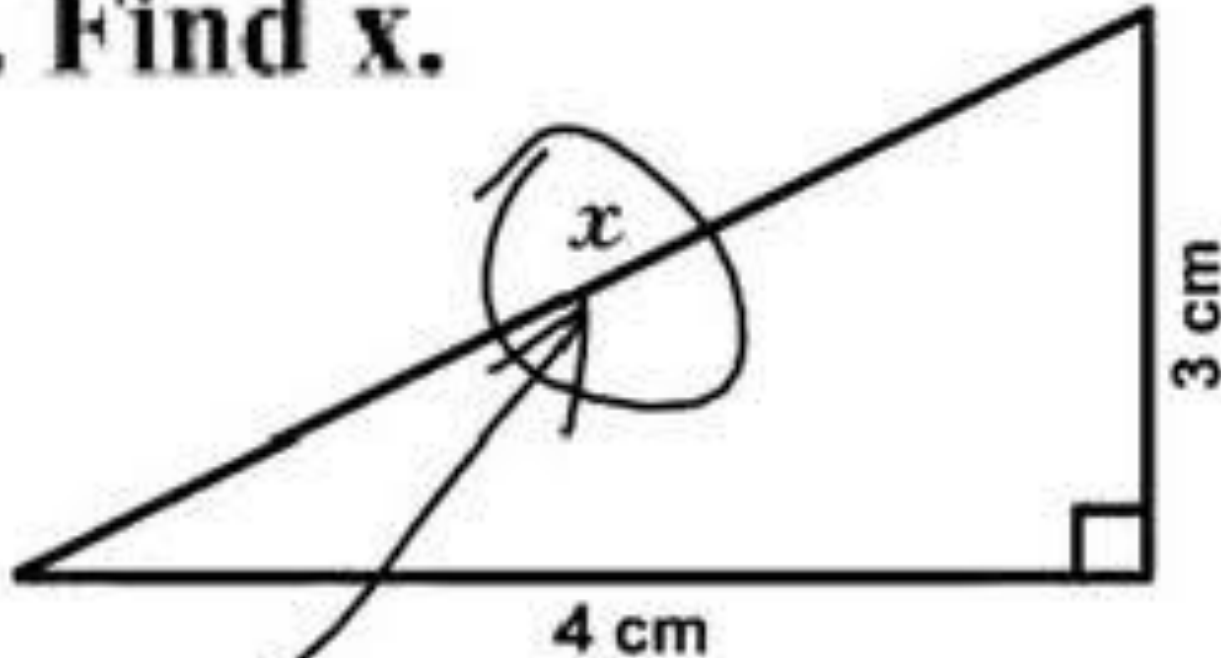


$$\text{Compliance} = \frac{dA}{dP_{tm}}$$

B

Tube Law

3. Find x .



Here it is

So what is apnea

- Reduction in flow for 10 seconds to less than 10% on a thermistor
- No desaturation
- No arousal needed



What is hypopnea (preferred)

- 30 % reduction in flow with nasal pressure
- 4% desaturation
- (preferred)



What is hypopnea (research)

- 50% reduction in flow with pressure
- 3% desaturation OR arousal
- (so do not need any desaturation)



What is RERA

- Depends:
- If you are using one definition of hypopnea then all other events are RERA
- It can be arousal only (no desaturation)
- It can be desaturation only (no arousal)



What is more important?

- Desaturation?
- Arousal?
- Desaturation with arousal?



Impact of arousal

- UARS is the prototype



UARS vs OSA

AHI <5 per hour

SaO₂ > 92



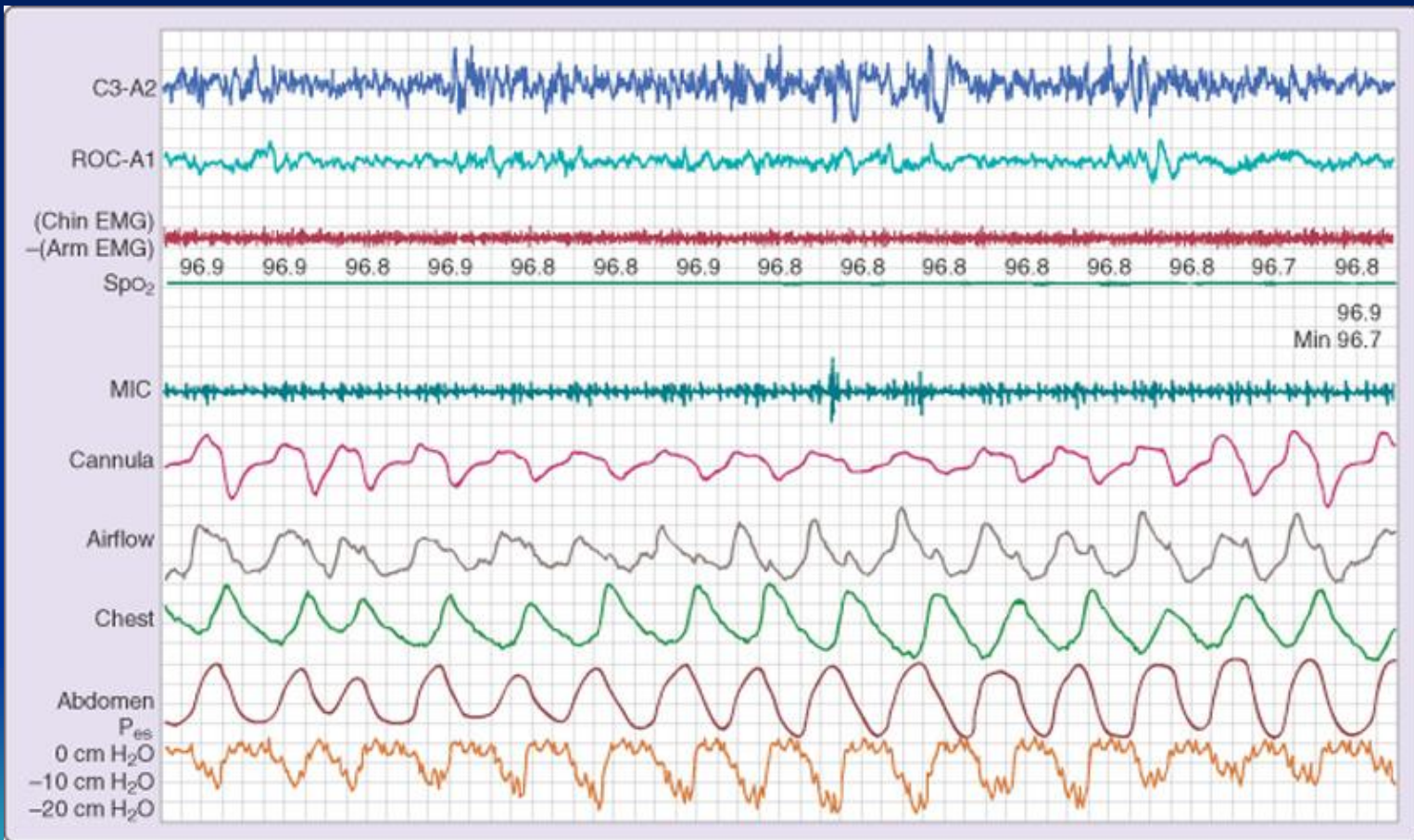
OSA vs UARS

- OSA
- EDS and fatigue
- Forgetful
- Personality change
- Headache
-

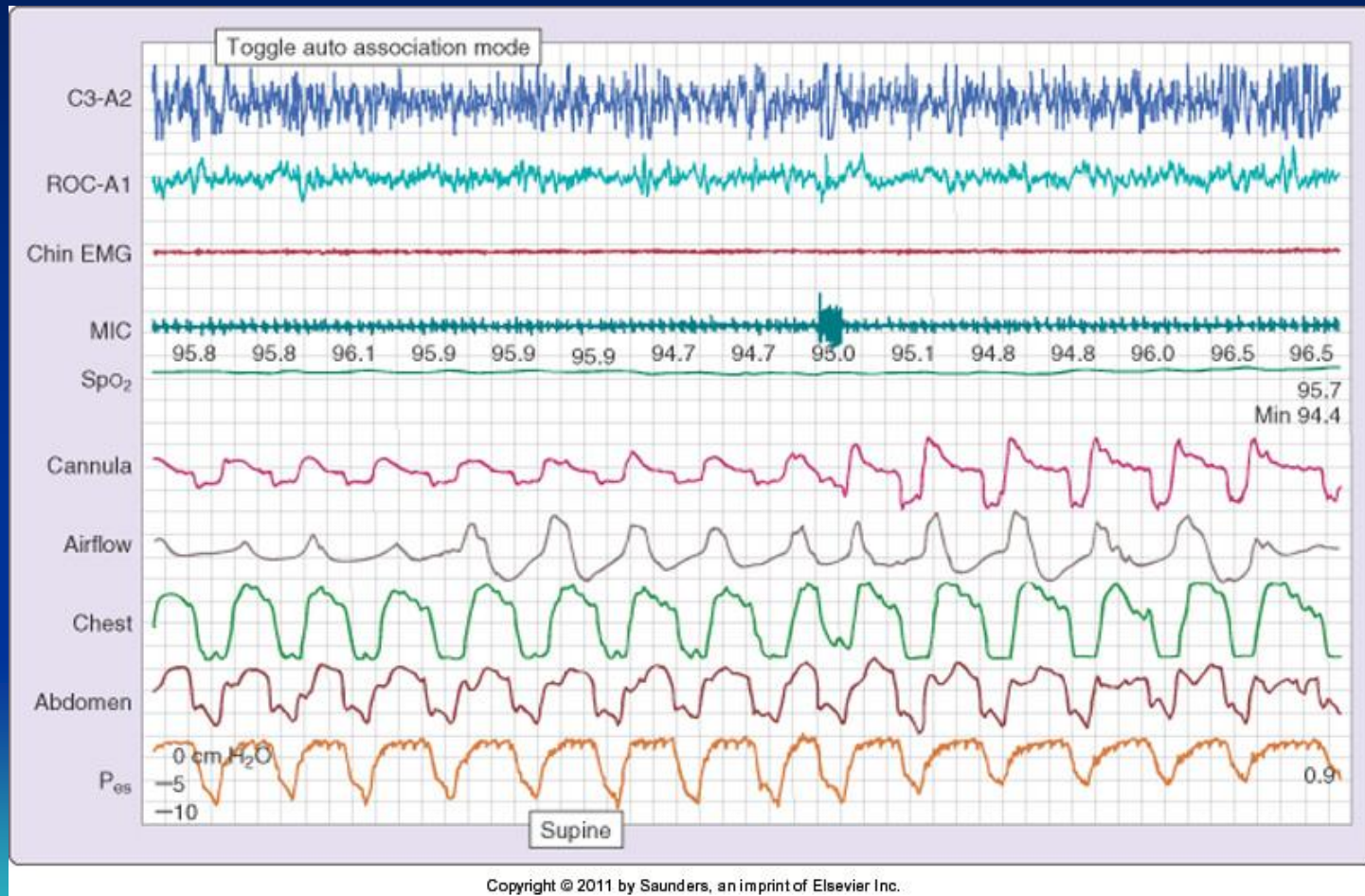
UARS
insomnia
fatigue
fibromyalgia
migraine
hypotension
dizziness



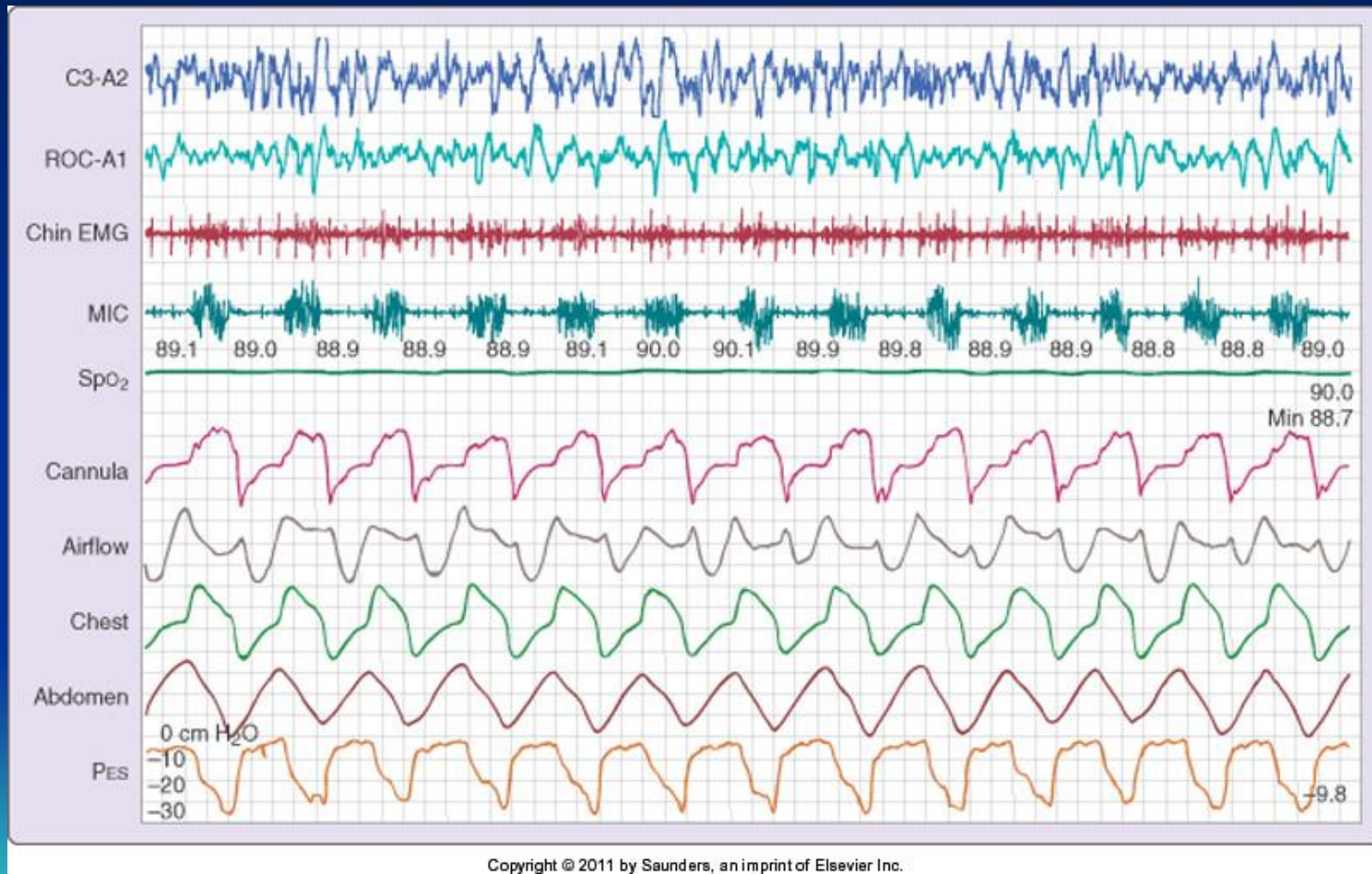
Progressive obstruction



Continuous obstruction no arousal



Continuous sustained effort



Marin 2005: Lancet:

Long-term cardiovascular outcomes in men with obstructive sleep apnoea-hypopnoea with or without treatment with continuous positive airway pressure: an observational study

Lancet 2005; 365: 1046-53 *Jose M Marin, Santiago J Carrizo, Eugenio Vicente, Alvar G N Agustí*



Marin outcomes

	Healthy men (n=264)	Simple snorers (n=377)	Untreated mild- moderate OSAH (n=403)	Untreated severe OSAH (n=235)	OSAH treated with CPAP (n=372)
Non-fatal cardiovascular events					
Number of events	12	22	36	50	24
Events per 100 person years	0.45	0.58	0.89	2.13*	0.64
Cardiovascular death					
Number of events	8	13	22	25	13
Events per 100 person years	0.3	0.34	0.55	1.06†	0.35

OSAH=obstructive sleep apnoea-hypopnoea syndrome; CPAP=continuous positive airway pressure. *p<0.0001 versus healthy men; †p=0.0012.

Table 2: Incidence of cardiovascular events during the 10-year follow-up in healthy men, snorers, and patients untreated and treated for OSAH

Buchner AJRCCM mild to

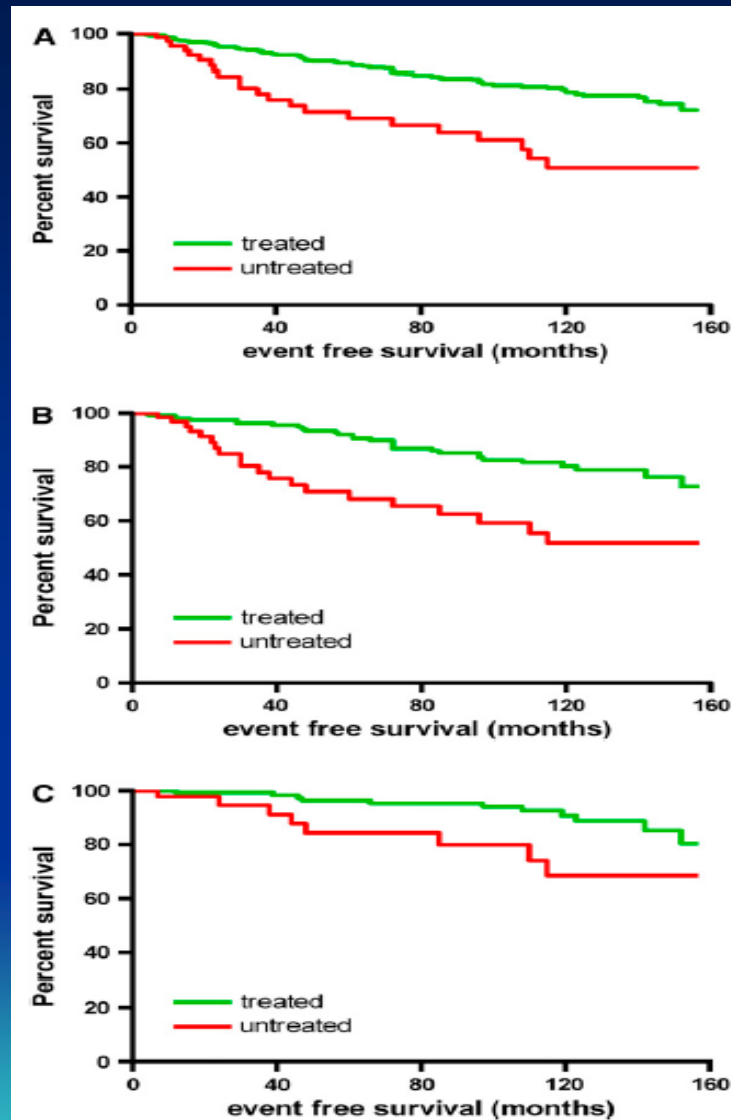


Figure 2. Kaplan-Meier estimates of the probability of event-free survival in (A) treated versus untreated patients with obstructive sleep apnea (OSA), (B) treated versus untreated patients with mild to moderate OSA, and (C) treated versus untreated patients with mild to moderate OSA without preexisting cardiovascular disease.

Marin survival curves

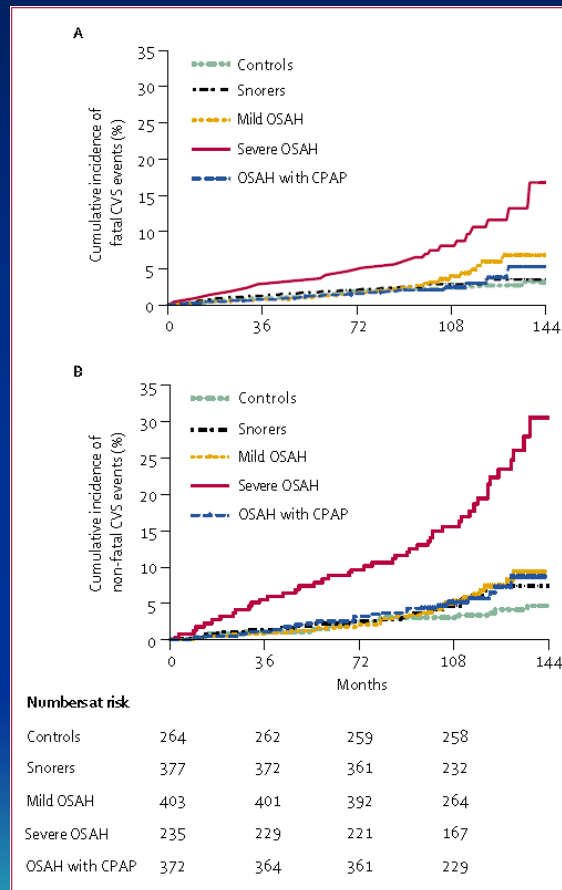
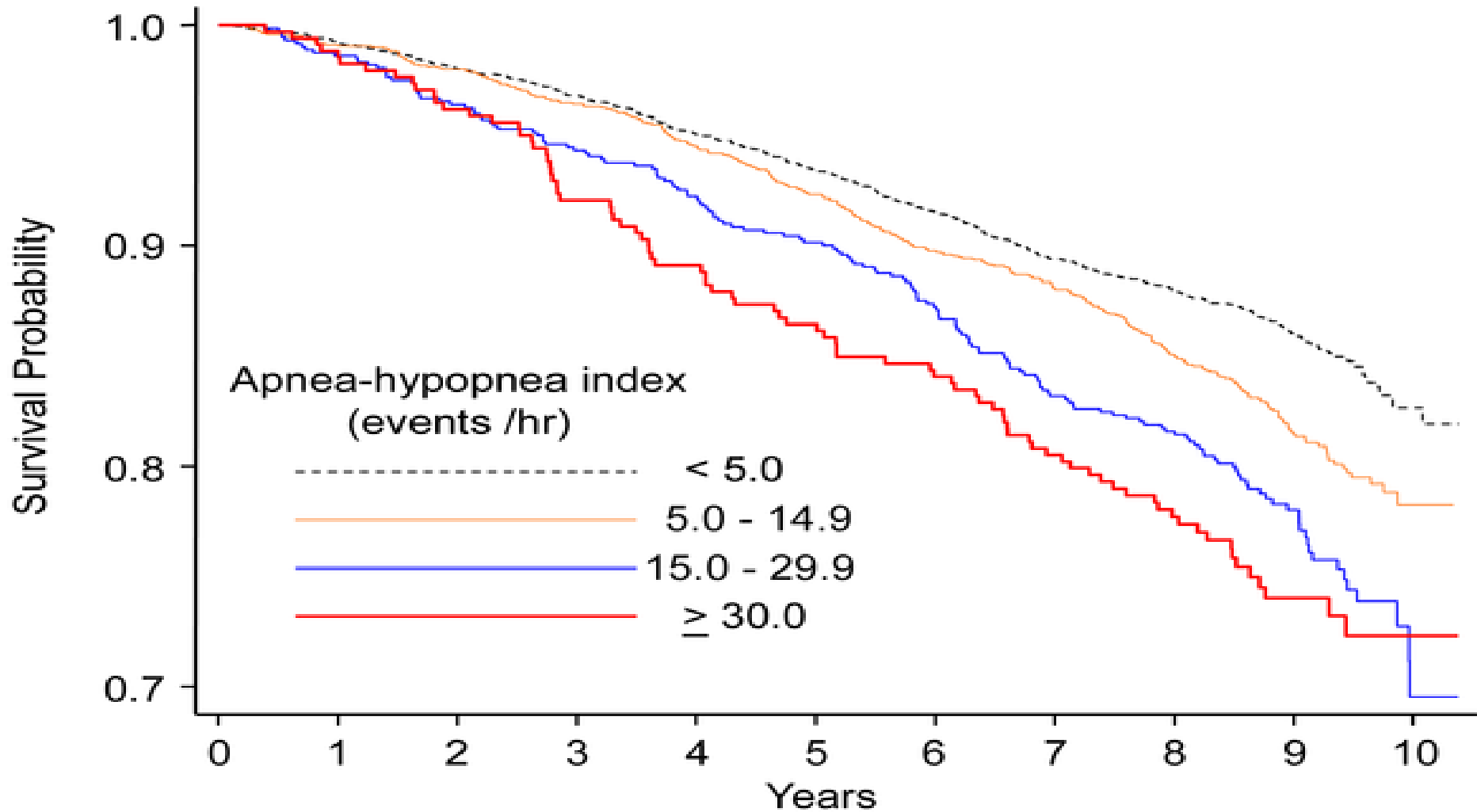


Figure 2: Cumulative percentage of individuals with new fatal (A) and non-fatal (B) cardiovascular events in each of the five groups studied

SDB and cardiovascular disease

- SHHS 6106 adults
- Odds ratio for arousal and mortality – no association
- Odds ratio for CV disease is the following with ODI of 4% desaturation
 - 1 for < 1.01 events per hour
 - 1.10 (1.01-3.20)
 - 1.33 (3.21 -7.69)
 - 1.41 (>7.7 per hour)

Mortality in SHHS



At risk:	6294	6205	6110	6001	5868	5732	5566	5411	4756	2357	300
Deaths:	0	59	143	241	359	478	616	757	875	989	1046

Summary for our review


- Oxygen desaturation is important
- $TsT_{90} < 2.7\%$ vs $> 2.7\%$
- hazard ratio 1.83 (1.3-2.7)
- Only measured apnea and hypopneas as 4% desaturation and called RDI



SHHS conclusions

- In conclusion, the Sleep Heart Health Study shows that sleep-disordered breathing is an independent predictor of mortality and that this association is not attributable to age, obesity, or other chronic medical conditions. Although the degree of nocturnal hypoxemia was an independent predictor of mortality, **arousal frequency and occurrence of central apneas were not**. Given the high and likely increasing prevalence of sleep-disordered breathing in the general population, additional research in the form of randomized clinical trials should be undertaken to assess if treatment can reduce premature mortality associated with this common and chronic disorder

Conclusions

- Large differences in AHI when varying definitions are used to score hypopneas
 - Difference in classifying SDB
 - Differences in arousal play a large role
 - Use of flow $\geq 30\%$ or $\geq 50\%$ did not alter the AHI when combined with $\geq 4\%$ desaturation
 - Adopt a single standardized hypopnea definition
- 

Hypoxemia and LDL

- **Conclusions: Nocturnal hypoxia was associated with increased oxidized LDL/total LDL in both unadjusted and adjusted analyses even** after taking into account obesity. In contrast, the AHI was not significantly associated with oxidized LDL/total LDL after adjusting for confounders. These data suggest that the degree of hypoxic burden rather than the frequency of respiratory disturbances is a risk factor for increased lipid peroxidation, and suggests the opportunity to assess the impact of improving overnight oxygenation as an intervention to mitigate the cardiovascular consequences of SDB.



Heavy Snoring as a Cause of Carotid Artery Atherosclerosis

- Sharon A. Lee^{1,2}; Terence C. Amis, PhD^{1,2,4}; Karen Byth, PhD^{2,4}; George Larcos, MBBS^{3,4}; Kristina Kairaitis, PhD^{1,2,4}; Tracey D. Robinson, PhD^{1,2}; John R. Wheatley, PhD^{1,2,4}
- *1Ludwig Engel Centre for Respiratory Research, 2Westmead Millennium Institute, 3Department of Nuclear Medicine and Ultrasound, Westmead Hospital and 4University of Sydney at Westmead Hospital, Westmead, NSW, Australia*
- ***SLEEP 2008;31(9):1207-1213.***



Snoring and stupidity

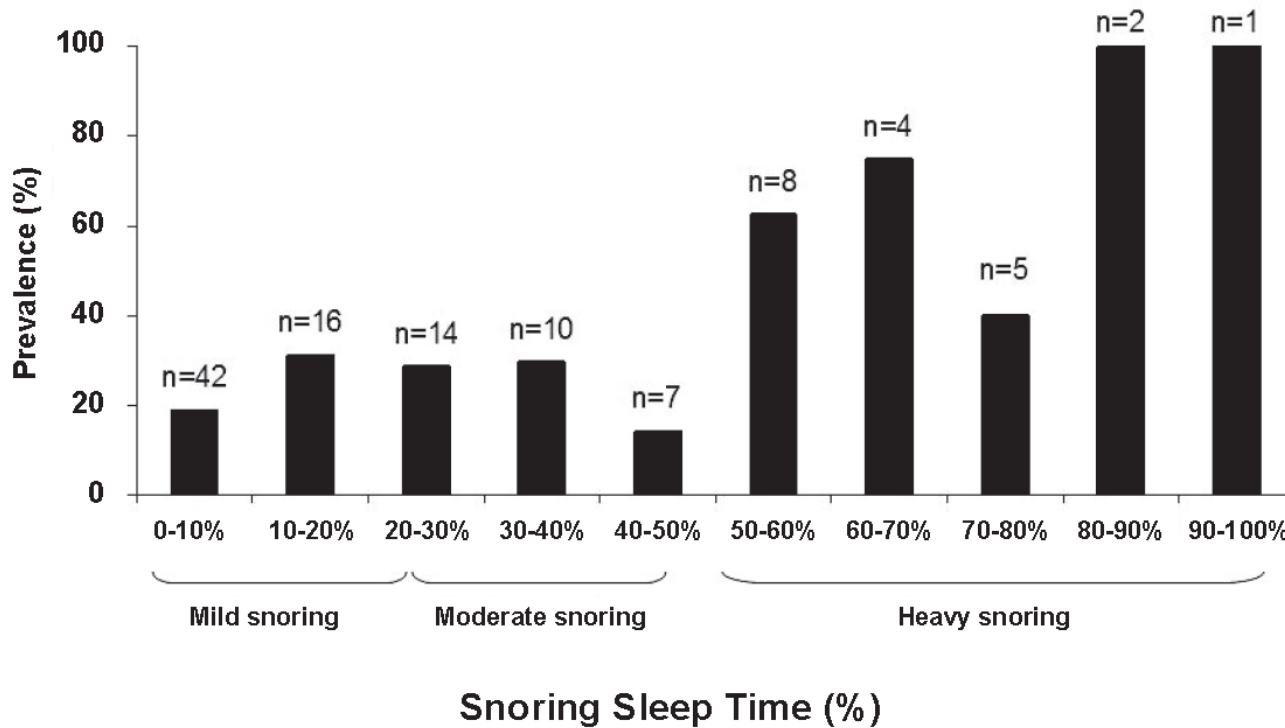


Figure 1— Carotid atherosclerosis prevalence stratified by snoring sleep time. Frequency histogram for the prevalence of carotid atherosclerosis grouped by percentiles of snoring sleep time. Note the substantially increased prevalence of carotid atherosclerosis for snoring sleep time longer than 50%. Snoring group classification based on snoring sleep time is shown on the x axis. Number of subjects falling within each snoring sleep time decile is shown on each column.

Snoring and the carotids

Table 1—Baseline Study Population Characteristics^a

	Whole group	Snoring status		
		Mild	Moderate	Heavy
Age, y	58.2 (± 7.9)	59.5 (±8.2)	56.6 (±4.9)	56.2 (±9.5)
Sex				
Men	59	29	15	15
Women	51	36	10	5
BMI, kg/m ²	27.5 (± 4.7)	25.7 (±3.2)	28.7(±4.6)	31.8 (±5.5)
Neck circumference, cm	38.2 (± 4.0)	36.5 (±3.2)	39.7(±3.5)	41.8 (±3.8)
Smoking history, %	27	20	40	35
Hypertension, %	27.3	18.5	36	45
Hyperlipidemic, %	69	61.5	76	85
Index, events/h				
AHI	10.9 (5.8 - 20.5)	8.4 (4.3 - 14.6)	12.7 (5.7 - 19.9)	20.7 (15.4 - 41.6)
AI	18.4 (13.3 - 25.4)	17.7 (13.4 - 23.2)	19.5 (14.5-25.0)	20.8 (12.8- 32.6)
ODI 3%	1.5 (0.4 - 3.7)	0.9 (0.3 - 2.3)	2.0 (0.5 - 3.4)	6.9 (2.9 - 11.5)
Hypoxic sleep time, % TST	0 (0 -0.7)	0 (0 - 0.5)	0 (0 - 1.0)	0.8 (0.1 - 8.1)

^aData are provided as mean (± SD) or median (interquartile range) except, sex, which is shown as number, and smoking history, hypertension status, and hyperlipidemia status, which are shown as a percentage of the group. BMI refers to body mass index, AHI, apnea-hypopnea index, ODI, oxygen desaturation index, AI, arousal index, TST, total sleep time.

More snoring more plaque

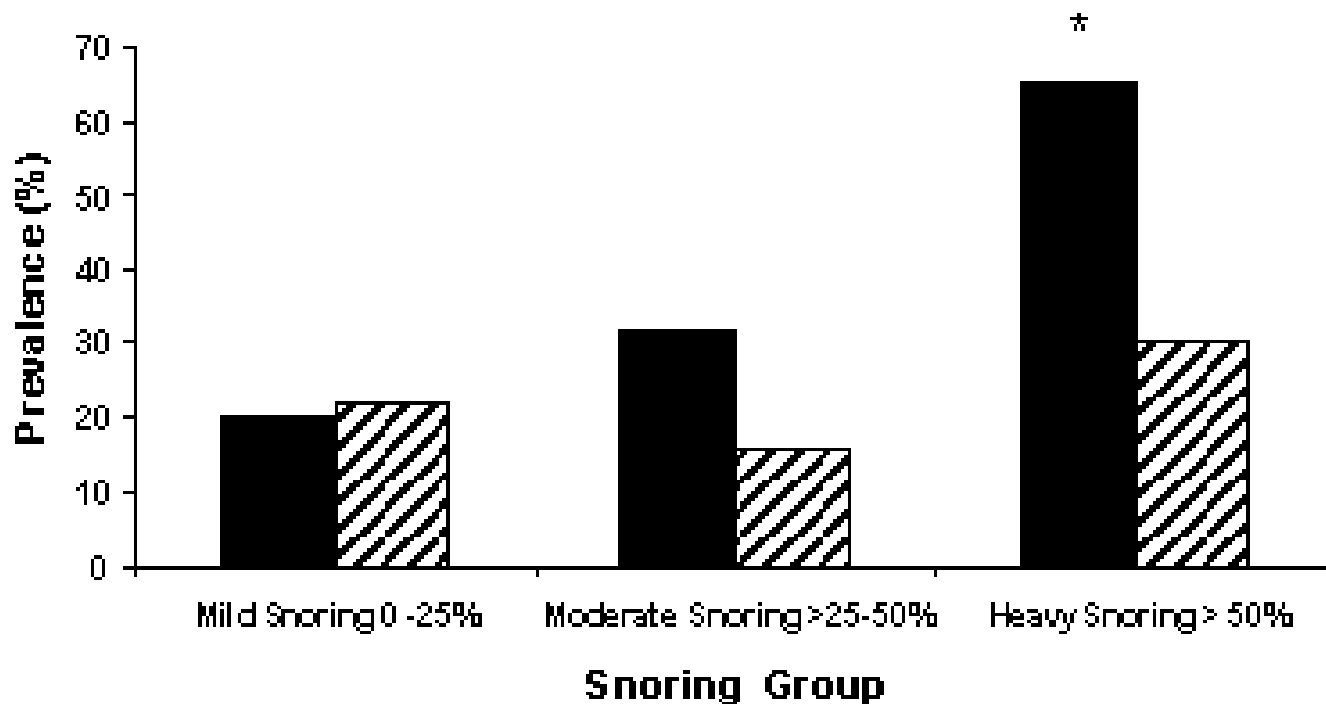


Figure 2—Prevalence of carotid and femoral atherosclerosis. Prevalence of carotid atherosclerosis (black columns) and femoral atherosclerosis (shaded columns) by snoring group. Note that the prevalence of carotid atherosclerosis increased progressively across the 3 groups [$*P < 0.04$ (χ^2)]. However, the prevalence of femoral atherosclerosis did not differ between groups.

Brain blood flow and hypoxemia

- Pizza F; Biallas M; Wolf M; Werth E; Bassetti CL. Nocturnal cerebral hemodynamics in snorers and in patients with obstructive sleep apnea: a near-infrared spectroscopy study.
- *SLEEP* 2010;33(2):205-210.



Brain blood flow detection

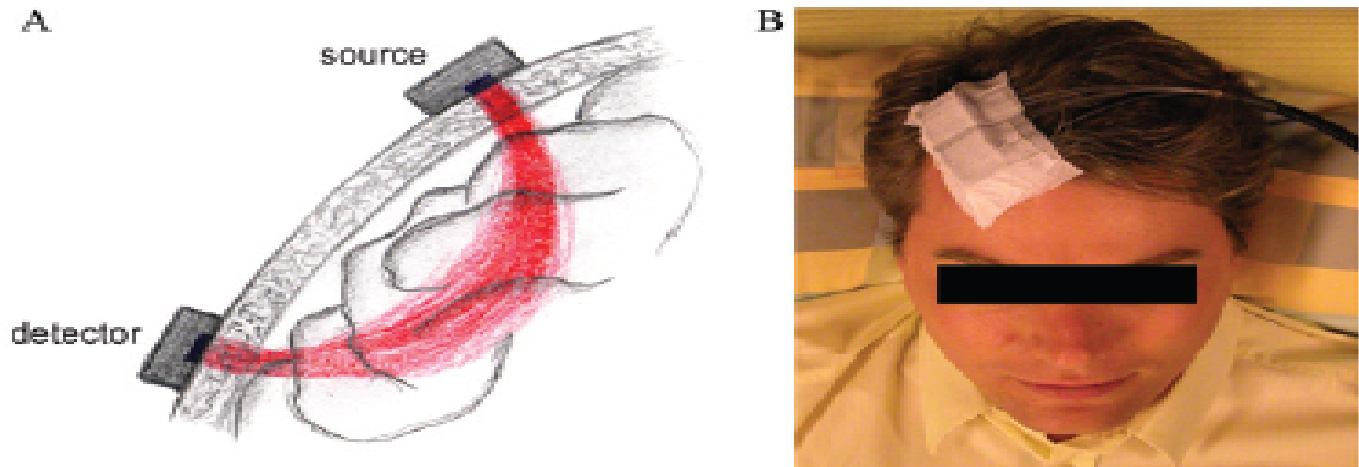


Figure 1—Light path of near-infrared (NIRS) light from the light source to the detector through the tissue (A) and positioning of the NIRS optode on the right forehead (B).

Figure 1A shows the light path of NIRS light from a single light source through superficial layers (skin and skull) to the cerebral tissue and back to the light detector. The multidistance approach uses different light sources at specified distances from a common light detector to allow the subtraction of the influence of superficial tissue from NIRS recording. The light path is bent and reaches a maximum depth of about 2 cm beneath the center of the multidistance sensor, defining the region of sensitivity of NIRS that originates at the light source and extends to the detector.

Figure 1B shows an example of the position of the NIRS optode (i.e., a sensor with 4 light sources and 1 detector) on the right forehead. The NIRS optode is fixed to the head by a medical adhesive, before being shielded from external light with a cotton bend.

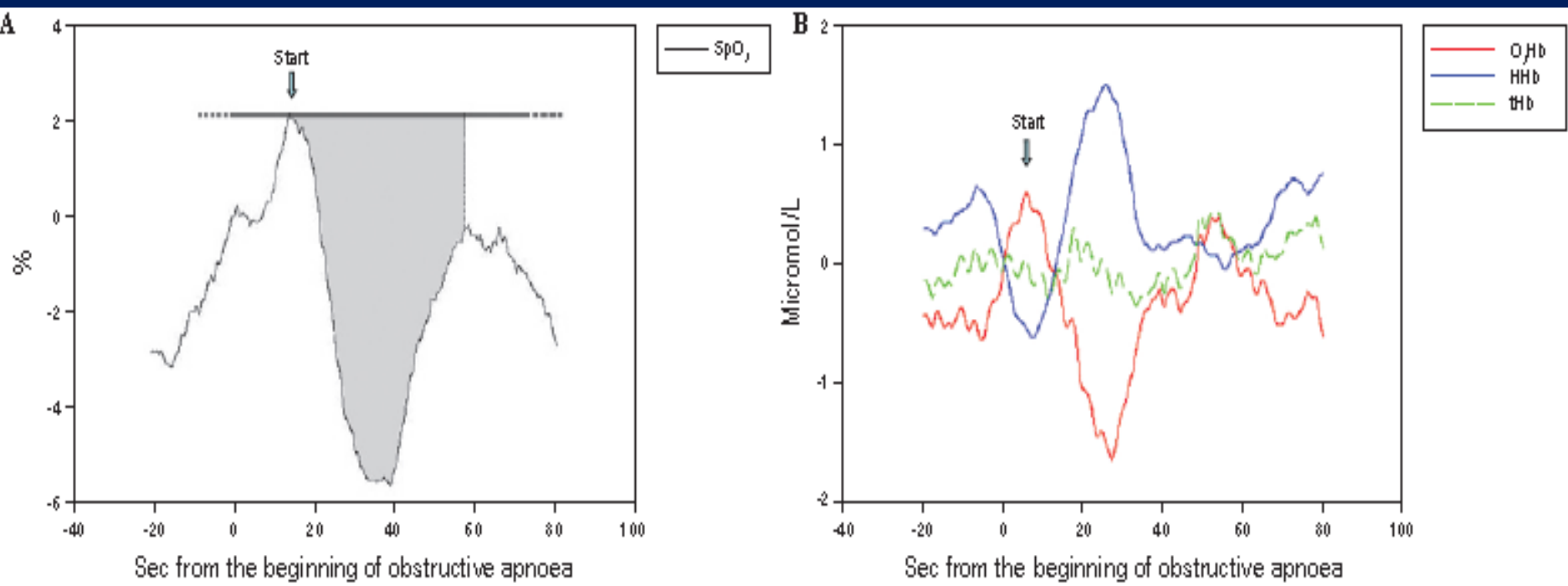


Figure 2—Averaged signals of (A) peripheral oxygen saturation (SpO₂), and (B) oxygenated (O₂Hb), deoxygenated (HHb), and total hemoglobin (tHb) concentrations during obstructive apneas in non-rapid eye movement (NREM) sleep of a patient with severe obstructive sleep apnea syndrome (OSAS). *Figure 2A* shows the average of the SpO₂ signal occurring during obstructive apnea in NREM sleep in a patient with severe OSAS. The arrow marks the start of the respiratory event; the grey color depicts the area of the integral calculation between the maxima SpO₂ levels (respiratory-event duration). *Figure 2B* shows the averages of O₂Hb, HHb, and tHb concentration signals during obstructive apnea in NREM sleep of a patient with severe OSAS. The arrow marks the start of the integral calculation that is computed across the respiratory-event duration. After 5 seconds from the beginning of the obstructive apnea, the O₂Hb decreases and the HHb increases abruptly (which corresponds with a severe tissue oxygen desaturation), whereas the tHb shows a concomitant transient increase. After 50 seconds from the beginning of the event, the O₂Hb, HHb, and tHb concentrations are back to baseline values.

Hypoxia does matter

Our results suggest a complex threshold mechanism (inclusive of quality and frequency of breathing alterations during sleep) in the determination of cerebral hemodynamics during SDB. During hypopneas, the brain efficiently prevents severe hypoxia. We speculate that the cerebral hemodynamics associated with hypopnea could be a cortical activation pattern evoked by the incoming progressive cerebral hypoxia (i.e., an increase of tHb and O₂Hb mirrored by a decrease of HHb) analogous to those changes found in functional NIRS studies.²⁶⁻²⁸ A failure of cerebral compensatory mechanisms appears only in patients with severe obstructive SDB. We speculate that the recurrence of obstructive apneas across the night above a specific frequency (in terms of AHI) could exhaust the cerebrovascular reserve.

Hypoxia does matter

Sleep Measures and Morning Plasma TNF- α Levels in Children with Sleep-Disordered Breathing

David Gozal, MD^{1,2}; Laura D. Serpero, PhD²; Leila Kheirandish-Gozal, MD^{1,2}; Oscar Sans Capdevila, MD^{2,3}; Abdelnaby Khalyfa, PhD^{1,2}; Riva Tauman, MD^{2,4}



TNF and mean latencies

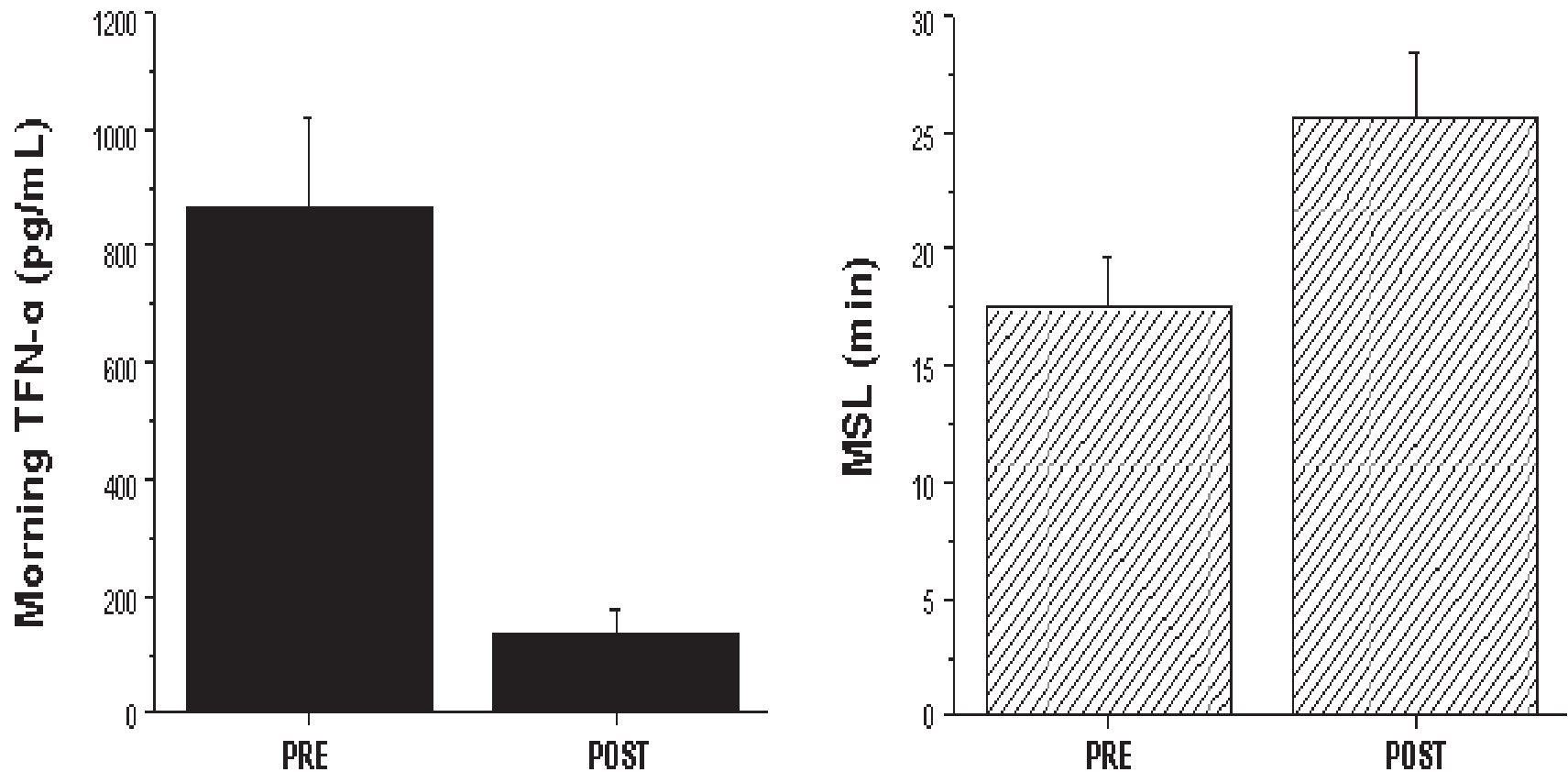


Figure 3—Tumor necrosis factor (TNF)- α morning plasma levels and mean sleep latencies (MSL) in 22 children with obstructive sleep apnea before (PRE) and after (POST) surgical adenotonsillectomy. (PRE vs POST for both TNF- α levels and MSL: $P < 0.001$)

MSLT and TNF levels

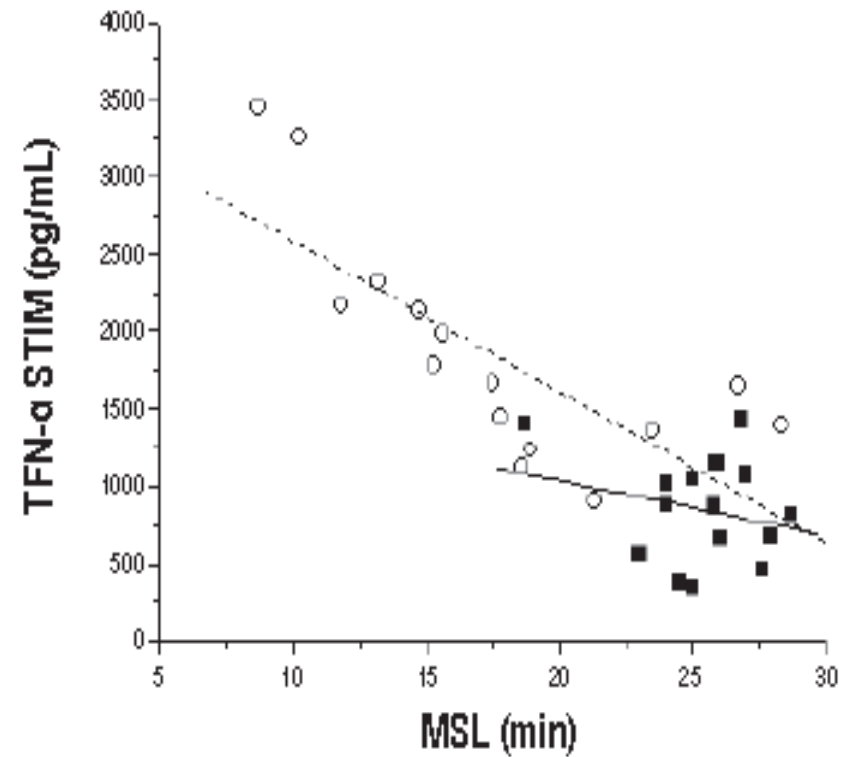
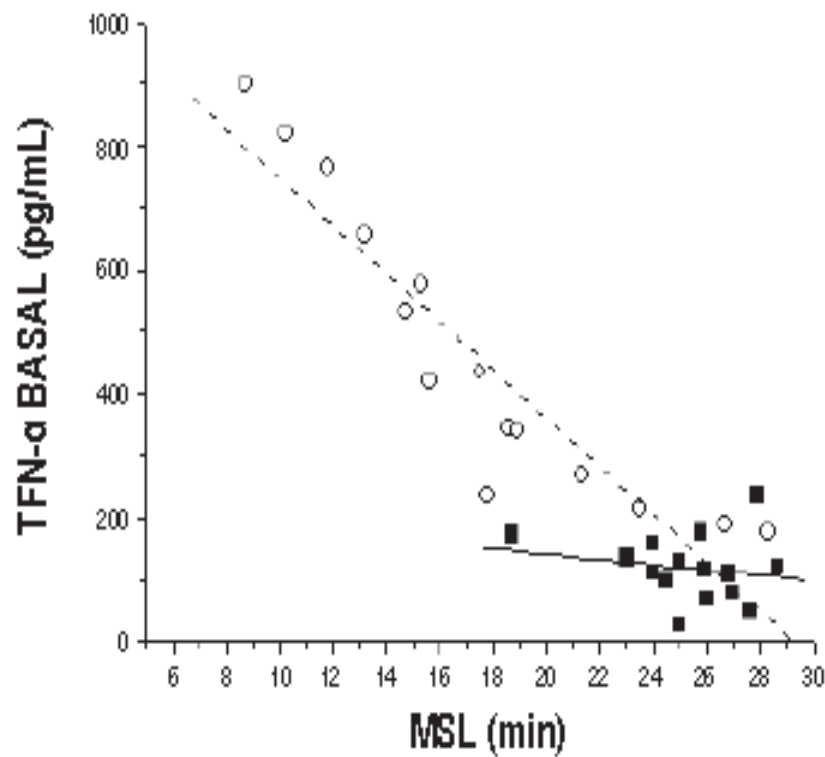


Figure 2—Scattergrams of individual basal and after lipopolysaccharide stimulation tumor necrosis factor (TNF)- α supernatant levels obtained by ex vivo blood cultures and plotted against corresponding mean sleep latencies (MSL) in 15 children with obstructive sleep apnea and 15 age-, sex-, ethnicity-, and body mass index-matched control subjects.

TNF and AHI

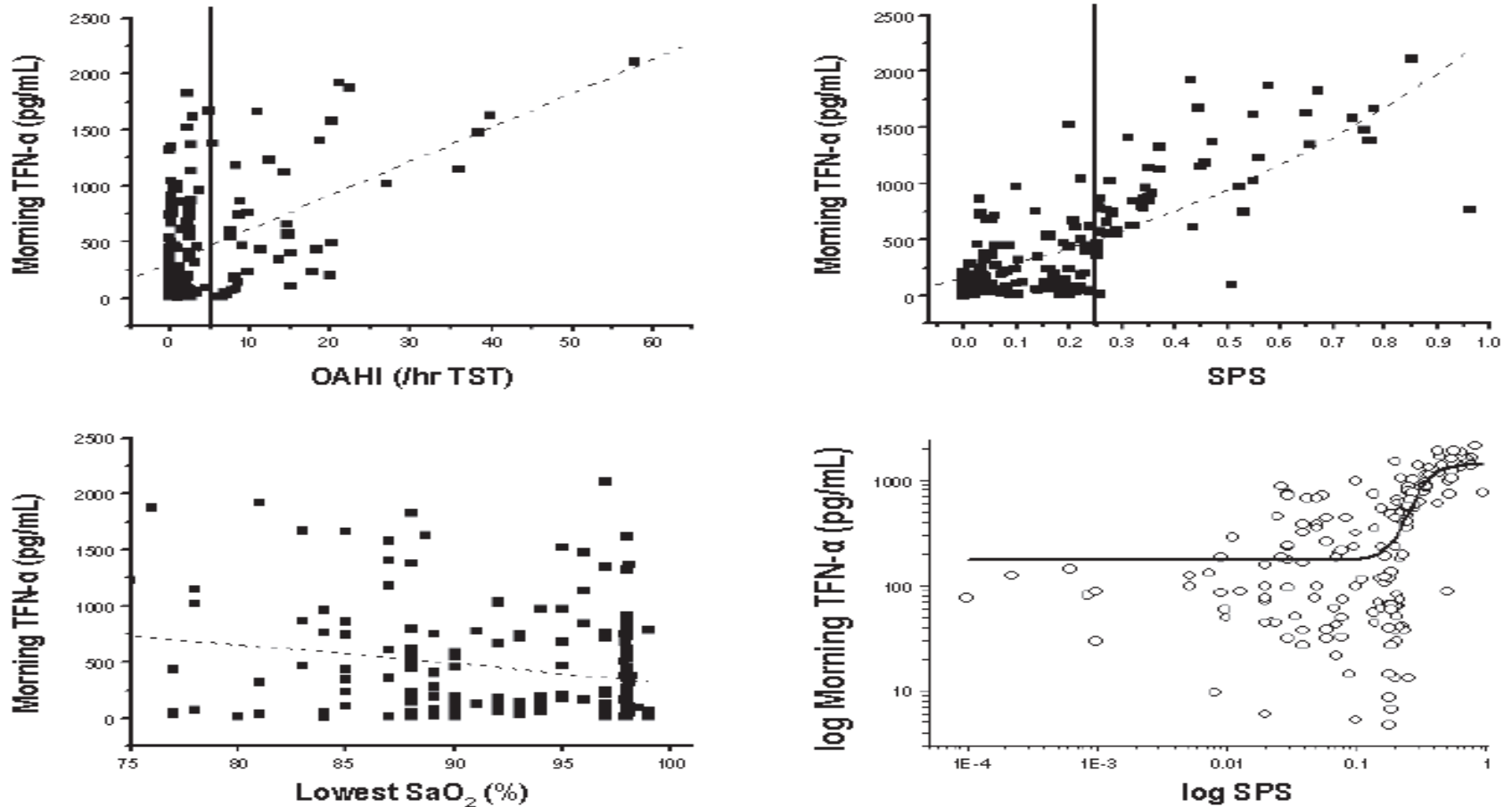


Figure 1—Scattergrams of individual tumor necrosis factor (TNF)- α morning plasma levels plotted against corresponding obstructive apnea-hypopnea index (OAH), nadir SaO₂, and sleep pressure score (SPS) in 270 habitually snoring children and 28 nonsnoring control subjects. Linear regression lines are shown and were highly significant for OAH (r^2 : 0.24; P < 0.001) and SPS (r^2 : 0.52; P < 0.000001) but not with nadir SaO₂ (r^2 : 0.05; p > 0.05). The vertical lines show OAH = 5 and SPS = 0.25 as cutoffs for disease severity. The right bottom panel shows log TNF- α plotted against log SPS, with the vertical line representing SPS = 0.25 whereas the curvilinear line represents the sigmoidal fit function (r^2 : 0.64; P < 0.00000001). Please note the marked take off of TNF- α morning plasma concentrations once SPS exceeds the 0.25 cutoff value (7).

3 Vascular disease with mild OSA

- AHI >5
- 163 pts and 22 snorers with B mode US
- Intima/Media thickness
- 0.686mm vs. 0.581mm
- P=0.004
- Increase P selectin, leptin, IL6, CRP all increase with hypoxemia
- Lin et al: 0660: A215:Sleep volume 32, Abstract supplement, 2009.

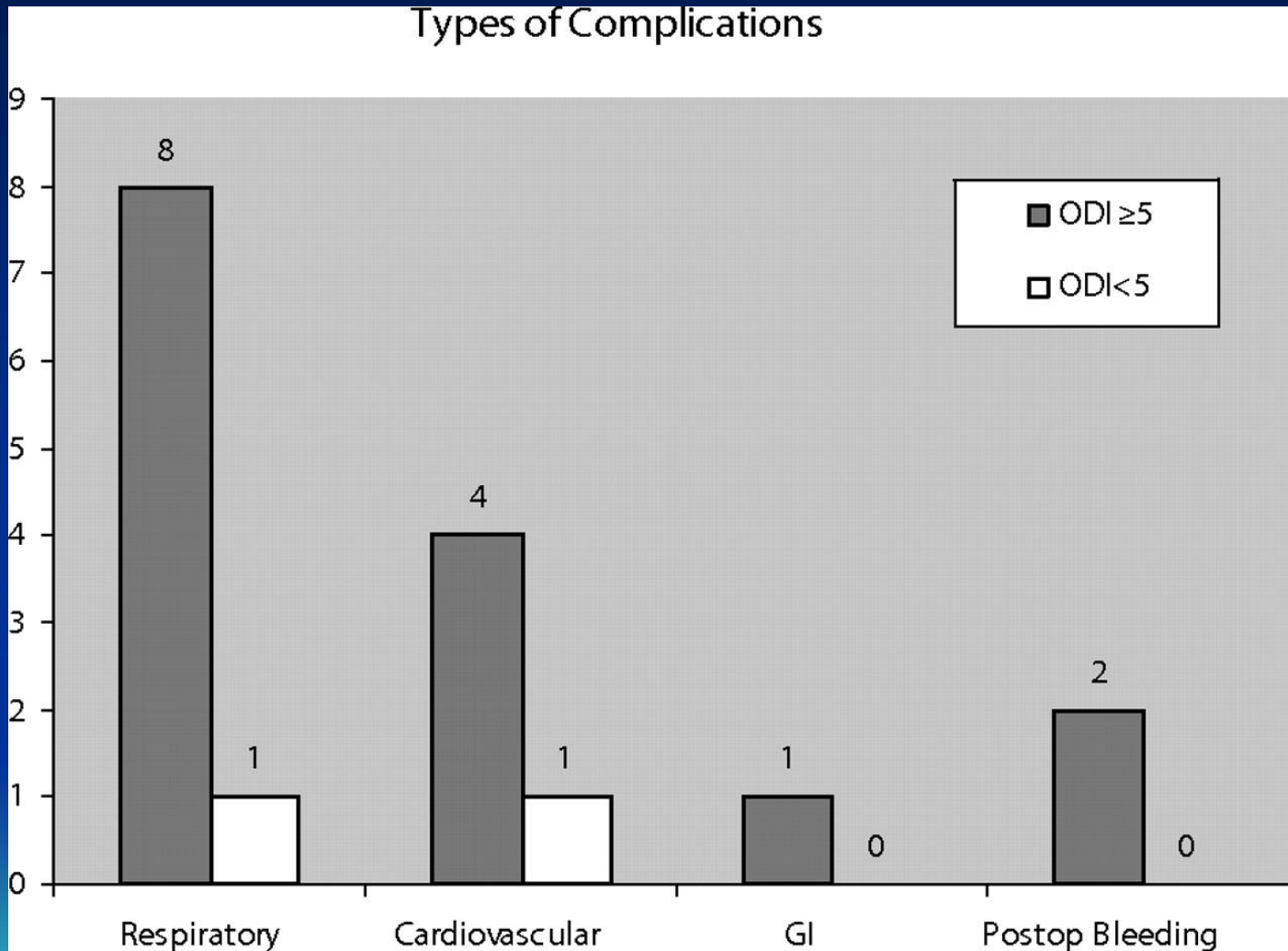


4 ODI severity

- ODI4% or T90(% sleep below 90%)
- Mild 5-20 mod 20-40 severe >40
- <5 - 2.7% complication rate
- 5-15 - 13.8% complication rate
- >15 - 17.5% complication rate
- 12% occurred in the ODI<5
- 88% occurred in ODI>5



Tabulation of the types of complications based on ODI grouping



Hwang D. et al. Chest 2008;133:1128-1134

Sleep medicine pearls

1. SHHS with AI of 1-10 had ▲cv disease
2. AI of >5 predicts MI (OR =23.3.)
3. OSAS has 4.5 increase in A fib
4. OSA reduces vasopressor response
5. OSA increases opioid ventilatory suppression



Sleep medicine pearls

- Cricomental distance >1.5 cm no OSA
Tsui
- Mandible to hyoid >2.0 cm predicts OSA
- P selectin CD 62P adhesion molecule increases in hypoxemia
- IL6, Leptin and CRP increase in apnea
- Platelet reactivity relates to ODI and not AHI



Case 1

- 56 yr female with daytime headache
- Breathing slowly
- venous angioma brainstem bleed between pons and medulla in 1993
- Normal exam Normal PFT BMI 22.4
- Tegretol 1200: Neurontin 1000: Fentanyl 50: Statex 15: Eltroxin 50: Andriol 40
- Diagnosis?



Summary case 1

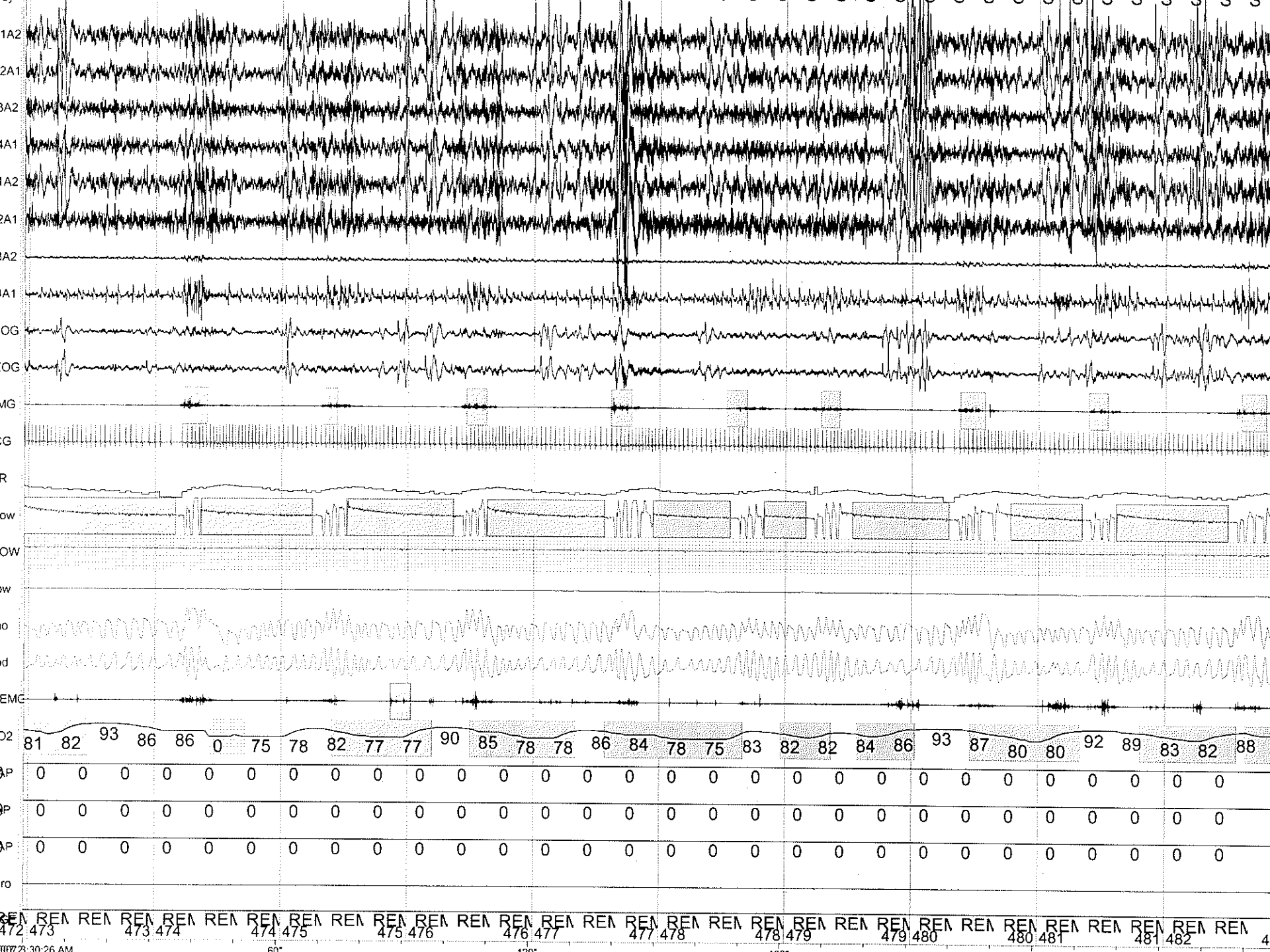
- Neurogenic hypoventilation
- Cannot breathe from brainstem injury
- Needs augmented support from ventilator or Bipap
- Cannot comfortably increase breathing rate
- Now on Bipap at 1 1/4 and rate of 8

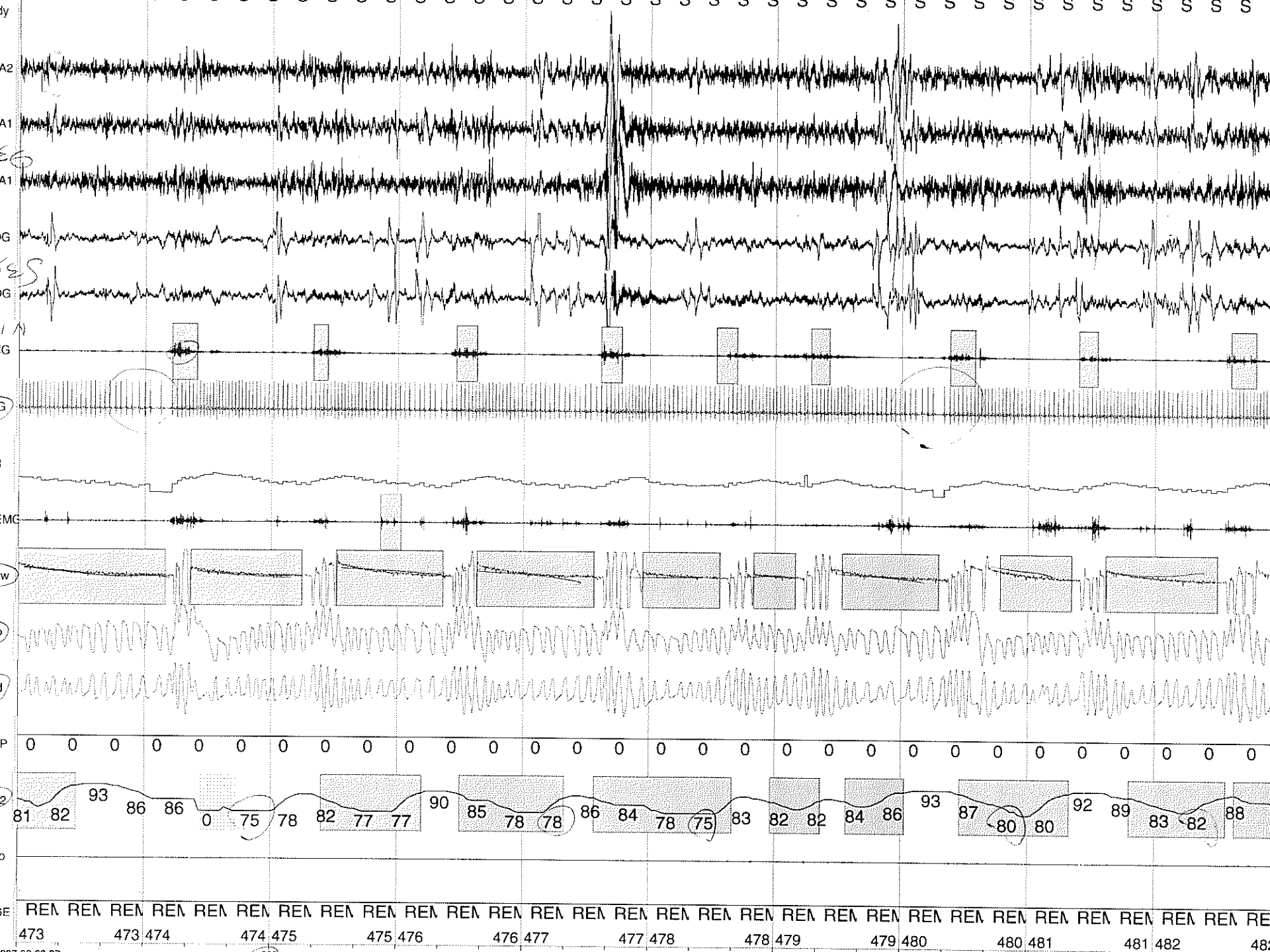


Case 2

- 52 year female with snoring
- BMI 50 (158 cm and 124 kg)
- Snores ++ and falls asleep driving
- Hypertensive, diabetic with venous stasis
- Diagnosis?







Summary case 2

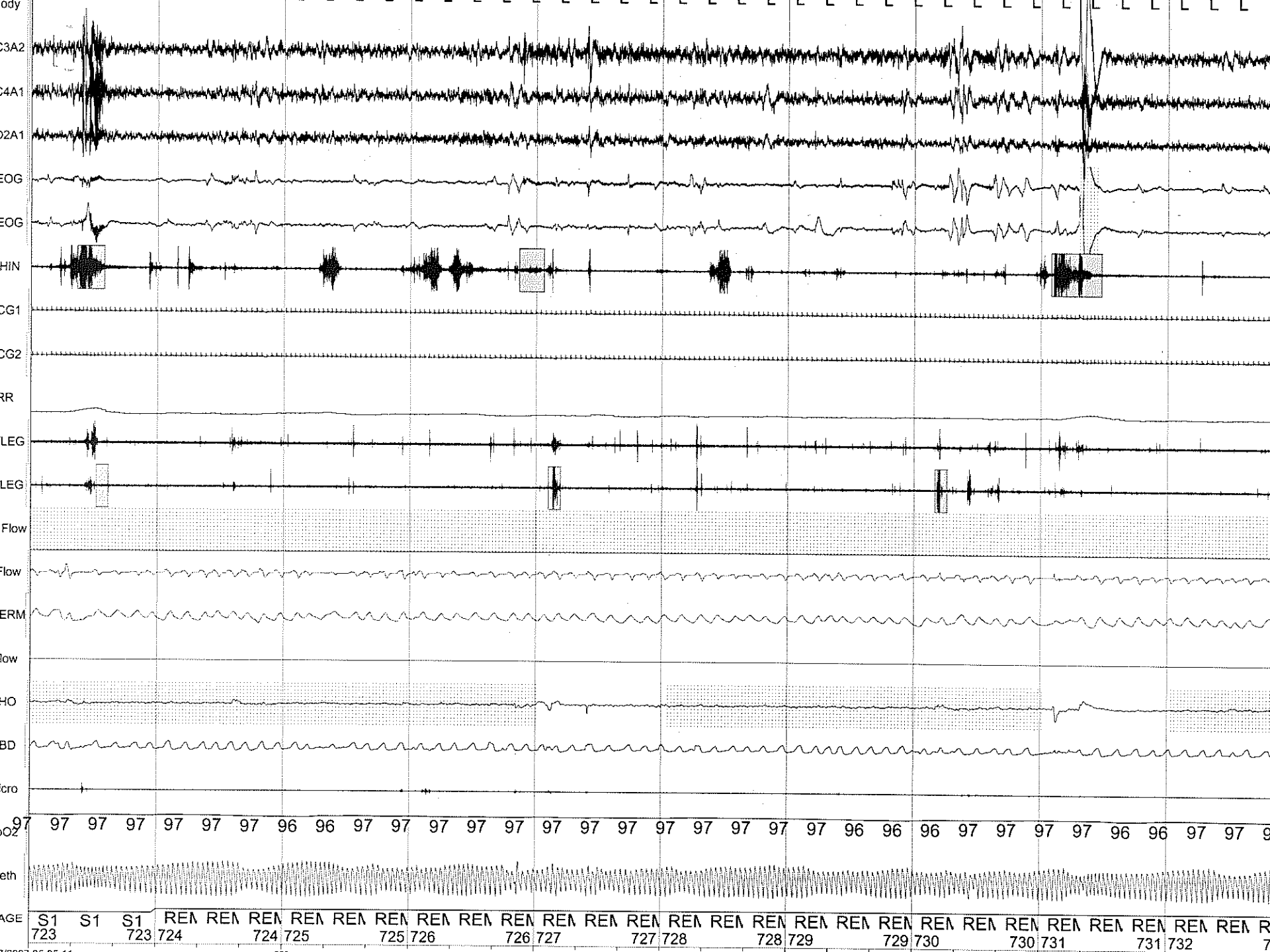
- Sleep apnea
- Common
- 70% of most sleep referrals
- Treatments are PAP, naso-oral surgery, dental appliance, facial and bariatric surgery, weight loss



Case 3

- 64 male with stridor
- 2 year history of stridor
- Impassive facies, cogwheel rigidity, poor postural reflexes
- Seen by 3 neurologists – NYD
- Due to see ent for teflon injection
- Sleep study performed ? Diagnosis?





Case 3 Diagnosis

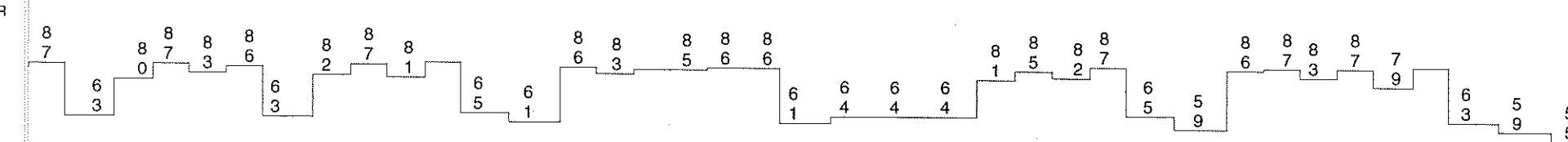
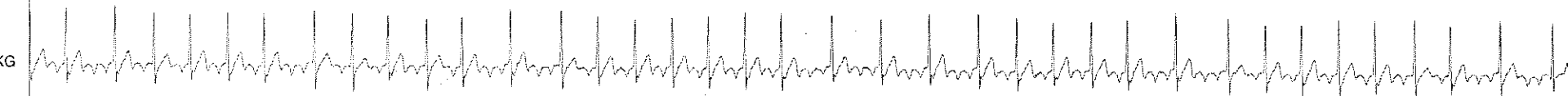
- MSA
- Developing Shy-Drager
- Parkinson's disorders
- RBD seen in 30%
- Excessive REM activity and injury
- Treat with clonazepam



Case 4

- 52 yr professor of accounting
- Marathon runner
- Episodic fainting in daytime
- Episodic seizure at night only
- Holter demonstrating sinus arrest
- Pacemaker inserted
- Seizures continue but daytime faints stop
- Diagnosis?





S2 S2

FLUTTER

Diagnosis case 4

- Neurocardiac syncope
- Temporal seizures induce nocturnal seizure
- Induce severe vagal hypertonicity and sinus arrest
- Needs to be on anticonvulsants
- Patient reports many marathon runners in his locale now have pacemakers?



Case 5

- 21 year female
- Sudden onset sleepiness hearing voices
- Was in Argentina June 2007
- 1 month studying Spanish
- Fell asleep to airport in taxicab!!
- No response to Risperdal
- Diagnosis and questions?

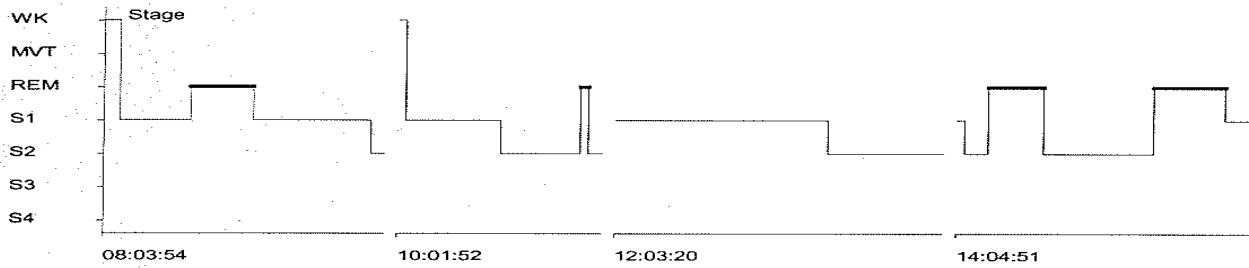


Multiple Sleep Latency Test (MSLT)

Patient identification

Last name :
 First name :
 Birth date :
 Sex :
 Acquisition date :

Hypnograms

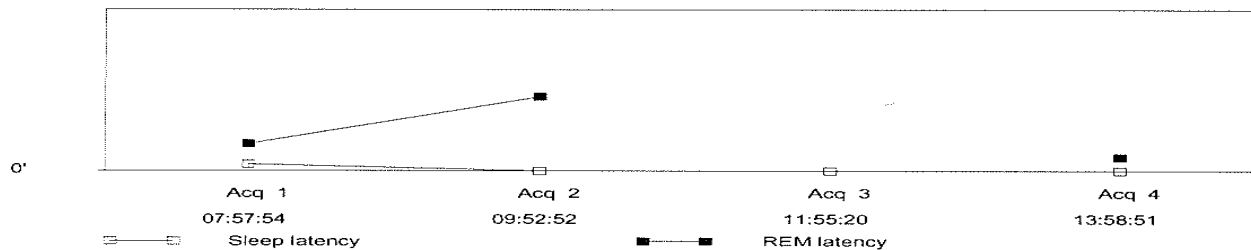


Summary

	Acq 1	Acq 2	Acq 3	Acq 4
Lights OFF	08:03:54	10:01:52	12:03:20	14:04:51
Sleep onset (SO)	08:04:54	10:02:22	12:03:20	14:04:51
Lights ON	08:21:54	10:14:52	12:24:20	14:23:51
Time in Bed	18'00"	13'00"	21'00"	19'00"
Tot Sleep Time	17'00"	12'30"	21'00"	19'00"
Sleep latency	01'00"	00'30"	00'00"	00'00"
REM latency	04'30"	11'00"	/	02'00"

Average sleep latency (4 values) : 00'22"
 Average REM latency (3 values) : 05'50"

Latencies evolution



Diagnosis case 5

- Narcolepsy
- Features cataplexy
- Sleep paralysis
- Hypnagogic hallucinations
- Voices and sleep attacks
- Treatment options depends on cataplexy



Case 6

- 49 yr female headache and poor sleep
- Chronic pain polychondritis
- Treatment from the pain clinic
- Sleepy on exam and saturations varying from 75-95 on forced ventilation
- Medications include Fentanyl patch and now on Methadone 160mg
- Diagnosis?



Diagnosis case 6

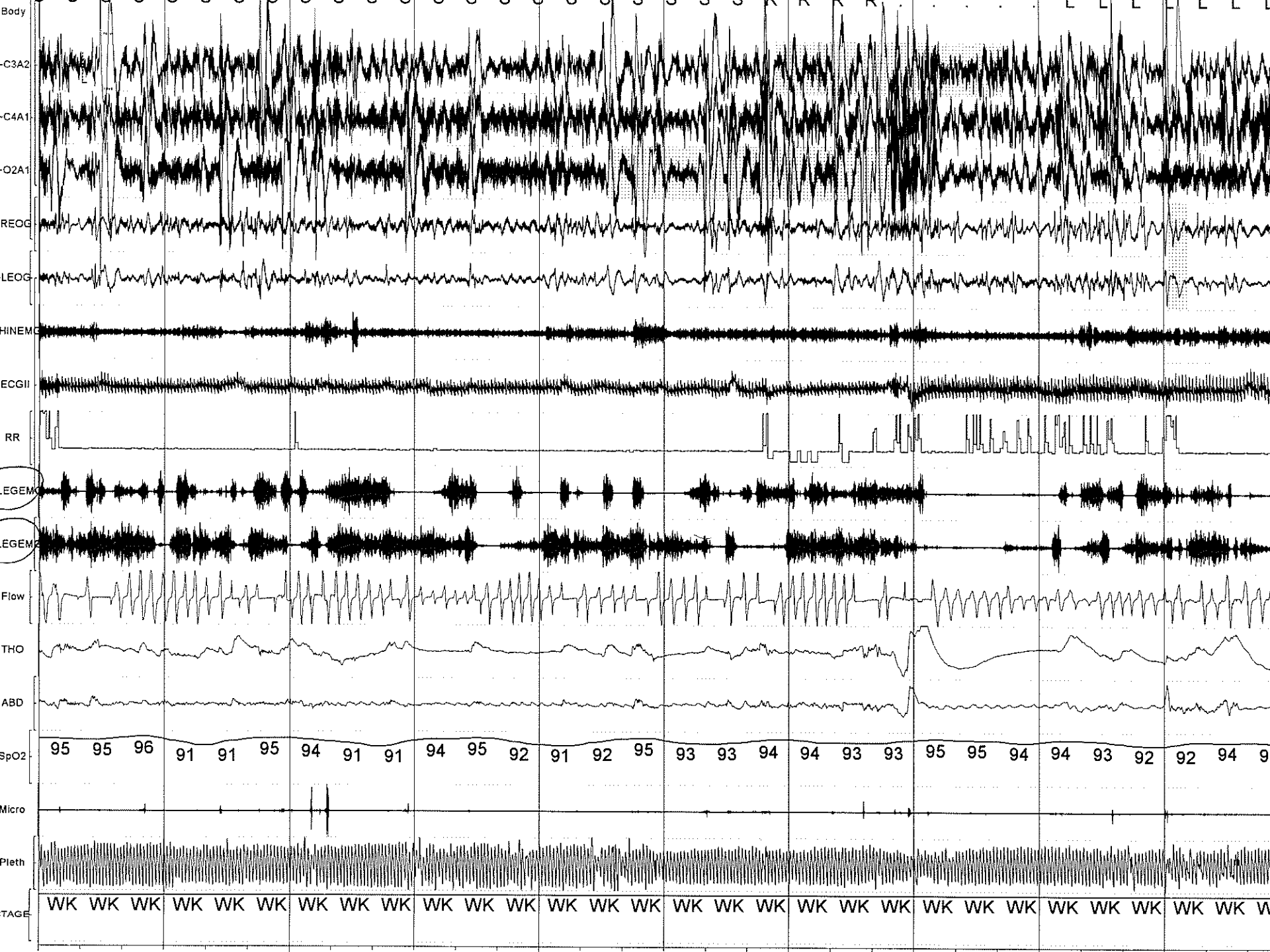
- Respiratory suppression from opioids
- Methadone induces central apnea and very poor ventilatory response



Case 7

- 41 yr carpet layer with twitching of arms and legs especially in evening
- Bed is a wreck in morning
- Normal chemistry and exam
- Smoker with normal pfts





Case 7 diagnosis

- Restless leg syndrome with periodic movement disorder in sleep and wake
- Pathology is dopaminergic and iron
- Need to check ferritin
- Use dopaminergics but get the odd gambler



Case 8

- Unusual activity at night
- Anticonvulsants tried and not helpful
- Sleep eeg is evaluated



Case 8 diagnosis

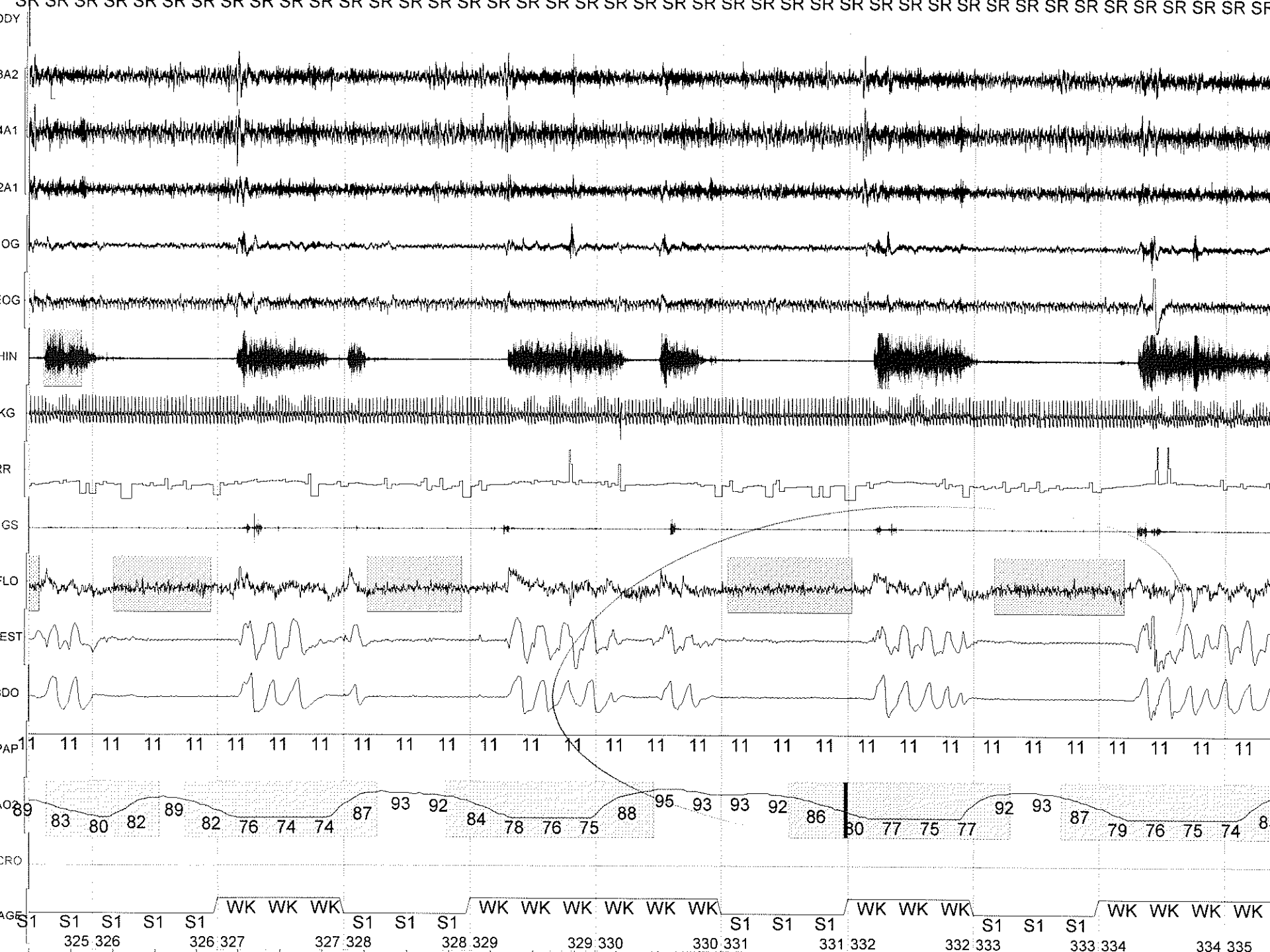
- Nocturnal seizure
- Taking dilantin and tegretol
- Doubled the dosing of tegretol and get eeg suppression
- Drug “coma”

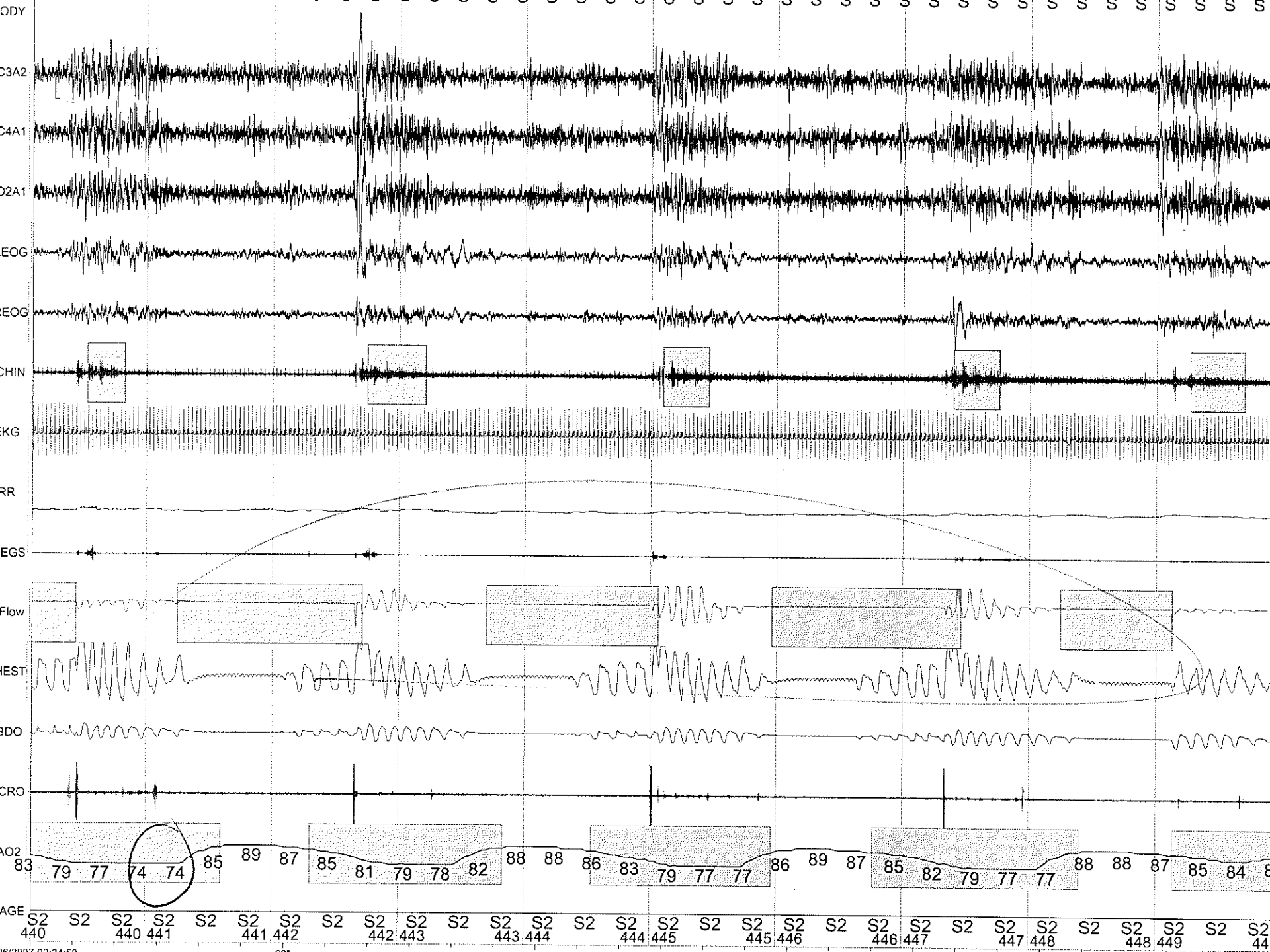


Case 9

- Cardiac disease with 20% EF
- Breathless during day and night
- Worse when supine







Case 9 Diagnosis on PSG

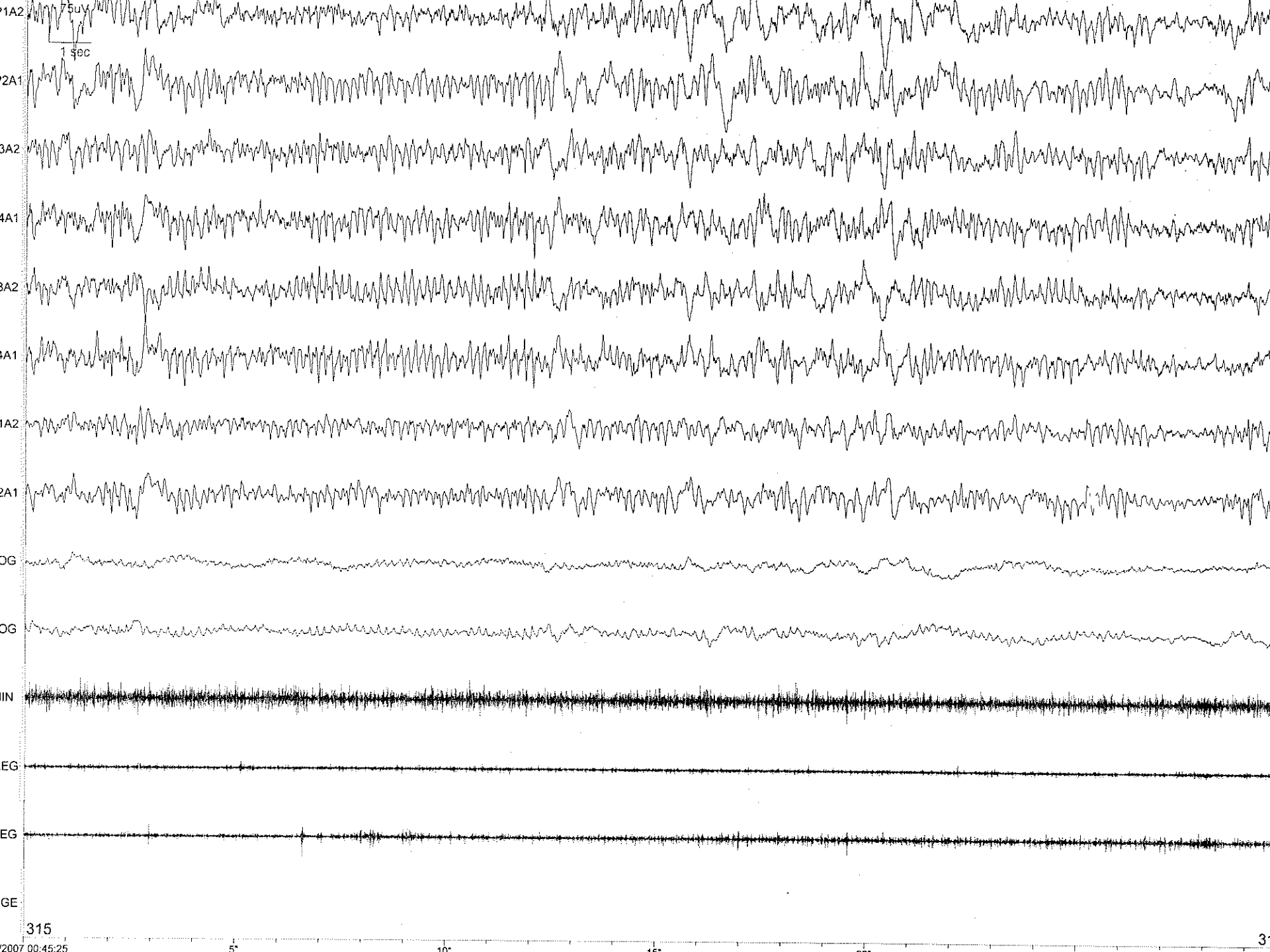
- Central sleep apnea
- Transitional changes and loop gain result in destabilization of breathing pattern
- Self perpetuates
- Need to reduce sensitivity and does well with oxygen (some) cpap (some) servo-ventilator and added CO₂

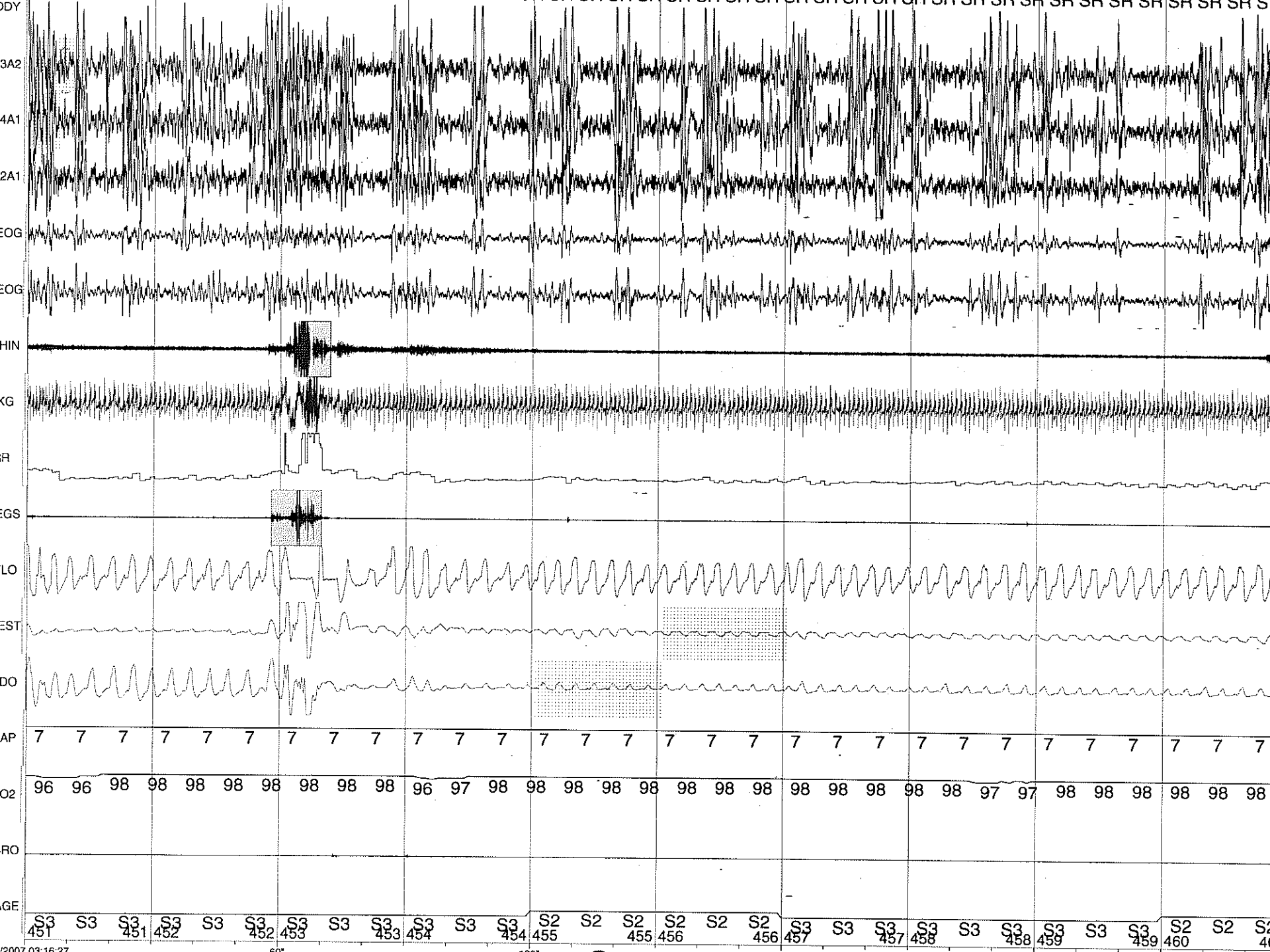


Case 10

- 44 year female with poor quality sleep
- Exam suggests features of “fibromyalgia”
- Poor mood and libido
- No energy
- “Sick and tired of being sick and tired”
- All labs normal as is exam







Diagnosis case 10

- Some will be alpha sleep pattern
- Good response to cpap on occasion
- Hypnotics are used
- Guaifenesin (anecdotal)
- Check 25 OH-Vit D etc.
- The REM rebound ones do well

