A quels aspects du sommeil faut-il prêter le plus d'attention lors de la prise en charge du patient psychiatrique ?

ANPSSSM 15/6/2011

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Xavier Preud'homme: Disclosure

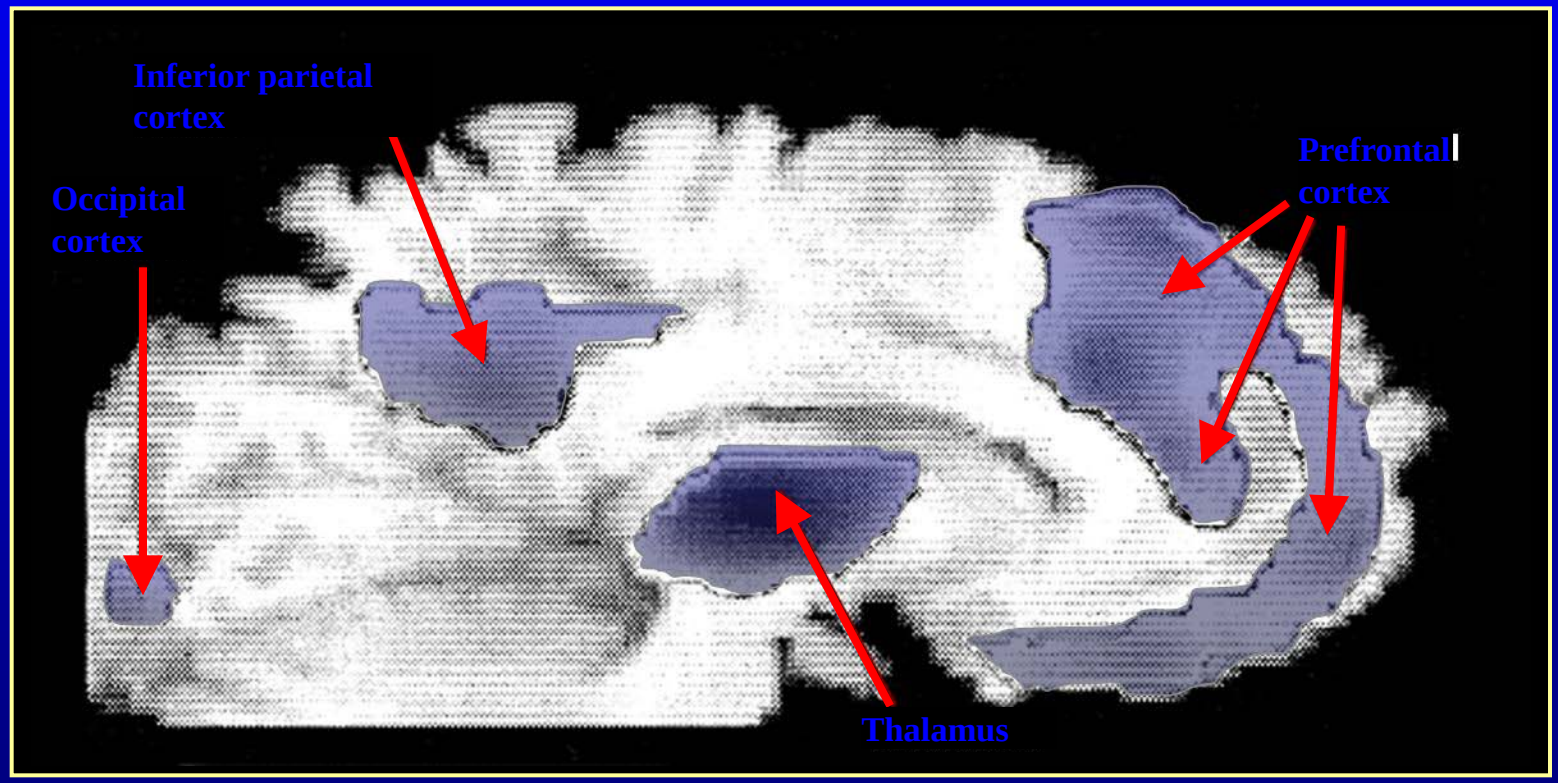
1) Investigateur principal – Bourse d'étude de Pfizer
   Analyse de la variabilité du segment RR afin de comparer déterminer l'équilibre sympathovagal chez le sujet présentant une hyperactivité de la vessie et chez le sujet sain
2) Investigateur secondaire – NIH-NIDDK
   Etude des mécanismes des mictions nocturnes y compris de l'énurésie
3) Investigateur secondaire – Astellas
   Etude de l'instabilité du muscle detrusor pendant le sommeil chez la femme
4) Investigateur principal – Bourse d'étude du CFAR Duke/NIH
   Est-il possible d'améliorer l'adhérence à la trithérapie du SIDA en traitant l'insomnie par thérapie cognitivo-comportementale?

Objectifs pédagogiques

- Survol de la physiologie et physiopathologie du sommeil chez l'humain
- Tisser les liens avec les pathologies psychiatriques
- Insister sur le potentiel d'interactions
- Organiser la présentation en fonction des approches diagnostiques des problematiques du sommeil
- Présenter les actualités thérapeutiques
- Questions - Réponses

L’impact de la privation du sommeil après 24 hr. d’éveil = Ethanol of 80

^{18}FDG PET Study of Healthy, Sleep-Deprived Adults, Showing Decreased Metabolism in the Thalamus, Prefrontal Cortex, and Inferior Parietal Cortex



FDG, fluorodeoxyglucose; PET, positron emission tomography

Thomas M et al. *J Sleep Res*. 2000;9:335-352.

The Cumulative Cost of Additional Wakefulness: Dose-Response Effects on Neurobehavioral Functions and Sleep Physiology From Chronic Sleep Restriction and Total Sleep Deprivation

Hans P.A. Van Dongen, PhD;[1] Greg Maislin, MS, MA;[1] Janet M. Mullington, PhD;[2] David F. Dinges, PhD[1]

[1]Unit for Experimental Psychiatry, Division of Sleep and Chronobiology, Department of Psychiatry, and Center for Sleep and Respiratory Neurobiology, University of Pennsylvania School of Medicine; [2]Beth Israel Deaconess Medical Center and Harvard Medical School

Objectives: To inform the debate over whether human sleep can be chronically reduced without consequences, we conducted a dose-response chronic sleep restriction experiment in which waking neurobehavioral and sleep physiological functions were monitored and compared to those for total sleep deprivation.

Design: The chronic sleep restriction experiment involved randomization to one of three sleep doses (4 h, 6 h, or 8 h time in bed per night), which were maintained for 14 consecutive days. The total sleep deprivation experiment involved 3 nights without sleep (0 h time in bed). Each study also involved 3 baseline (pre-deprivation) days and 3 recovery days.

Setting: Both experiments were conducted under standardized laboratory conditions with continuous behavioral, physiological and medical monitoring.

Participants: A total of $n = 48$ healthy adults (ages 21–38) participated in the experiments.

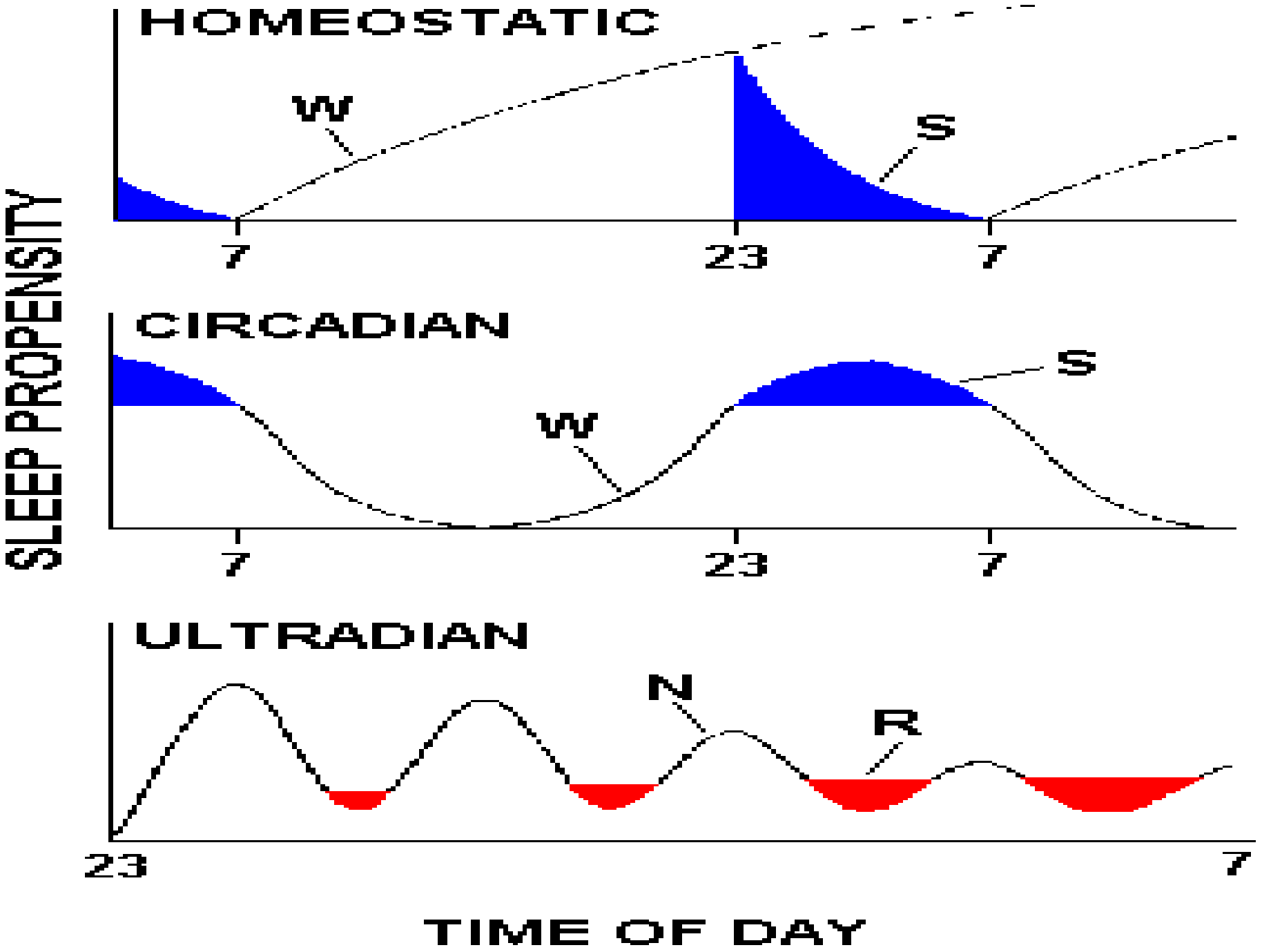
Interventions: Nocturnal sleep periods were restricted to 8 h, 6 h or 4 h per day for 14 days, or to 0 h for 3 days. All other sleep was prohibited.

Results: Chronic restriction of sleep periods to 4 h or 6 h per night over 14 consecutive days resulted in significant cumulative, dose-dependent deficits in cognitive performance on all tasks. Subjective sleepiness ratings showed an acute response to sleep restriction but only small further increases on subsequent days, and did not significantly differentiate the 6 h and 4 h conditions. Polysomnographic variables and δ power in the non-REM sleep EEG—a putative marker of sleep homeostasis—displayed an acute response to sleep restriction with negligible further changes across the 14 restricted nights. Comparison of chronic sleep restriction to total sleep deprivation showed that the latter resulted in disproportionately large waking neurobehavioral and sleep δ power responses relative to how much sleep was lost. A statistical model revealed that, regardless of the mode of sleep deprivation, lapses in behavioral alertness were near-linearly related to the cumulative duration of wakefulness in excess of 15.84 h (s.e. 0.73 h).

Conclusions: Since chronic restriction of sleep to 6 h or less per night produced cognitive performance deficits equivalent to up to 2 nights of total sleep deprivation, it appears that even relatively moderate sleep restriction can seriously impair waking neurobehavioral functions in healthy adults. Sleepiness ratings suggest that subjects were largely unaware of these increasing cognitive deficits, which may explain why the impact of chronic sleep restriction on waking cognitive functions is often assumed to be benign. Physiological sleep responses to chronic restriction did not mirror waking neurobehavioral responses, but cumulative wakefulness in excess of a 15.84 h predicted performance lapses across all four experimental conditions. This suggests that sleep debt is perhaps best understood as resulting in additional wakefulness that has a neurobiological "cost" which accumulates over time.

Key Words: chronic sleep restriction, partial sleep deprivation, total sleep deprivation, cognitive performance, subjective sleepiness, cumulative deficits, sleep debt, wake extension, core sleep, sleep need

Citation: Van Dongen HPA, Maislin G, Mullington JM, Dinges DF. The cumulative cost of additional wakefulness: dose-response effects on neurobehavioral functions and sleep physiology from chronic sleep restriction and total sleep deprivation. SLEEP 2003;2:117-126.


HOMEOSTATIC
W
S
7
23
7
CIRCADIAN
S
W
7
23
7
ULTRADIAN
N
R
23
7
SLEEP PROPENSITY
TIME OF DAY

Interindividual Variation in Sleep Duration and Its Association With Sleep Debt in Young Adults

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[1]Division of Sleep Medicine, Brigham and Women's Hospital and Harvard Medical School, Boston, MA; [2]Surrey Sleep Research Centre, School of Biomedical and Molecular Sciences, University of Surrey, Guildford, UK

Study Objective: To determine whether variation in sleep duration reflects variation in sleep need or self-imposed sleep restriction.

Design: After habitual bedrest duration of participants was assessed during a 2-week outpatient protocol, volunteers were scheduled to sleep according to this schedule for 1 week prior to and the first night after admission to a general clinical research center. The inpatient protocol included multiple sleep latency testing on the second day and sleep recordings during a bedrest extension protocol that included 16 hours of sleep opportunity (12 hours at night and 4 hours at midday) for 3 consecutive days.

Setting: Outpatient monitoring followed by inpatient assessment of sleep.

Participants: Seventeen healthy volunteers (10 women) aged 18-32 years without clinical sleep disorders.

Interventions: Extension of sleep opportunity.

Measurements and Results: The habitual bedrest duration varied from 6.1 to 10.3 hours. Individuals with shorter habitual bedrest duration fell asleep more quickly and frequently during the multiple sleep latency test than did those with longer habitual bedrest duration. On the first day of extended sleep opportunity, the total sleep time of all individuals was greater than their habitual bedrest duration; the average increase in total sleep time was 4.9 hours (P = 0.001). The increase in total sleep time declined across the 3 day bedrest-extension protocol (P = 0.003 for trend). During the third day of increased sleep opportunity, the total sleep time was negatively associated with habitual bedrest duration (P = 0.005); individuals with shorter habitual bedrest duration continued to sleep more than those with longer habitual bedrest duration.

Conclusion: Those individuals with shorter habitual sleep durations carry a higher sleep debt than do those with longer habitual sleep duration. Interindividual variation in sleep duration may primarily reflect variation in self-selected sleep restriction or wake extension.

Keywords: Sleep, homeostasis, recovery, sleep duration

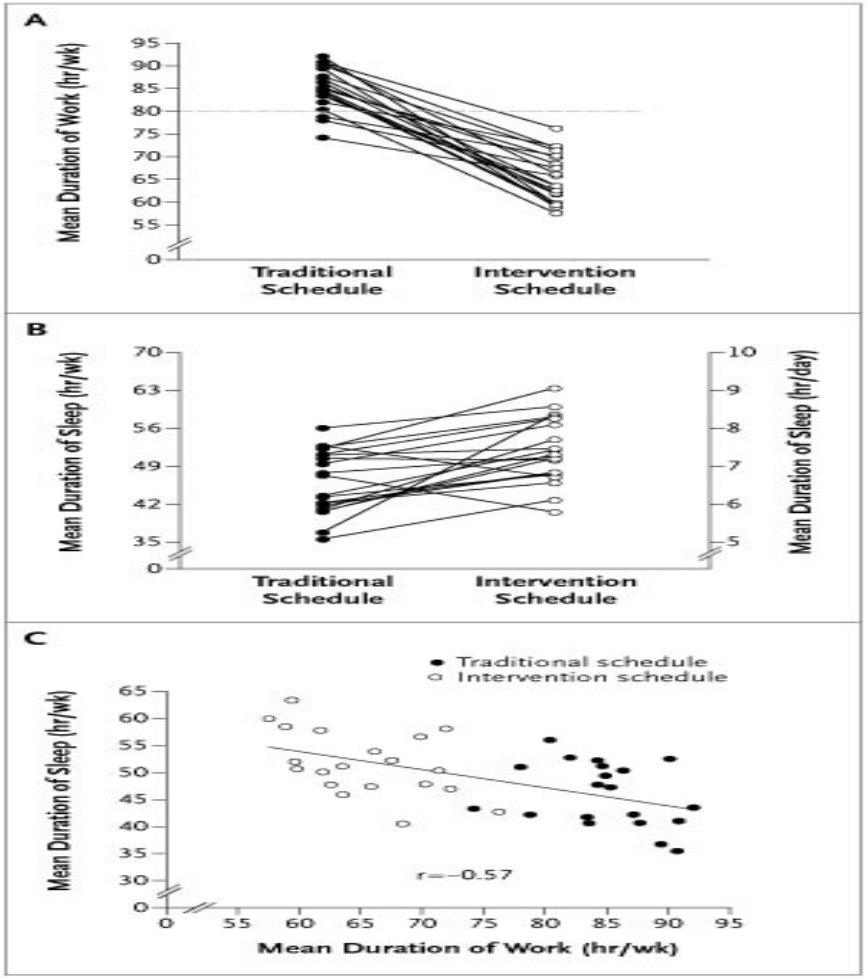
Citation: Klerman EB; Dijk DJ. Interindividual variation in sleep duration and its association with sleep debt in young adults. *SLEEP* 2005;28(10): 1253-1259.

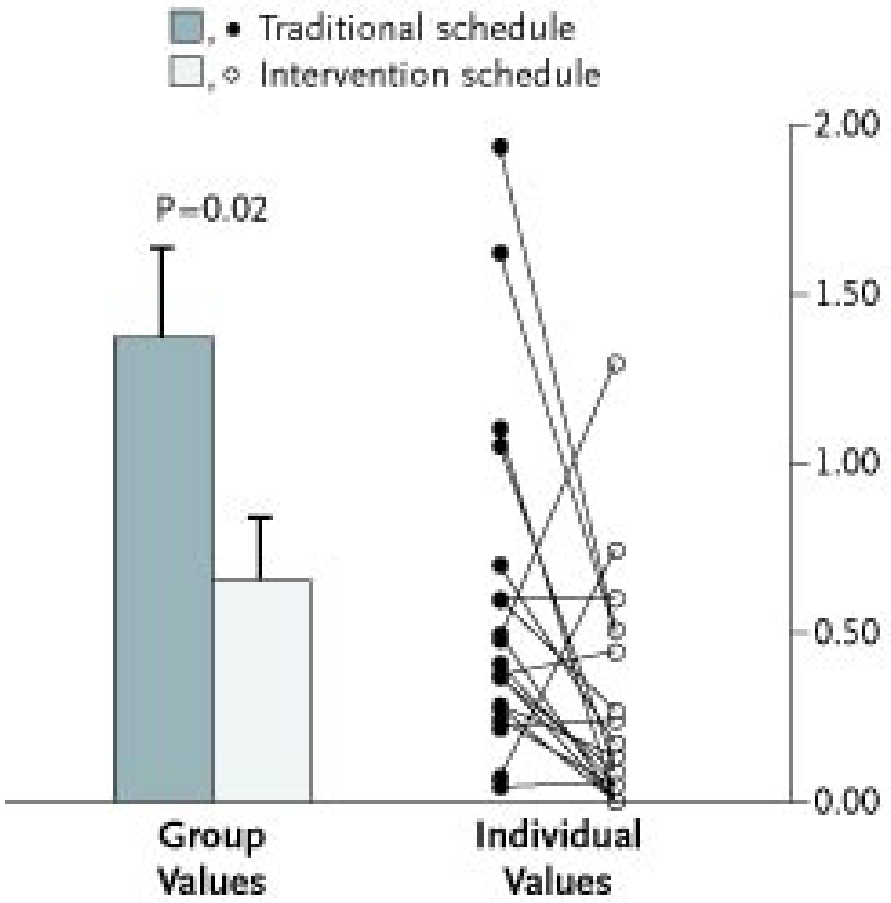
INTRODUCTION

SELF-REPORTED SLEEP DURATION VARIES CONSIDERABLY BETWEEN INDIVIDUALS. RECENT SURVEY DATA

covery function, although the timing of recovery is modulated by the biological circadian clock.[10-12] Variation in sleep duration may reflect differences in either the sleep homeostatic process or the circadian process. More specifically, these variations may

Mean (+SE) Number of Attentional Failures among the 20 Interns as a Group and Individually while Working Overnight (11 p.m. to 7 a.m.) during the Traditional Schedule and the Intervention Schedule





Lockley, S. et al. N Engl J Med 2004;351:1829-1837

The NEW ENGLAND JOURNAL of MEDICINE

Inadequate Sleep as a Risk Factor for Obesity: Analyses of the NHANES I

James E. Gangwisch, PhD[1]; Dolores Malaspina, MD, MPH[2]; Bernadette Boden-Albala, Dr.PH[3]; Steven B. Heymsfield, MD[4]

[1]Mailman School of Public Health, Department of Epidemiology; [2]Department of Psychiatry, Division of Clinical Neurobiology; [3]Department of Neurology and Department of Sociomedical Sciences; [4]Obesity Research Center, St. Luke's-Roosevelt Hospital Center; Columbia University, College of Physicians and Surgeons, New York, NY

Study Objectives: Sleep deprivation has been hypothesized to contribute toward obesity by decreasing leptin, increasing ghrelin, and compromising insulin sensitivity. This study examines cross-sectional and longitudinal data from a large United States sample to determine whether sleep duration is associated with obesity and weight gain.

Design: Longitudinal analyses of the 1982-1984, 1987, and 1992 NHANES I Followup Studies and cross-sectional analysis of the 1982-1984 study.

Setting: Probability sample of the civilian noninstitutionalized population of the United States.

Participants: Sample sizes of 9,588 for the cross-sectional analyses, 8,073 for the 1987, and 6,981 for the 1992 longitudinal analyses.

Measurements and Results: Measured weight in 1982-1984 and self-reported weights in 1987 and 1992. Subjects between the ages of 32 and 49 years with self-reported sleep durations at baseline less than 7 hours had higher average body mass indexes and were more likely to be obese than subjects with sleep durations of 7 hours. Sleep durations over 7 hours were not consistently associated with either an increased or decreased likelihood of obesity in the cross-sectional and longitudinal results. Each additional hour of sleep at baseline was negatively associated with change in body mass index over the follow-up period, but this association was small and statistically insignificant.

Conclusions: These findings support the hypothesis that sleep duration is associated with obesity in a large longitudinally monitored United States sample. These observations support earlier experimental sleep studies and provide a basis for future studies on weight control interventions that increase the quantity and quality of sleep.

Keywords: Sleep, obesity, insulin resistance

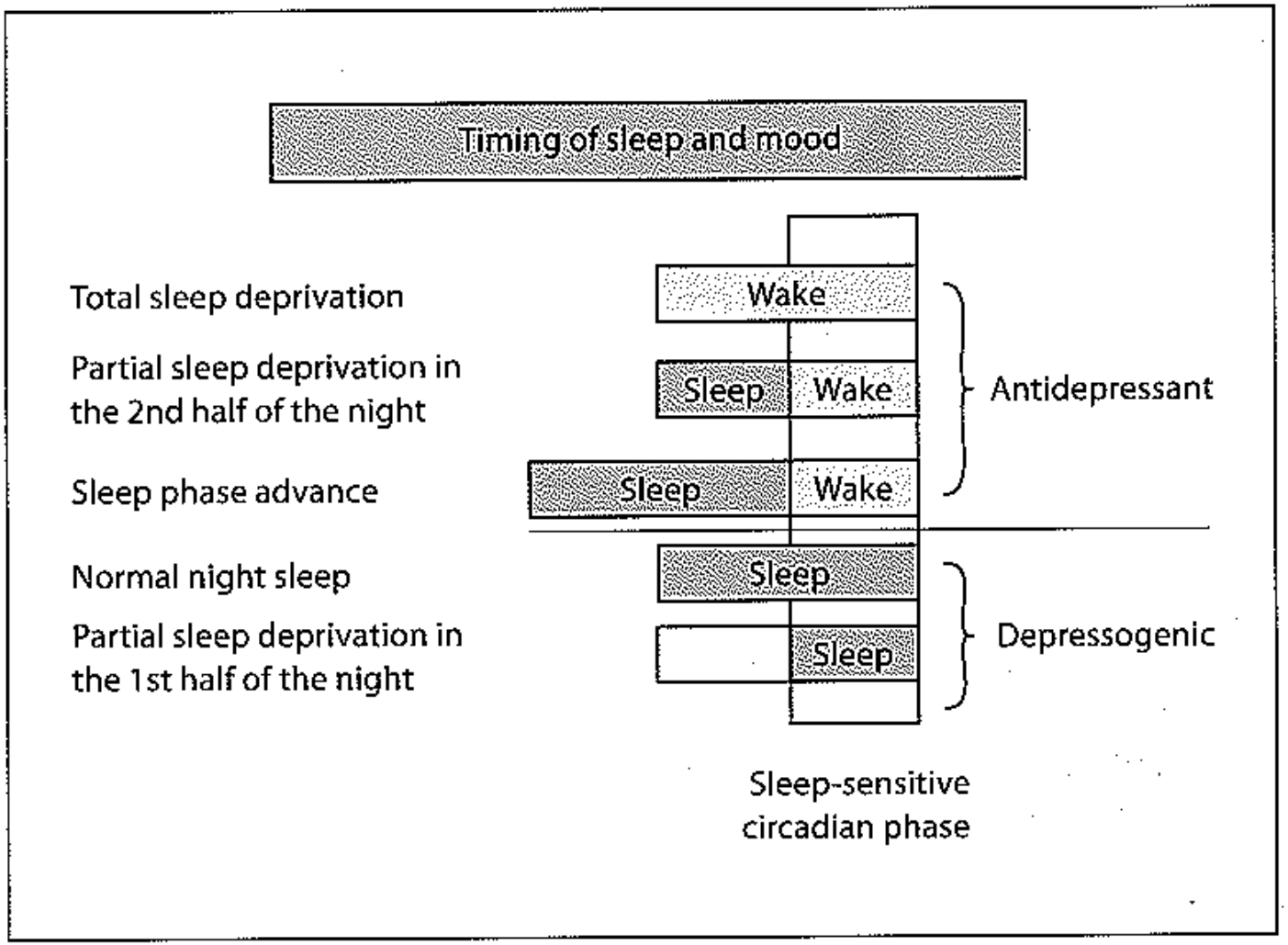
Citation: Gangwisch JE; Malaspina D; Boden-Albala B et al. Inadequate sleep as a risk factor for obesity: analyses of the NHANES I. *SLEEP* 2005;28(10): 1289-1296.

2004 *Annals of Internal Medicine*
4 hr de sommeil => - 18% leptine, +28% ghreline, + 24% appétit

in healthy young lean men.[6] C
results obtained after the subje
condition and after a sleep-re
tailment condition resulted in
ghrelin levels, and markedly ele

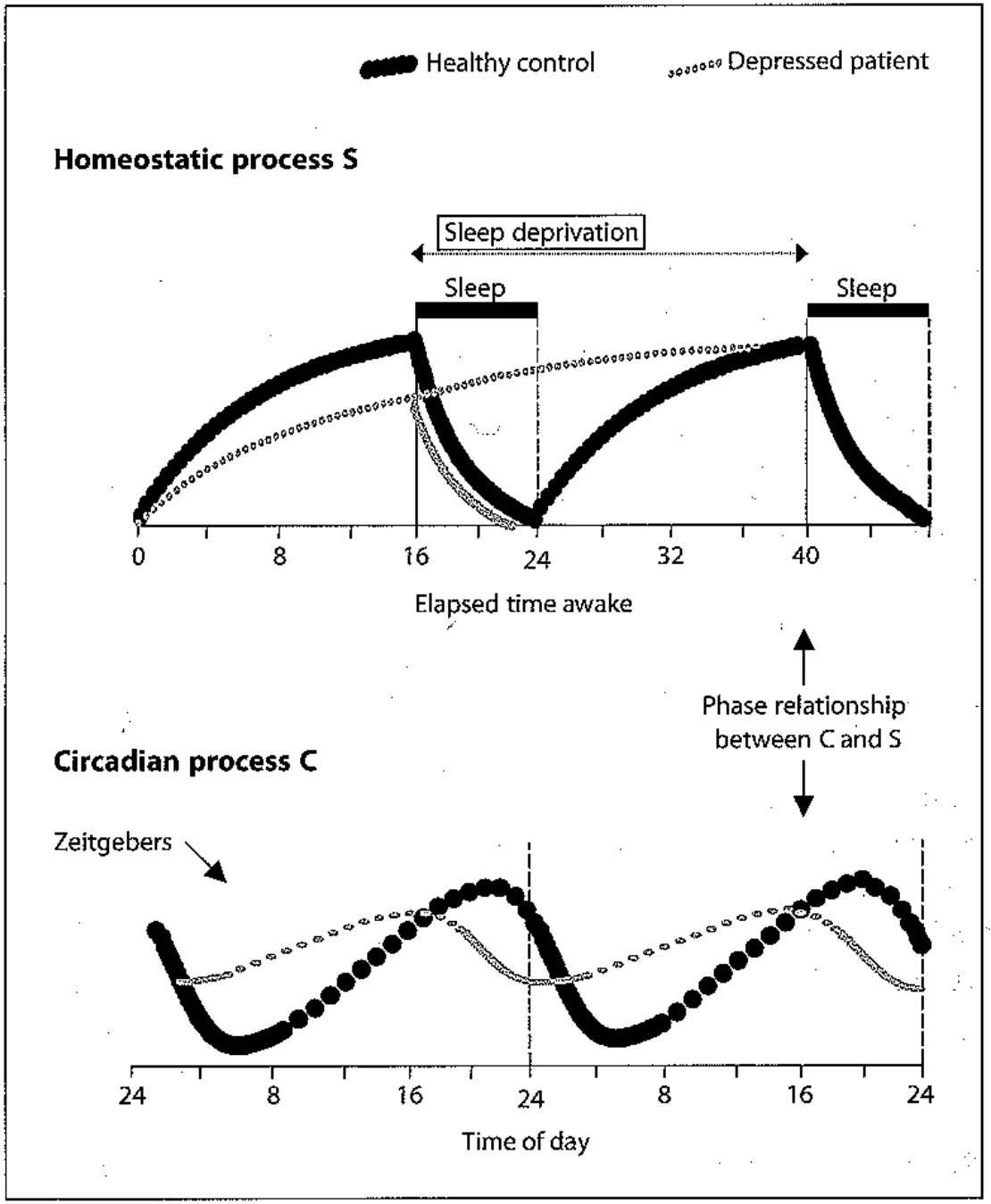
reaching epidemic proportions throughout the developed world

Paradigme de la privation de sommeil

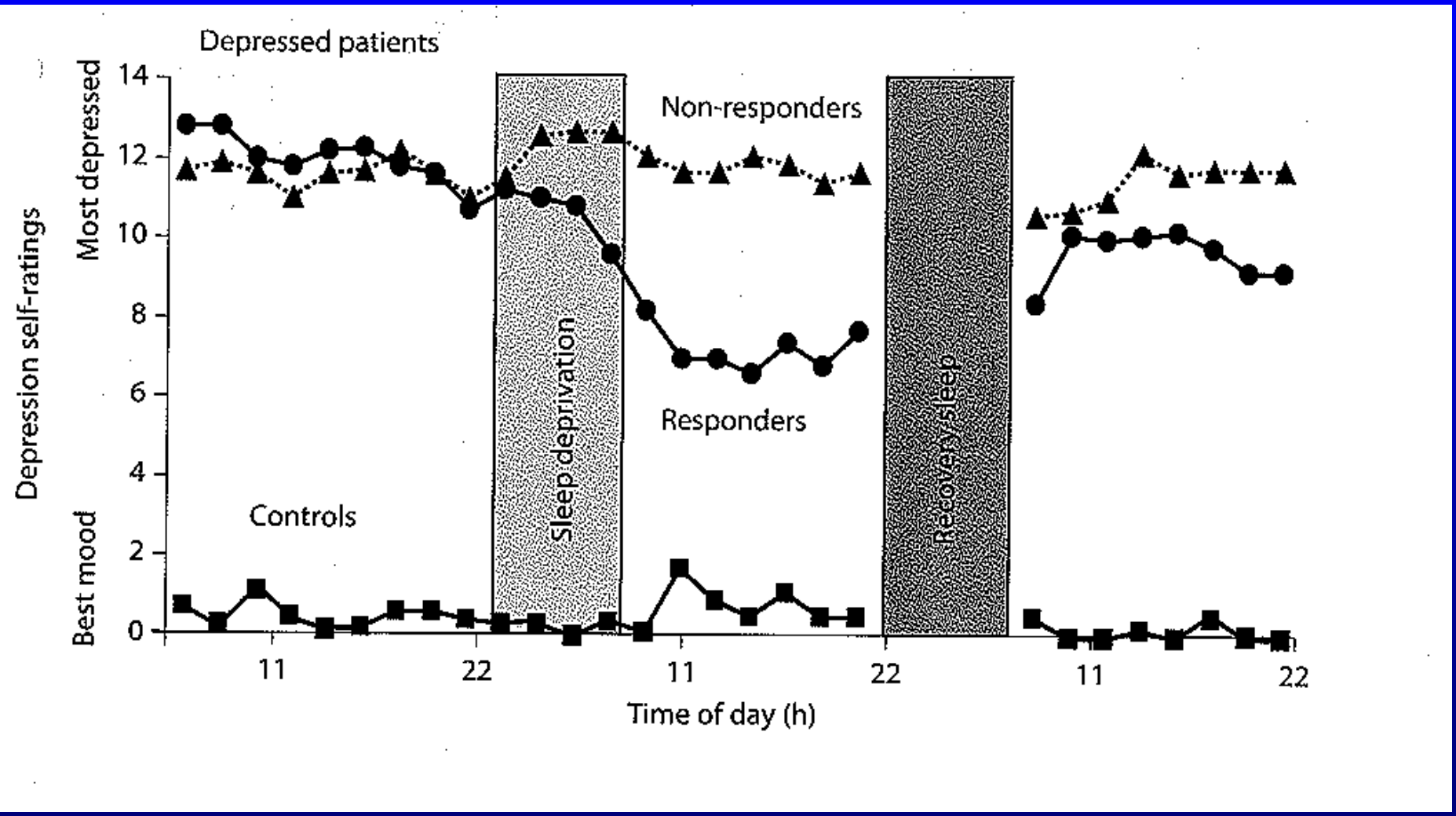


Privation de sommeil

5% => hypomanie
6% => manie
33% => ok
> 1 nuit (BZD)



Wake Therapy: “Thérapie par l’eveil”



Dark Therapy: “L’anti-lumière”

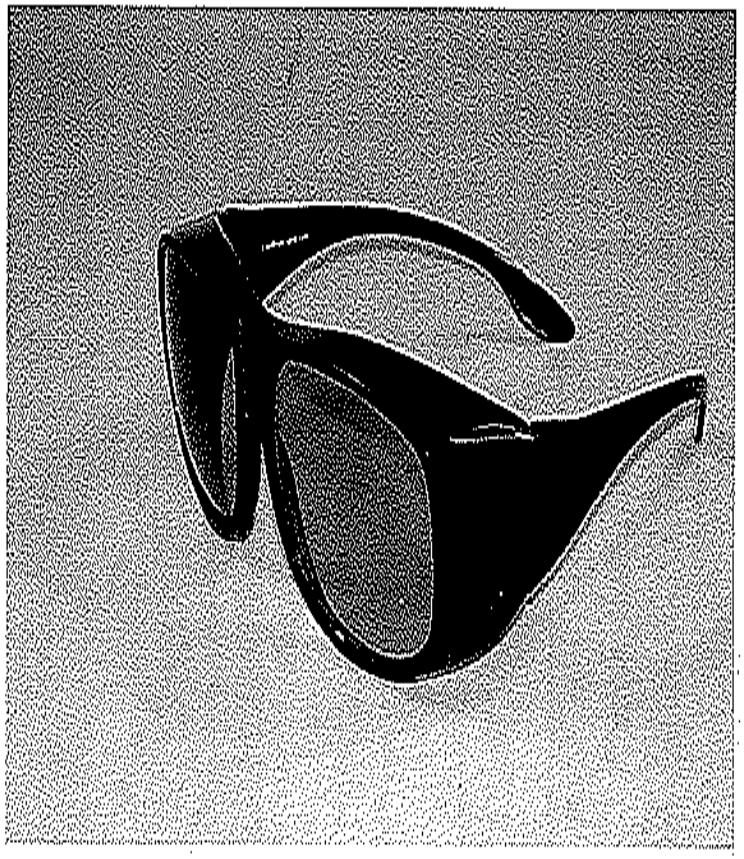
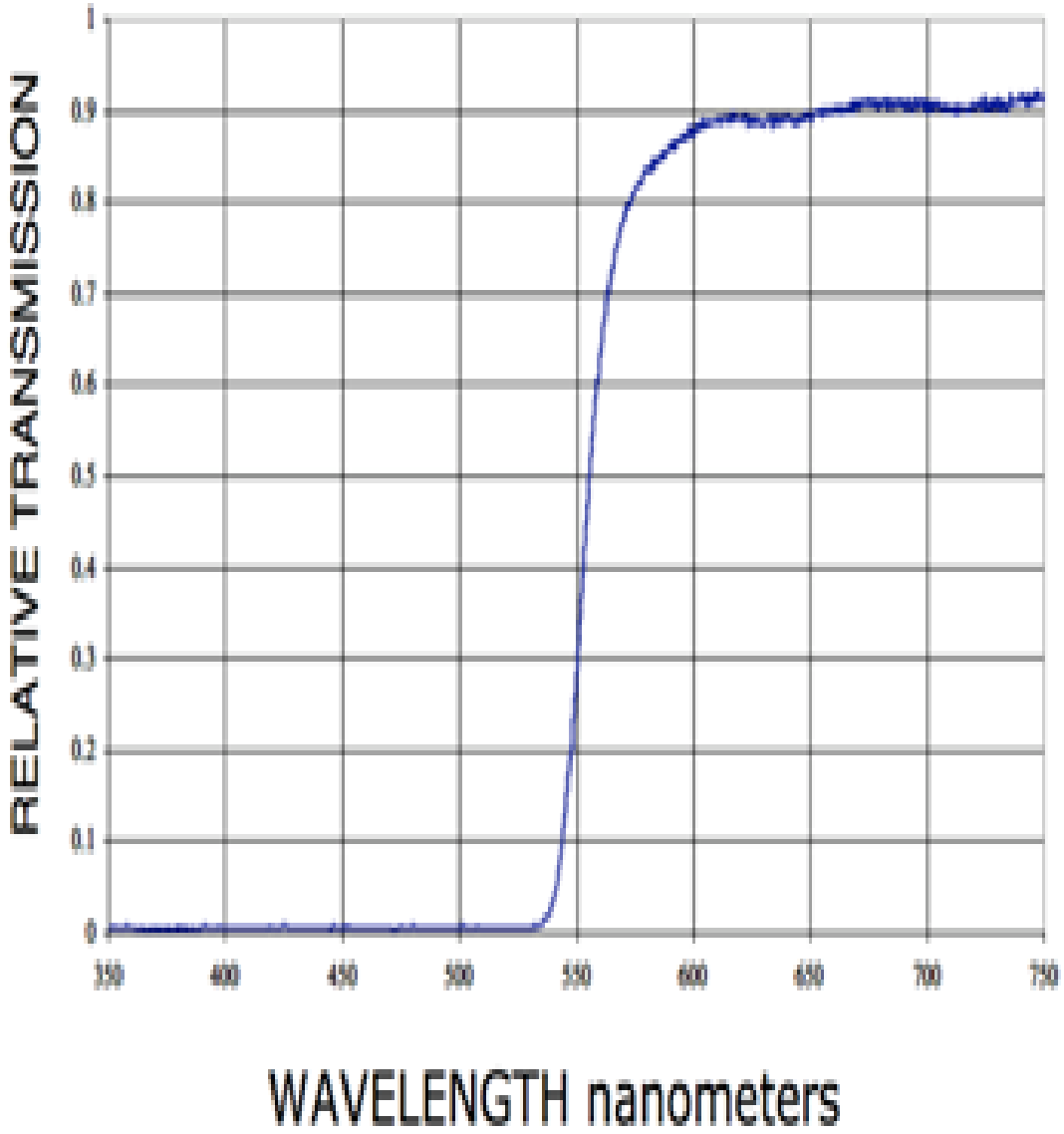


Fig. 19. Amber filters in glasses worn to filter short-wavelength light <535 nm (Center for Environmental Therapeutics).



Critères du diagnostic de l'insomnie primaire selon le DSM-IV

Significant Distress or Impairment in Function <u>for 1 month</u>

AND

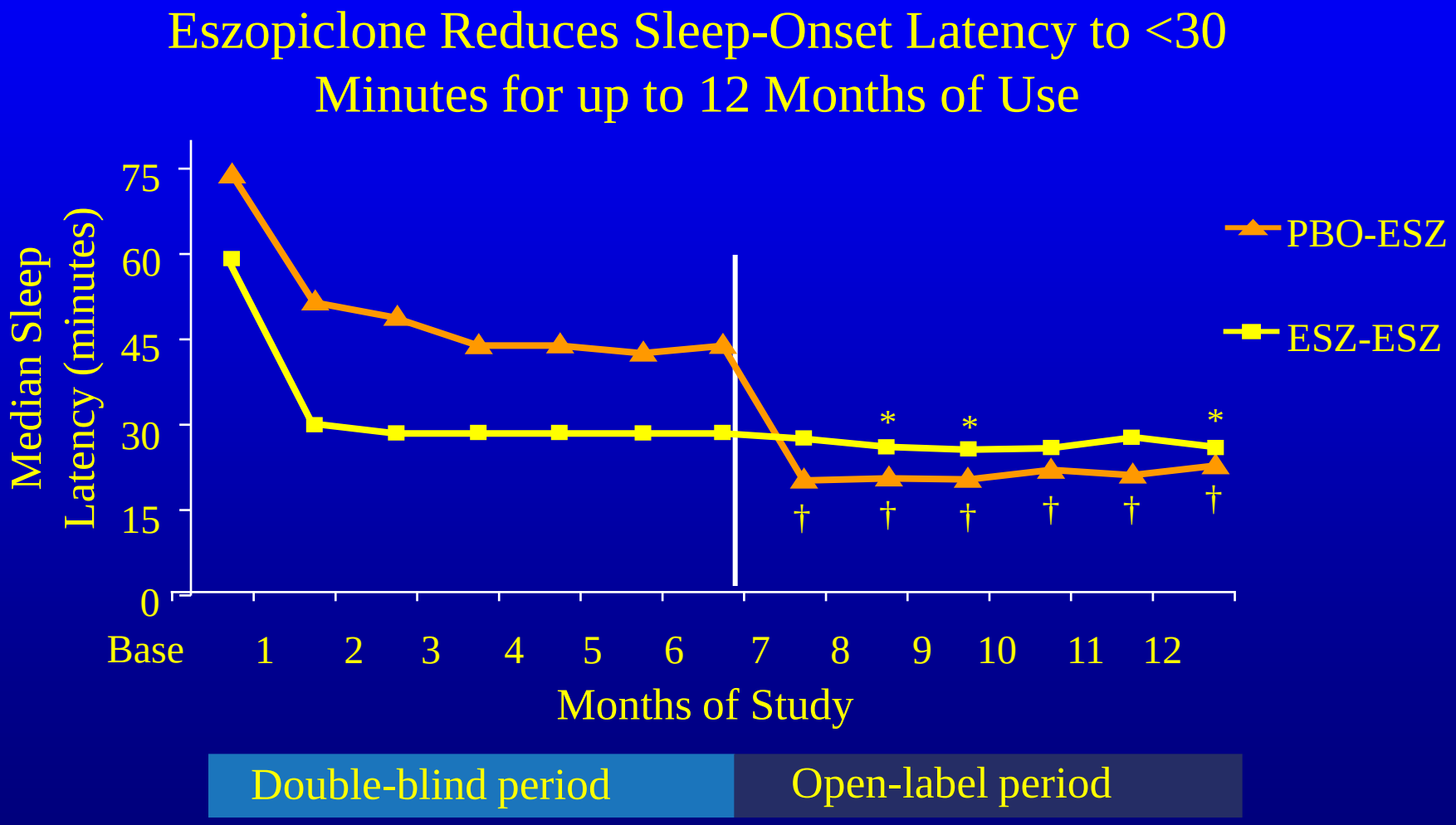
Difficulty falling asleep ***and/or*** **Difficulty staying asleep** ***and/or*** **Non-restorative sleep**

American Psychiatric Association. *DSM-IV TR*. 2000.

L’agenda du sommeil

Date	10/08/08	10/09/08	10/11/08	10/12/08	10/13/08
Nighttime Medications					
Alcohol					
Bed Time					
Wake Time					
Time Out of Bed in a.m.					
Awakenings and Time Awake for Each One					
Quality of Sleep (0-5)					
Feeling of Restoration in the a.m. (0-5)					

Latence de sommeil (subjective): 6 & 12 mois sous eszopiclone



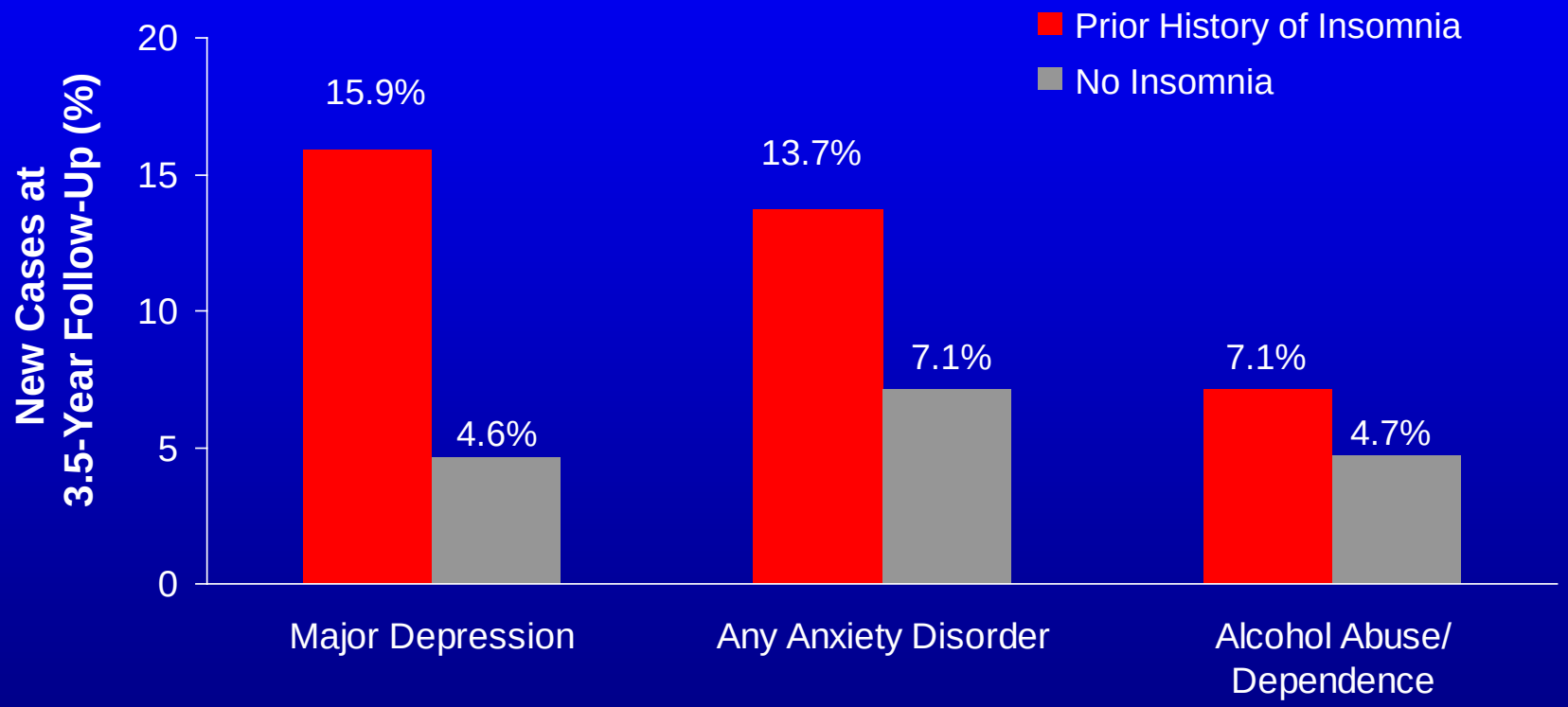
Roth T, et al. *Sleep Med.* 2005;6:487-95.

Indications pour une aproche non pharmacologique

- Hygi*è*ne du sommeil
- **Thérapie cognitivo-comportementale => Succ*è*s = celui des hypmotiques**
- Approche cognitive
- Approche comportementale:
  Diminuer le stimulus
  Restreindre le temps de sommeil

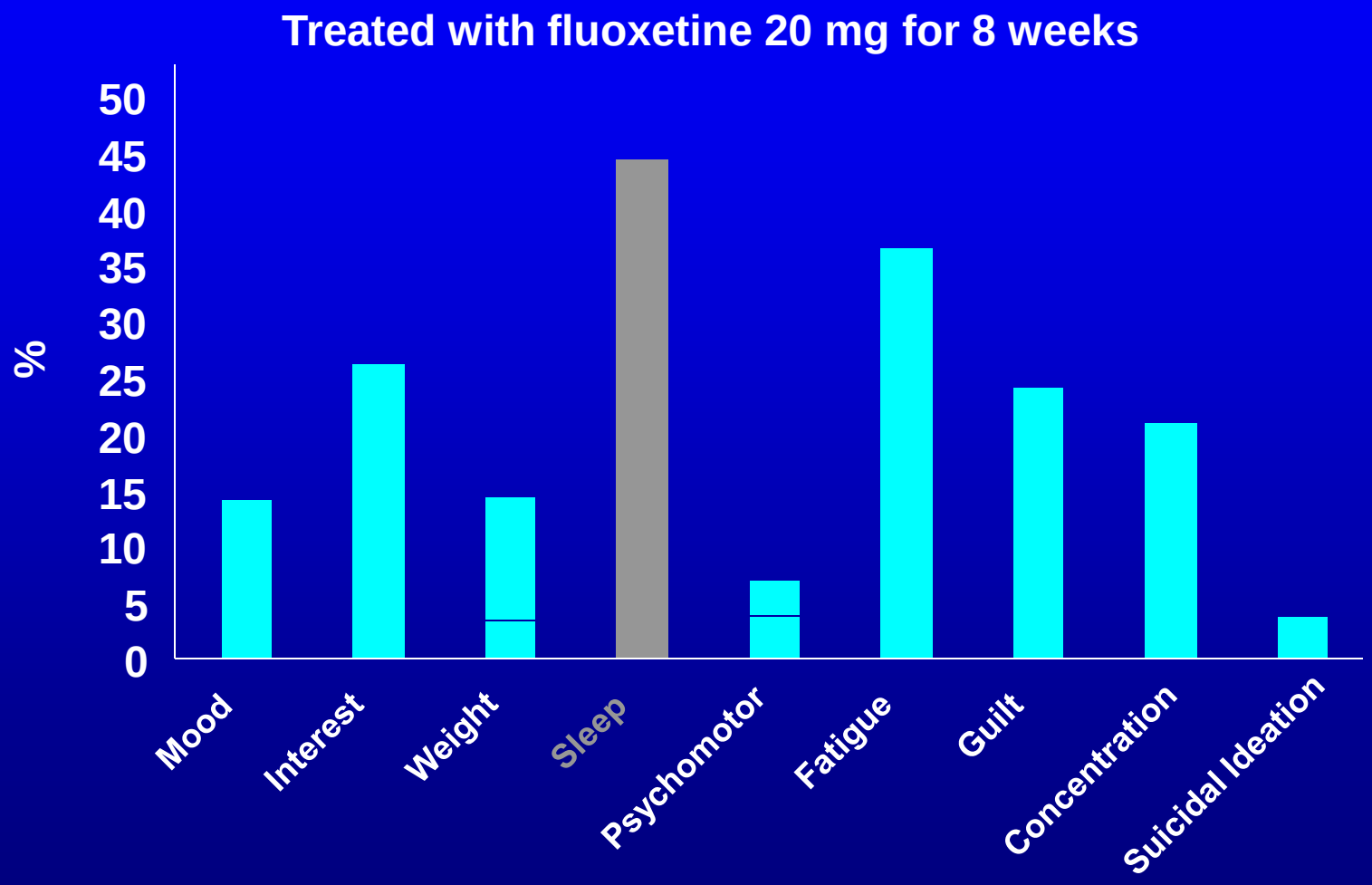
Wohlgemuth and Krystal, 2005; Krystal et al., 1998

Insomnie = risque de développer d'autres maladies mentales



Breslau N, et al. *Biol Psychiatry*. 1996;39:411-418. NIH. *Sleep*. 2005;28:1049-1057.

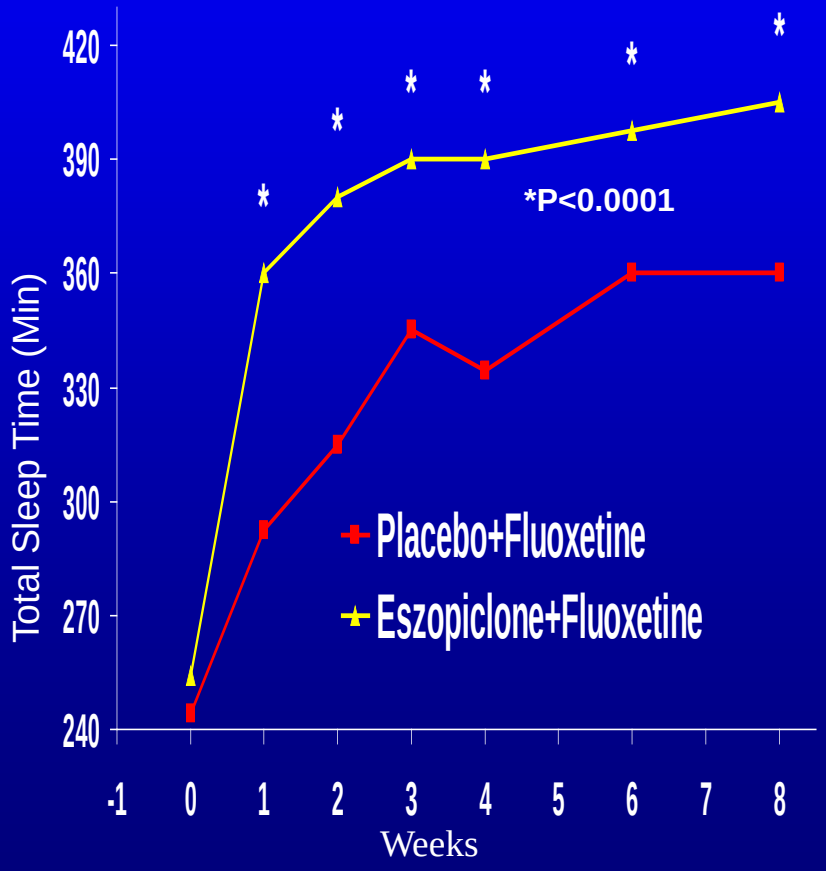
Symptoms résiduels chez les patients déprimés en rémission



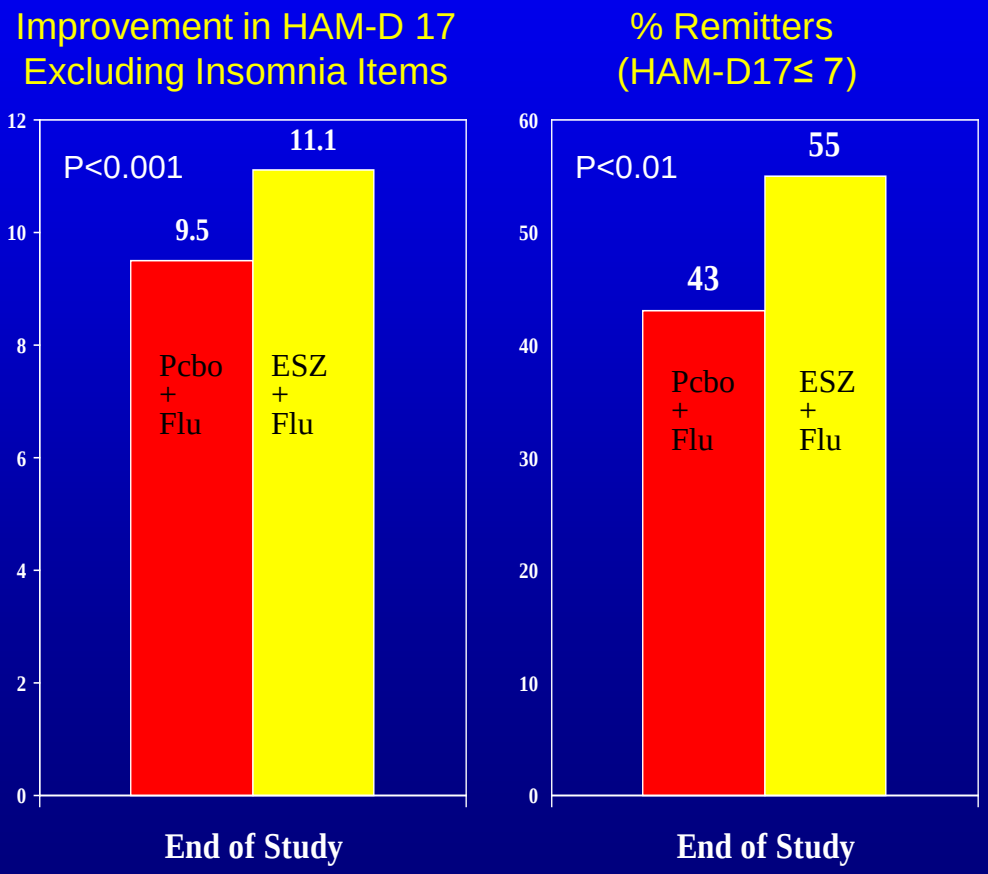
Nierenberg AA, et al. *J Clin Psychiatry.* 1999;60:221-225.

8 Week Double-Blind, Randomized Trial-N=545 Fluoxetine+Eszopiclone vs. Fluoxetine+Placebo

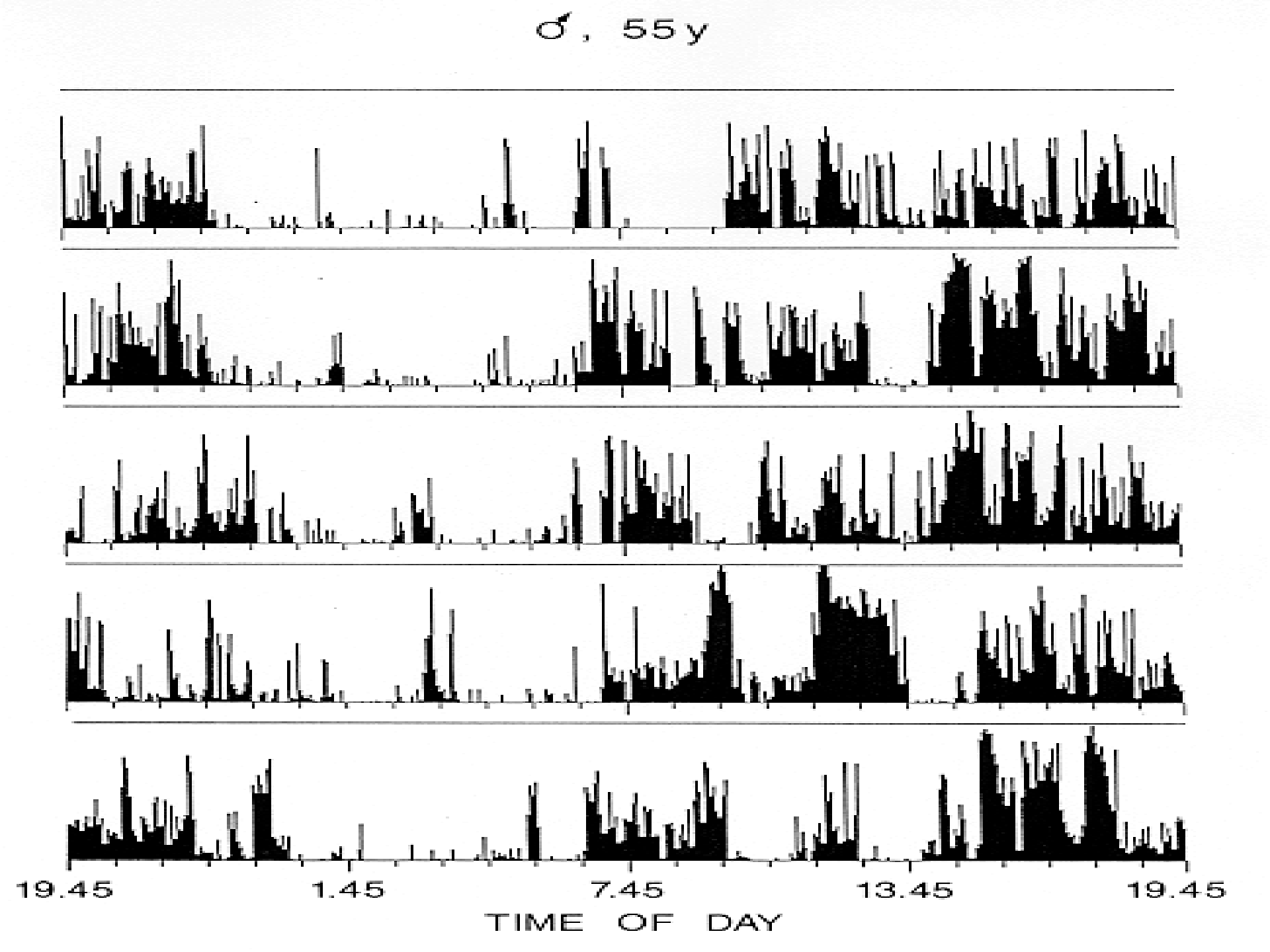
Insomnia Efficacy



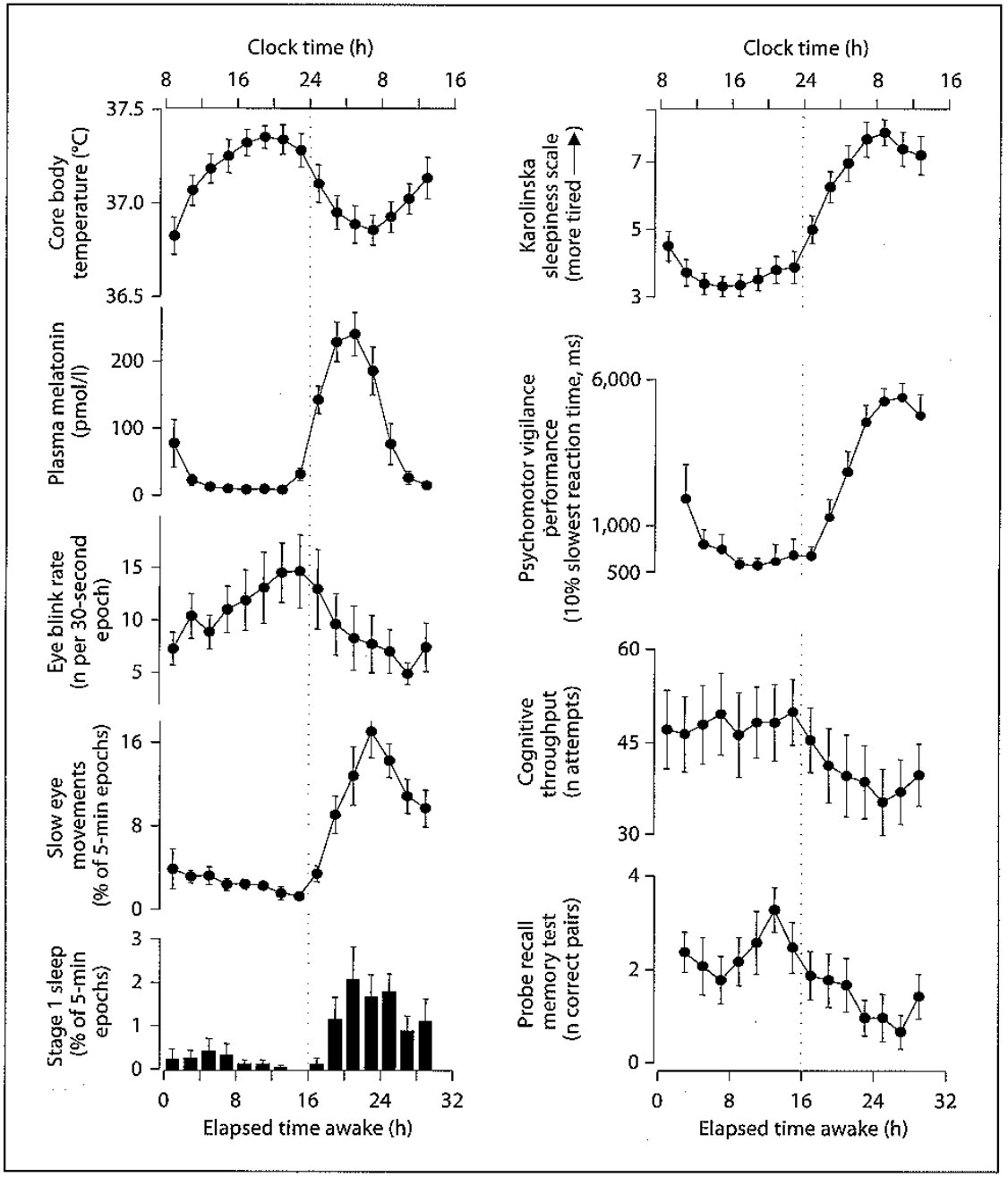
Significant Effect on Depression Response



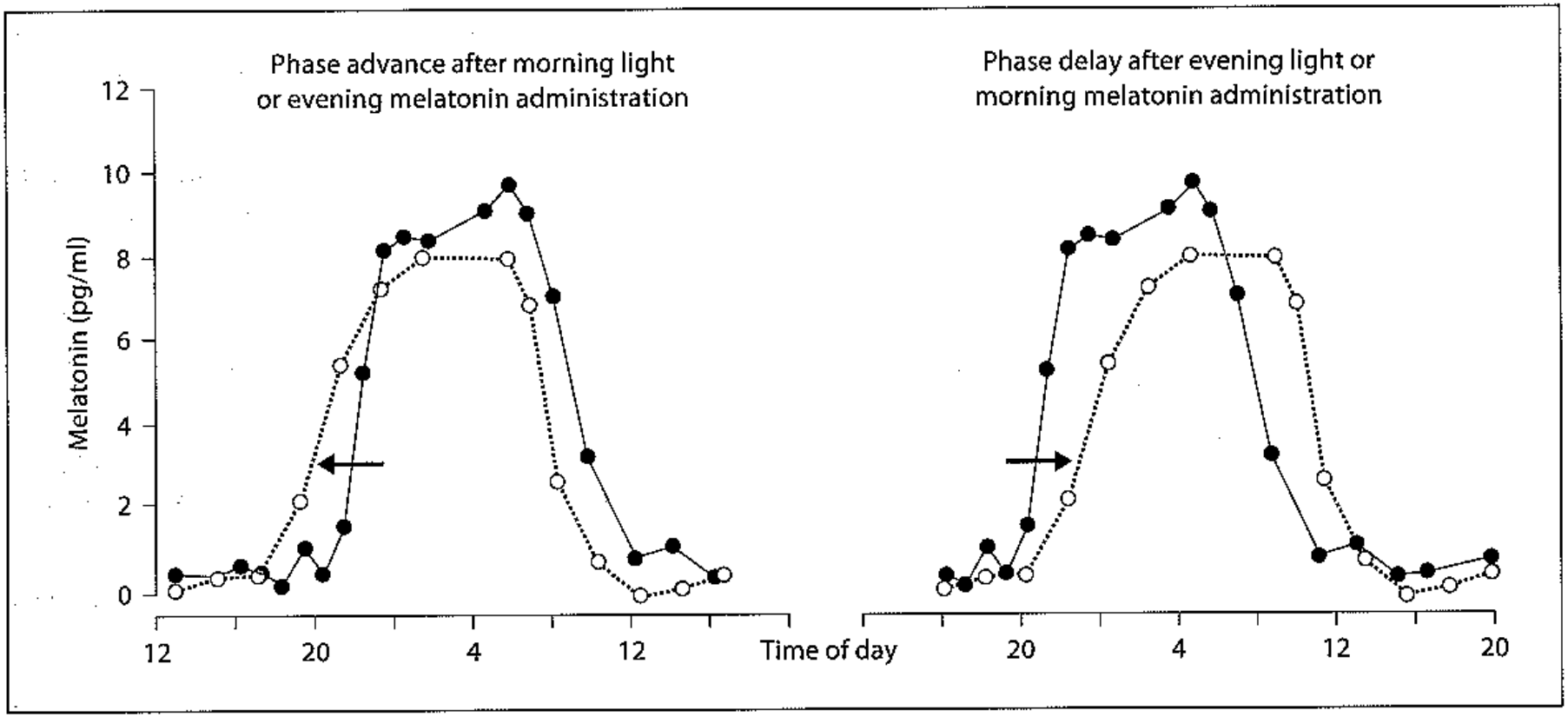
Fava M, McCall WV, Krystal A, Rubens R, Caron J, Wessel T, Amato T, Roth T. Biological Psychiatry. 2007.


♂, 55y
19.45
1.45
7.45
13.45
19.45
TIME OF DAY

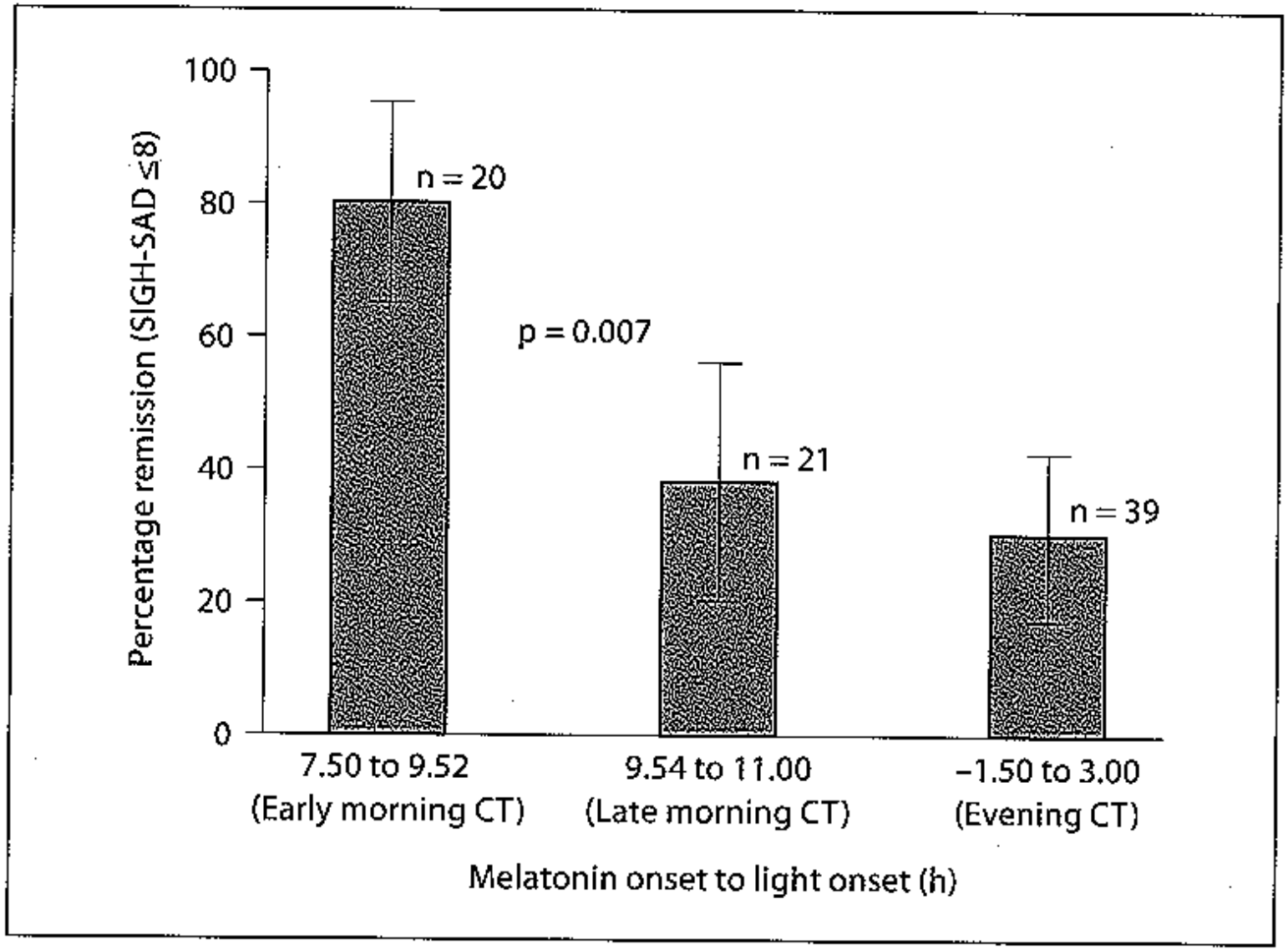
Melatonine


Clock time (h)
8
16
24
8
16
Core body temperature (°C)
37.5
37.0
36.5
Plasma melatonin (pmol/l)
200
100
0
Eye blink rate (n per 30-second epoch)
15
10
5
Slow eye movements (% of 5-min epochs)
16
8
0
Stage 1 sleep (% of 5-min epochs)
3
2
1
0
0
8
16
24
32
Elapsed time awake (h)
Clock time (h)
8
16
24
8
16
Karolinska sleepiness scale (more tired →)
7
5
3
Psychomotor vigilance performance (10% slowest reaction time, ms)
6,000
1,000
500
Cognitive throughput (n attempts)
60
45
30
Probe recall memory test (n correct pairs)
4
2
0
0
8
16
24
32
Elapsed time awake (h)

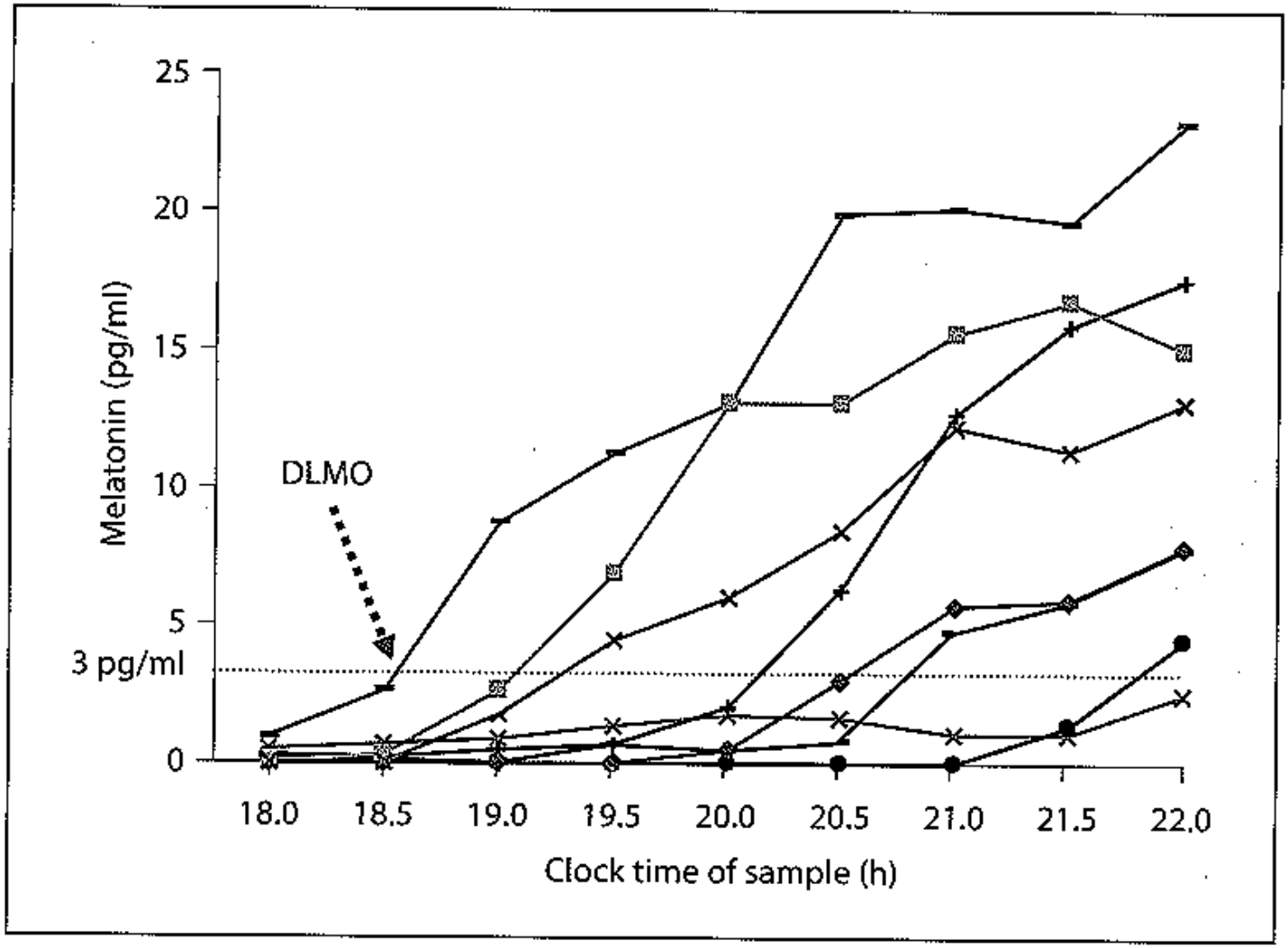
Principes de la chronothérapie



Luminothérapie



Paradigme du DLMO



Morningness-Eveningness Questionnaire (MEQ)

Table 6. Timing of morning light therapy based on MEQ score (beginning of 10,000 lx, 30-min session, approximately 8.5 h after estimated melatonin onset)

MEQ score	Begin light at	MEQ score	Begin light at	MEQ score	Begin light at
16–18	[08:45]	39–41	07:15	62–65	05:45
19–22	[08:30]	42–45	07:00	66–68	05:30
23–26	08:15	46–49	06:45	69–72	05:15
27–30	08:00	50–53	06:30	73–76	05:00
31–34	07:45	54–57	06:15	77–80	[04:45]
35–38	07:30	58–61	06:00	81–84	[04:30]

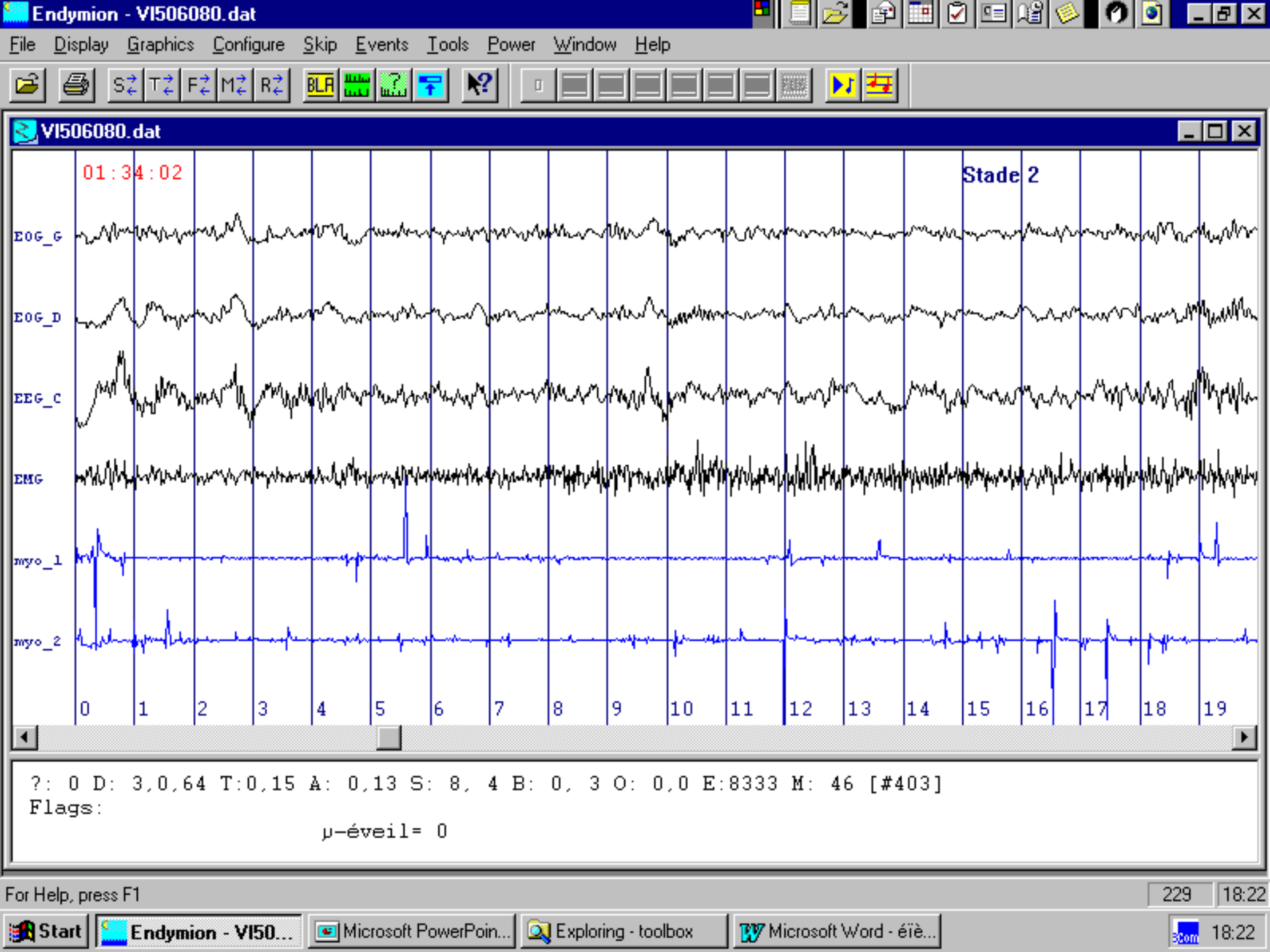
The algorithm has not been clinically evaluated with scores below 23 and above 76 (from Terman and Terman

Demence

Although many attempts with light therapy in these difficult patients have produced mixed results, recent work shows great promise. One 3-week trial has compared morning, evening, and daylong enhanced room light exposure [101]. Both morning and all-day exposure advanced the circadian sleep-wake cycle with increased sleep duration. As expected, evening light delayed the sleep cycle without sleep improvement. The orderliness of results – which was most evident in the most severely demented subgroup – warrants further development of circadian lighting regimens in long-term care facilities.

Cauchemards & Troubles du sommeil paradoxal (REM)

- Cauchemards
  Crise d'épilepsie ? => PSG avec video
  Etat de stress post-traumatique => Prasozine
  ATTENTION prédit le mieux le suicide (surtout chez la femme)
- Pathologie du sommeil REM
  Crise d'épilepsie ? => PSG avec video
  Apnées? => PSG
  Suivi du patient >50% avec Parkinson 10 ans plus tard
  Démence avec Lewy bodies & sensibilité aux neuroleptiques


Endymion - VI506080.dat
File Display Graphics Configure Skip Events Tools Power Window Help
VI506080.dat
01:34:02
Stade 2
EOG_G
EOG_D
EEG_C
EMG
myo_1
myo_2
0 1 2 3 4 5 6 7 8 9 10 11 12 13 14 15 16 17 18 19
?: 0 D: 3,0,64 T:0,15 A: 0,13 S: 8, 4 B: 0, 3 O: 0,0 E:8333 M: 46 [#403]
Flags:
µ-éveil= 0
For Help, press F1
229
18:22
Start
Endymion - VI50...
Microsoft PowerPoin...
Exploring - toolbox
Microsoft Word - éïè...
18:22

PMLS & RLS

- PLMS = 5% des causes d’hypersomnie
- Urémie
- Anémia => Manque de fer
- Grossesse
- Manque d’acide folique
- Diabete
- Arthrite rheumatoide
- ATD surtout les SSRIs
- Etats de manque

Upper Airway Sites Contributing to OSA

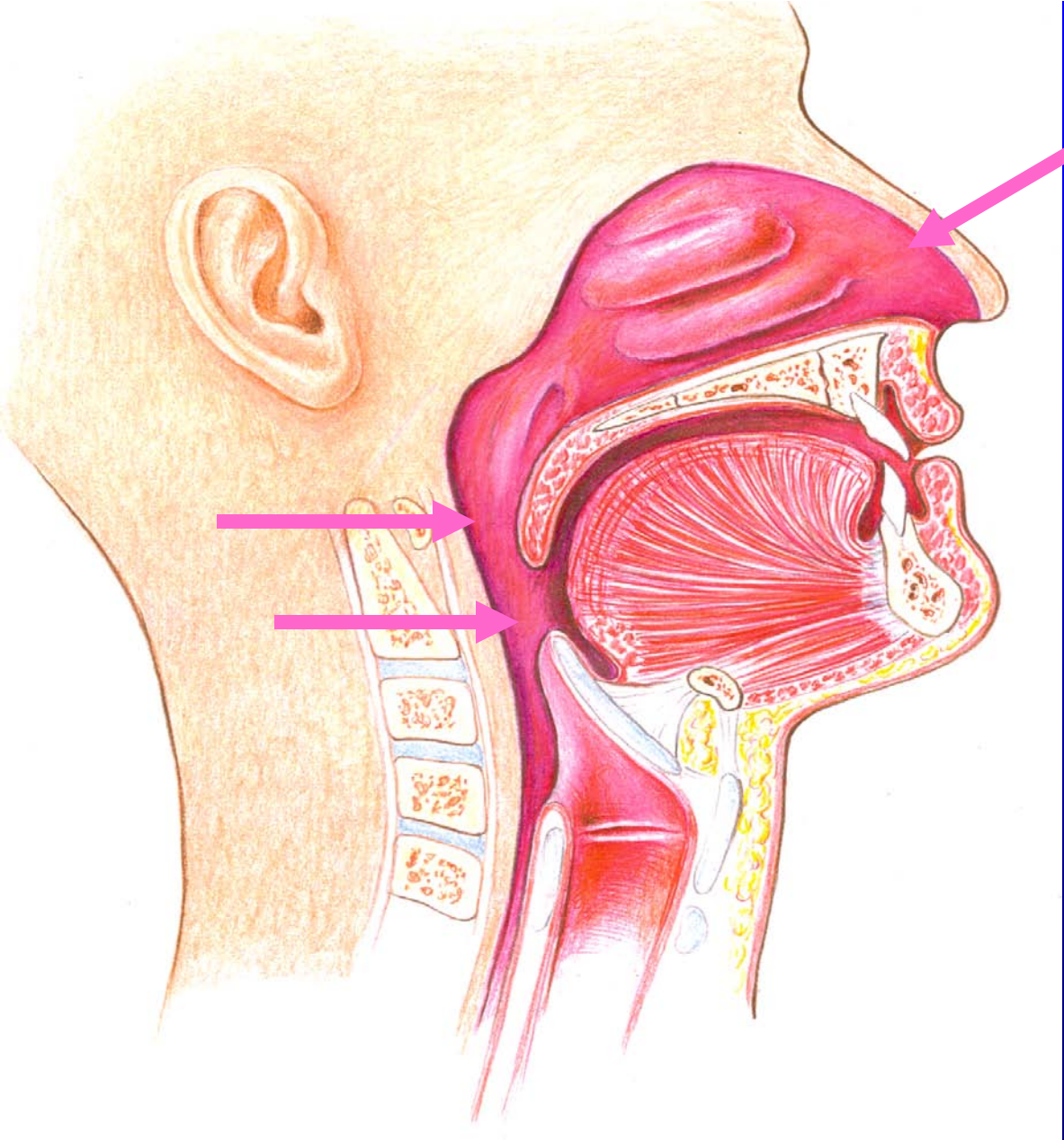


TABLE 1. Prevalence of obstructive sleep apnea from three studies with similar design and methodology

Study Location	n	Age Range (*years*)	Estimated Prevalence of AHI ≥ 5 events/hour (*% [95% CI]*)		Estimated Prevalence of AHI ≥ 15 events/hour (*% [95% CI]*)	
			Men	Women	Men	Women
Wisconsin*	626	30–60	24 (19–28)	9 (6–12)	9 (6–11)	4 (2–7)
Pennsylvania†	1,741	20–99	17 (15–20)	Not given	7 (6–9)	2 (2–3)
Spain‡	400	30–70	26 (20–32)	28 (20–35)	14 (10–18)	7 (3–11)

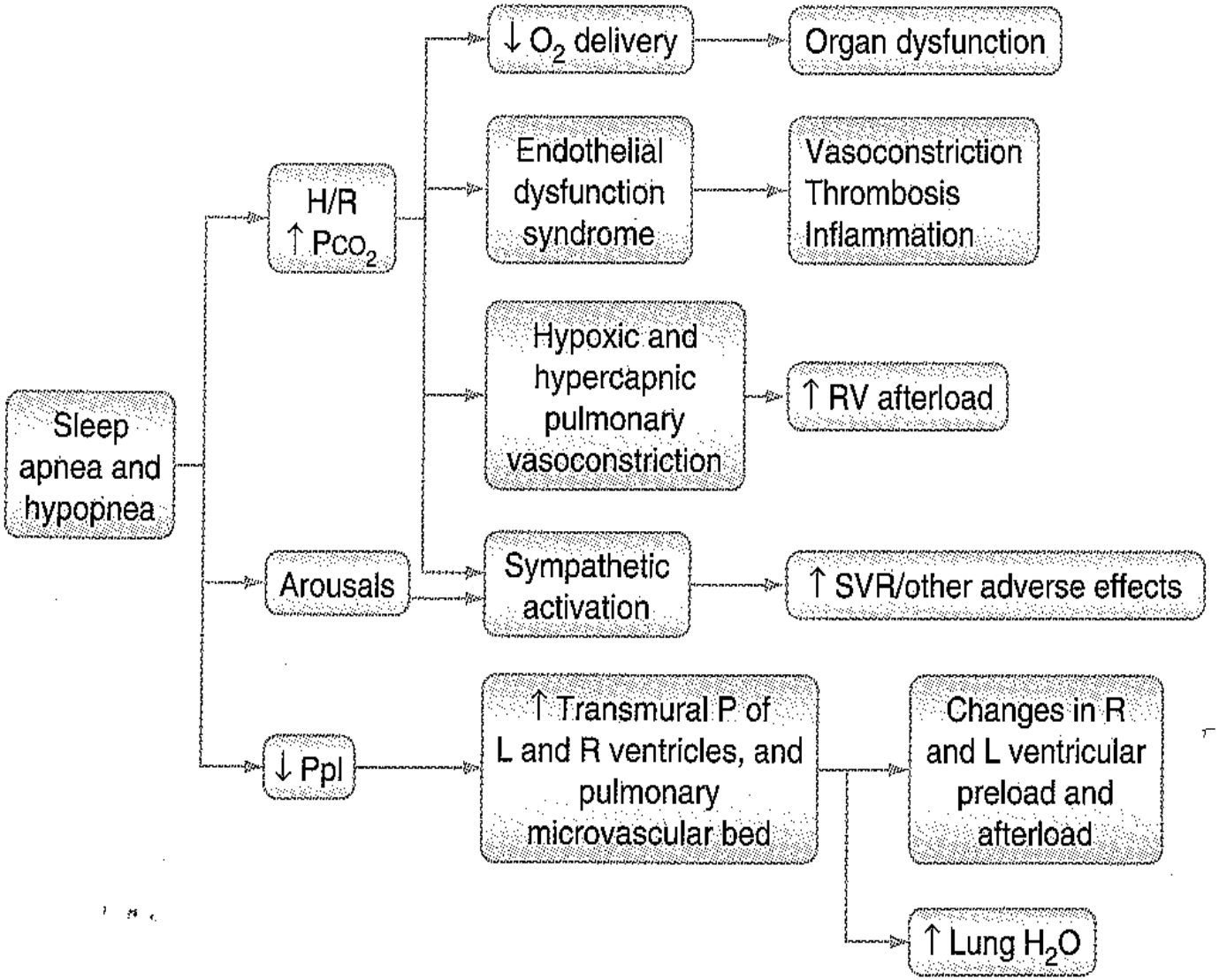
Definition of abbreviation: AHI = apnea–hypopnea index.

* Young and coworkers (11).

† Bixler and coworkers (15, 16).

‡ Durán and coworkers (17).

Cardiovascular Effects of Sleep Apnea



A meta-analysis of clinical screening tests for obstructive sleep apnea. Ramachandran SK, Josephs LA. *Anesthesiology 2009 Apr;110(4):928-39.*

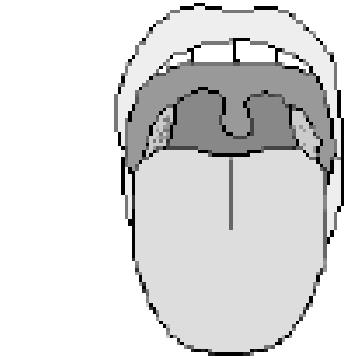
What are the best clinical screening tests for obstructive sleep apnea?

Subjects	*Methods*	*Outcomes*
26 published clinical screening tests for OSA that had been compared to standard overnight PSG. • Questionnaire: 8 • Clinical prediction tests: 18	Meta-analysis. The following were determined for each screening test: (a) diagnostic ORs (ratio of the odds of positivity in persons with OSA relative to the odds of positivity in those without the disease), and (b) false negative rates (frequency of persons with OSA and a negative screening test).	As screening tests for OSA, test elements possessing higher diagnostic accuracy included BMI, history of HTN, and nocturnal choking. The most accurate questionnaires and clinical models were Berlin questionnaire, Sleep Disorders Questionnaire, Kushida morphometry index, and Battagel combined clinical-cephalometry model. However, accuracy of screening tests was variable in repeated validation studies.

BMI: body mass index; HTN: hypertension; OR: odds ratio; OSA: obstructive sleep apnea; PSG: polysomnography

Conclusion Most clinical screening tests, because of false negative rates, missed a significant proportion of persons with obstructive sleep apnea.

Dx of OSA :Mallampati Classification



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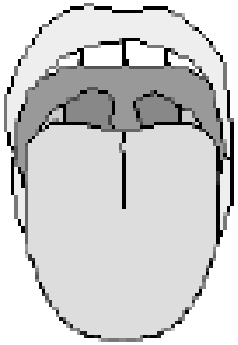

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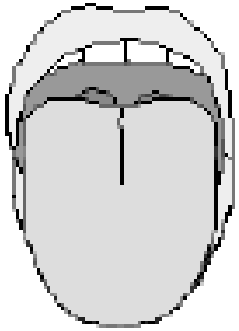

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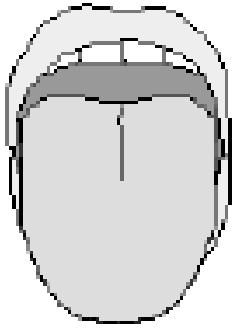

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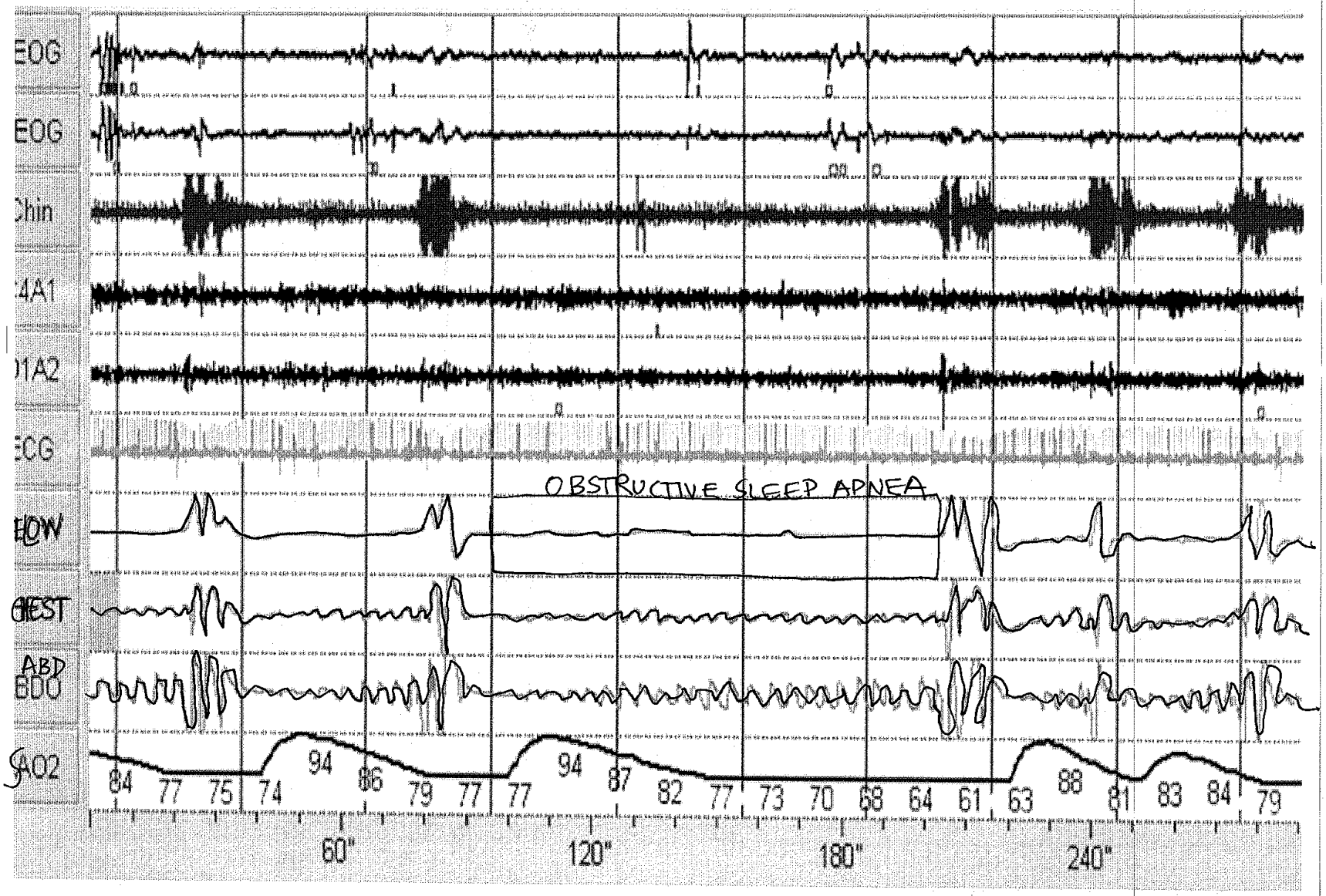
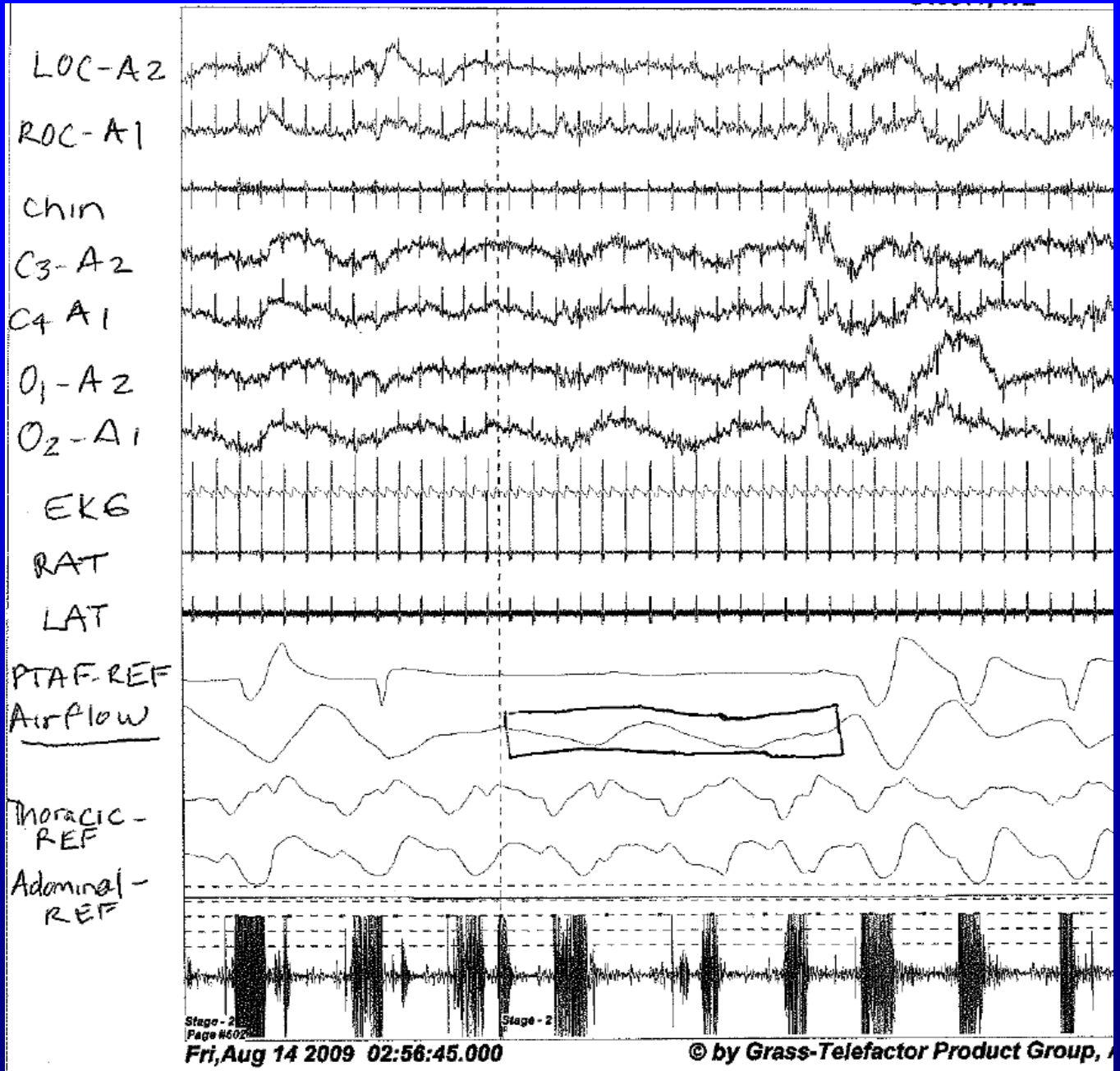

EOG
EOG
Chin
C4A1
O1A2
ECG
FLOW
CHEST
ABD
SAO2
OBSTRUCTIVE SLEEP APNEA
84 77 75 74 94 86 79 77 77 94 87 82 77 73 70 68 64 61 63 88 81 83 84 79
60"
120"
180"
240"


LOC-A2
ROC-A1
Chin
C3-A2
C4 A1
O1-A2
O2-A1
EKG
RAT
LAT
PTAF-REF
Airflow
Thoracic-REF
Adominal-REF
Stage - 2
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Stage - 2
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Diagnosis of obstructive sleep apnea syndrome and its outcomes with home portable monitoring.
Tonelli de Oliveira AC, Martinez D, Vasconcelos LF, Gonçalves SC, Lenz MC, Fuchs SC, Gus M, Abreu-Silva EO, Moreira LB, Fuchs FD. *Chest 2009 Feb;135(2):330-6.*

How does portable respiratory monitoring performed at home compare to either portable respiratory monitoring or polysomnography performed in the laboratory for the diagnosis of obstructive sleep apnea?

Subjects	*Methods*	*Outcomes*
157 subjects with OSA. • Age: 45 ± 12 years • Female: 27% • AHI: 31 ± 29	Subjects with suspected OSA underwent, in random order, attended laboratory PRM + PSG or home-PRM on 2 different nights less than 48 hours apart. PRM assessments included position sensor, pressure transducer, chest belt, nasal cannula and pulse oximeter. Data on 149 laboratory PRM and 121 home PRM were obtained.	For detecting AHI > 5, compared to PSG, laboratory PRM had • Sensitivity: 95.3% • Specificity: 75% • PLR: 3.8 • NLP: 0.11 For detecting AHI > 5, compared to PSG, home PRM had • Sensitivity: 96% • Specificity: 64% • PLR: 2.7 • NLP: 0.05 There was a 16% data-loss rate due to technical problems associated with home-PRM.

AHI: apnea-hypopnea index; NLR: negative likelihood ratio; OSA: obstructive sleep apnea; PLR: positive likelihood ratio; PRM: portable respiratory monitoring; PSG: polysomnography

Conclusion In-home portable respiratory monitoring had comparable diagnostic accuracy for detecting obstructive sleep apnea as polysomnography.

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)

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)

* P=0.005 by the chi-square test for linear trend. AHI denotes apnea–hypopnea index, and CI confidence interval.

Yaggi, H. et al. N Engl J Med 2005;353:2034-2041

The NEW ENGLAND JOURNAL of MEDICINE

Excessive daytime sleepiness is an independent risk indicator for cardiovascular mortality in community-dwelling elderly: the three city study. Empana JP, Dauvilliers Y, Dartigues JF, Ritchie K, Gariepy J, Jouven X, Tzourio C, Amouyel P, Besset A, Ducimetiere P. *Stroke. 2009 Apr;40(4):1219-24. Epub 2009 Feb 26.*

Is excessive daytime sleepiness associated with increased mortality in elderly adults?

Subjects	*Methods*	*Outcomes*
8269 community-dwelling elderly adults. • Female: 60% • Age (mean): 74 ± 5.47 years	Population-based multicenter prospective study (Three City Study) Subjects were asked about the presence of EDS and nighttime sleep disturbance, and medication use for sleep or anxiety, at baseline. Mortality data were collected for over 6 years. *95% CI: 1.13 to 1.61	18.7% of subjects reported the presence of regular (14%) or frequent (4.7%) EDS at baseline. 10.1% used medications for stress/anxiety and 18% used medications for sleep. Subjects with EDS were older, more often depressed or diabetic, had higher BMI, and used more antihypertensive agents. At 6 years follow-up, there were 762 (9.2%) deaths, including cancer (n = 260; 34.1% of deaths) and CVD (n = 196; 25.7% of deaths). Even after adjustment for BMI, CVD disease and cardiovascular risk factors, EDS was associated with a significant increased risk (33%) of total mortality* and cardiovascular mortality, but not cancer mortality.

Continuous positive airway pressure treatment reduces mortality in patients with ischemic stroke and obstructive sleep apnea: a 5-year follow-up study. Martínez-García MA, Soler-Cataluña JJ, Ejarque-Martínez L, Soriano Y, Román-Sánchez P, Illa FB, Canal JM, Durán-Cantolla J. *Am J Respir Crit Care Med 2009 Jul 1;180(1):36-41.*

Does continuous positive airway pressure therapy reduce risk of mortality following ischemic stroke in persons with obstructive sleep apnea?

Subjects	*Methods*	*Outcomes*
166 subjects with ischemic stroke. • AHI < 10 [n = 31]; 10-19 [n = 39]; and ≥ 20 [n = 96] • Mean AHI: 26 • Age: 73.3 ± 11 years • Female: 41% • ESS: 9.1 ± 3.4 Exclusion criteria: • Prior CPAP use • Terminal disease stage • Heart or respiratory failure	Prospective observational study. Sleep studies were performed on all subjects 2 months after the stroke, and CPAP was offered to those with AHI ≥ 20. Subjects were followed for 5 years. Data on mortality were obtained from computer database and official death certificates. CPAP adherence was defined by > 4 hours of use per night for ≥ 70% of days.	Subjects with AHI ≥ 20 and did not tolerate CPAP [n = 68; 48 deaths] had an increase adjusted risk of mortality [HR, 2.69]* compared with subjects with AHI < 20 [n = 70; 26 deaths]. Subjects with AHI ≥ 20 and did not tolerate CPAP also had an increased adjusted risk of mortality [HR, 1.58]**,*** compared with those with AHI ≥ 20 and who tolerated CPAP [n = 28; 12 deaths]. Average CPAP use was 5.9 ± 2.2 hours. No differences in mortality were seen among subjects without OSA, with mild disease, and those who tolerated CPAP.

AHI: apnea-hypopnea index; CPAP: continuous positive airway pressure therapy; ESS: Epworth Sleepiness Scale; HR: hazards ratio; OSA: obstructive sleep apnea; *95% CI, 1.32-5.61, P = 0.009; **95% CI, 1.01-2.49; ***P = 0.04

Conclusion Long-term continuous positive airway pressure therapy in persons with moderate to severe obstructive sleep apnea reduced mortality following ischemic stroke.

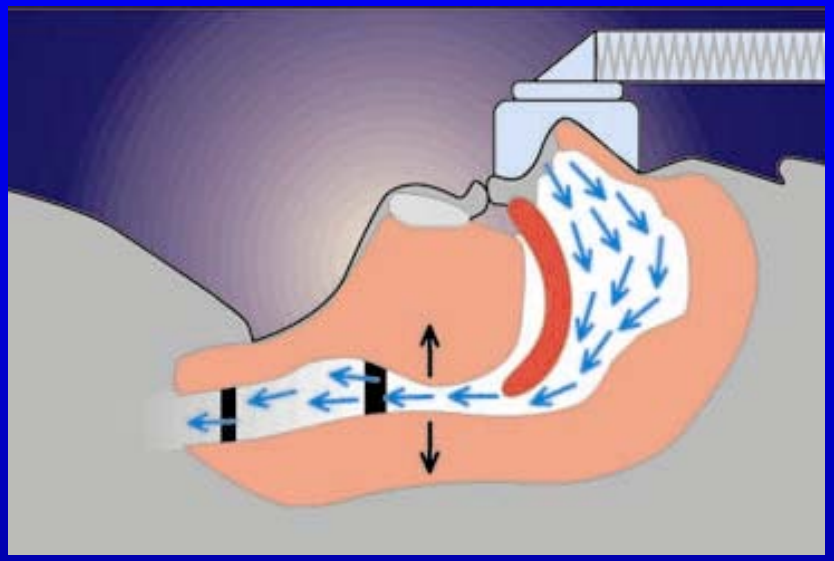
Treatment of OSA with: GOLD STANDARD: CPAP

Treatment

-CPAP treatment

–Positive pressure keeps airway open

–most effective



Clinical and polysomnographic predictors of short-term continuous positive airway pressure compliance. Collen J, Lettieri C, Kelly W, Roop S. *Chest 2009 Mar;135(3):704-9.*

What factors predict short-term compliance with continuous positive airway pressure therapy?

Subjects	*Methods*	*Outcomes*
400 consecutive subjects with newly-diagnosed OSA initiating CPAP therapy. • Female: 22% • Age (mean): 47 ± 8 years • BMI (mean): 30.3 ± 3.7 kg/m^2	Retrospective review of demographic and PSG variables correlated with short-term CPAP compliance after 4-6 weeks of treatment. Good compliance was defined as > 4 hours of use per night in > 70% of nights.	Good compliance was noted in 56.5% of subjects. • Mean nights used: 78.1% • Average use per night: 3.13 hours Better compliance was associated with: • Older age [48 ± 8 vs. 46 ± 7 years]* • One-time use of a sedative-hypnotic during CPAP titration [77% vs. 57.6%]** One-time use of a sedative/hypnotic during CPAP titration was associated with: • Longer sleep time [345 ± 42 vs. 314 ± 51 min] during PSG*** • Greater SE [84 ± 9% vs. 78 ± 11%] during PSG# • Lower RDI on final CPAP pressure [6 ± 7 vs. 10 ± 11]##

CPAP: continuous positive airway pressure; OSA: obstructive sleep apnea; PSG: polysomnography; SE: sleep efficiency; *P = 0.02; **P < 0.0005; ***P < 0.0005; #P < 0.0005; ##P = 0.04

Conclusion One-time use of sedative-hypnotics during polysomnography was associated with greater short-term adherence to therapy using continuous positive airway pressure.

Heart Rate and Blood Pressure

Table 3. Heart Rate and Blood Pressure.*

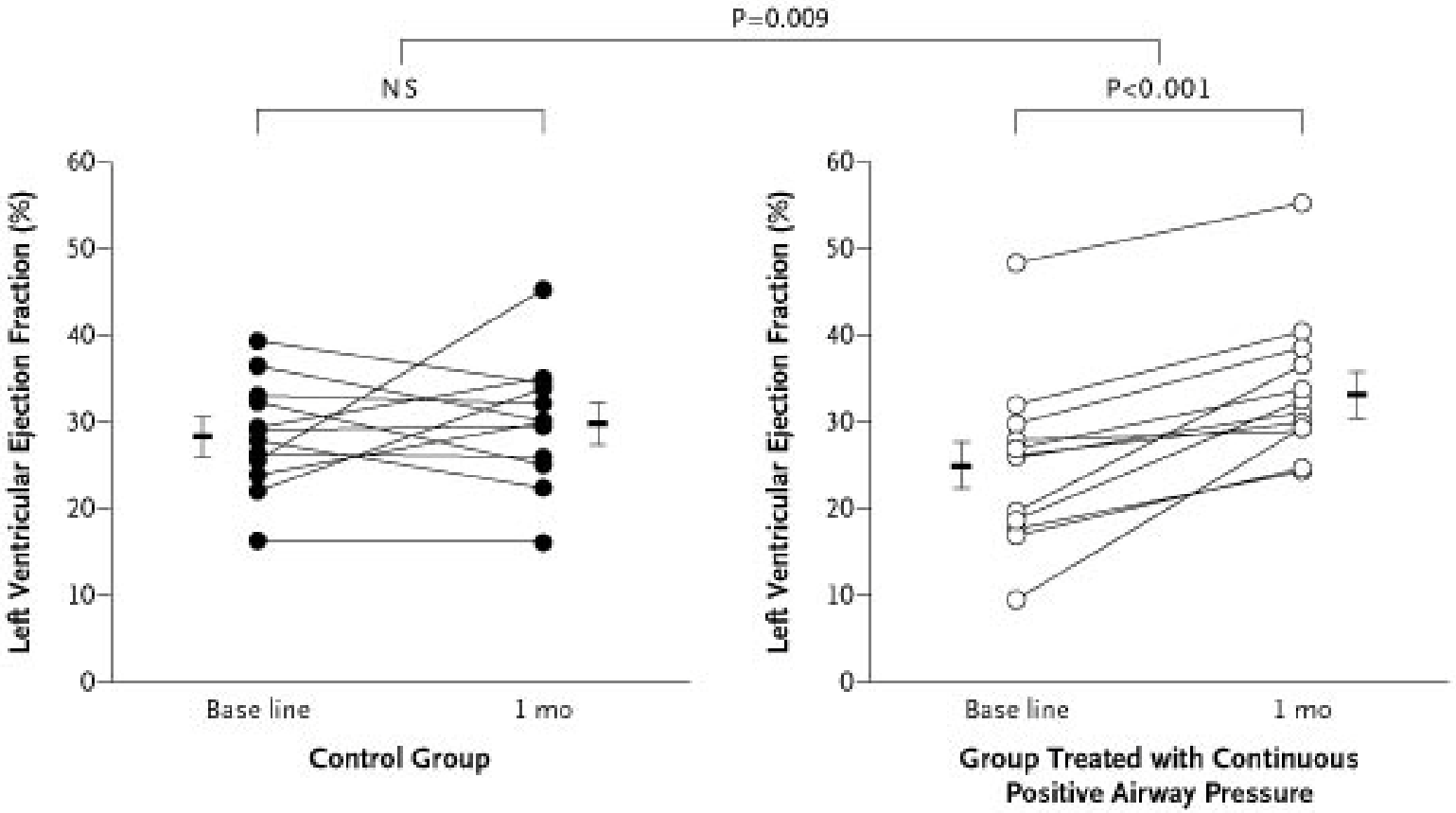
Variable	Control Group			Group Receiving Continuous Positive Airway Pressure		
	Base Line	1 Mo	P Value	Base Line	1 Mo	P Value
Heart rate (beats/min)	67±4	67±4	NS	68±3	64±3	0.007†
Systolic blood pressure (mm Hg)	128±7	134±8	NS	126±6	116±5	0.02‡
Diastolic blood pressure (mm Hg)	60±4	58±3	NS	62±4	59±2	NS

* NS denotes not significant. Plus–minus values are means ±SE. There were no significant differences in base-line values between the control group and the group given continuous positive airway pressure. Unless otherwise specified, P values are for the comparisons between base-line values and one-month values within the group.
† P=0.09 for the comparison between the groups.
‡ P=0.008 for the comparison between the groups.

Kaneko, Y. et al. N Engl J Med 2003;348:1233-1241

The NEW ENGLAND JOURNAL of MEDICINE

Individual Values for the Left Ventricular Ejection Fraction in All Patients



Kaneko, Y. et al. N Engl J Med 2003;348:1233-1241

In-hospital treatment of obstructive sleep apnea during decompensation of heart failure. Khayat RN, Abraham WT, Patt B, Pu M, Jarjoura D. *Chest 2009 Oct;136(4):991-7.*

Is there any benefit on cardiac outcomes of immediate in-patient diagnosis and treatment of obstructive sleep apnea in persons hospitalized for acutely decompensated heart failure?

Subjects	*Methods*	*Outcomes*
46 consecutive persons with ADHF and OSA. • AHI: ≥ 15 • Obstructive apnea index: ≥ 5 • ADHF: dyspnea, LVEF ≤ 45% and elevated LV pressure Exclusion criteria • Current PAP use for SDB • CSA • Hemodynamic instability • Respiratory insufficiency • Renal failure	Randomized controlled trial. Subjects underwent attended PSG sleep study within 2 days of hospital admission, and were randomized to either (a) *intervention* with in-hospital APAP treatment of OSA and standard therapy of ADHF [n = 23], or (b) *control* with only standard treatment of ADHF [n = 23]. Assessment included change in LVEF by echocardiography 3 nights post-randomization.	After 3 nights of randomization, improvement in LVEF was greater with intervention compared to control [4.6% difference in improvement; 4.5%* vs. -0.3%**]*** Changes in both LV end-systolic and end-diastolic volumes as well as weight loss were greater in the intervention group. Mean duration of APAP use in the intervention group was 3.2 ± 0.5 hours nightly.

ADHF: acutely decompensated heart failure; AHI: apnea-hypopnea index; APAP: auto-adjusting positive airway pressure; LV: left ventricle; LVEF: left ventricular ejection fraction; OSA: obstructive sleep apnea; PAP" positive airway pressure; PSG: polysomnography; SDB: sleep disordered breathing; *SE, 1.7%; **SE, 1.5%; ***P = 0.03

Effects of nocturnal continuous positive airway pressure therapy in patients with resistant hypertension and obstructive sleep apnea. Dernaika TA, Kinasewitz GT, Tawk MM. *J Clin Sleep Med. 2009 Apr 15;5(2):103-7.*

What are the long-term effects on blood pressure of continuous positive airway pressure therapy in hypertensive persons with obstructive sleep apnea?

Subjects	*Methods*	*Outcomes*
98 persons with OSA (AHI ≥ 5) and HTN. • Resistant HTN [n = 42] • Controlled hypertension [n = 56] Resistant HTN was defined as daytime BP of ≥ 140/90 mm Hg despite the use of ≥ 3 antihypertensive medications.	Retrospective chart review. BP was measured every 3 months after CPAP initiation for 1 year.	At the end of the follow-up period, mean difference in MAP was: • Resistant HTN: - 5.6* • Controlled HTN: - 0.8 mm Hg** De-escalation of antihypertensive treatment was noted after CPAP therapy in 71% of resistant HTN group but not in the controlled HTN group. Factors associated with decrease in MAP by 10% after 12 months of CPAP therapy included: • Baseline BP*** • Diuretic therapy# Neither baseline AHI or hours of CPAP use were associated with decrease in MAP.

AHI: apnea-hypopnea index; BP: blood pressure; CPAP: continuous positive airway pressure; HTN: hypertension; MAP: mean arterial pressure; OSA: obstructive sleep apnea; *95% CI -2.0 to -8.7 mm Hg; p = 0.03; **95% CI -2.9 to 3.3 mm Hg; p = 0.53; ***OR 5.4, 95% CI 2.3 to 8.9; p = 0.01; #OR 3.2, 95% CI 1.8 to 6.1; p = 0.02

Sustained use of CPAP slows deterioration of cognition, sleep, and mood in patients with Alzheimer's disease and obstructive sleep apnea: a preliminary study. Cooke JR, Ayalon L, Palmer BW, Loredo JS, Corey-Bloom J, Natarajan L, Liu L, Ancoli-Lsrael S. *J Clin Sleep Med 2009 Aug 15;5(4):305-9.*

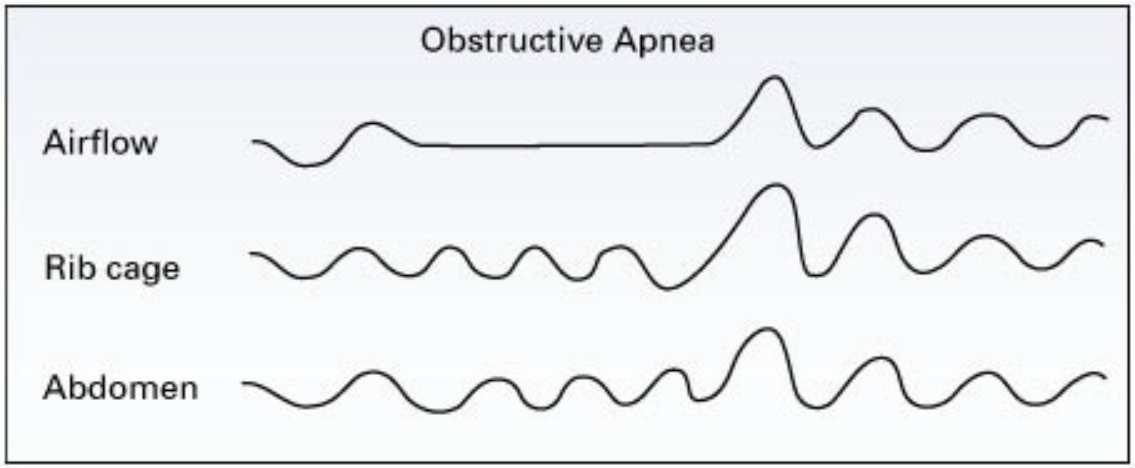
What are the long-term effects on cognition of continuous positive airway pressure therapy of obstructive sleep apnea in persons with Alzheimer's disease?

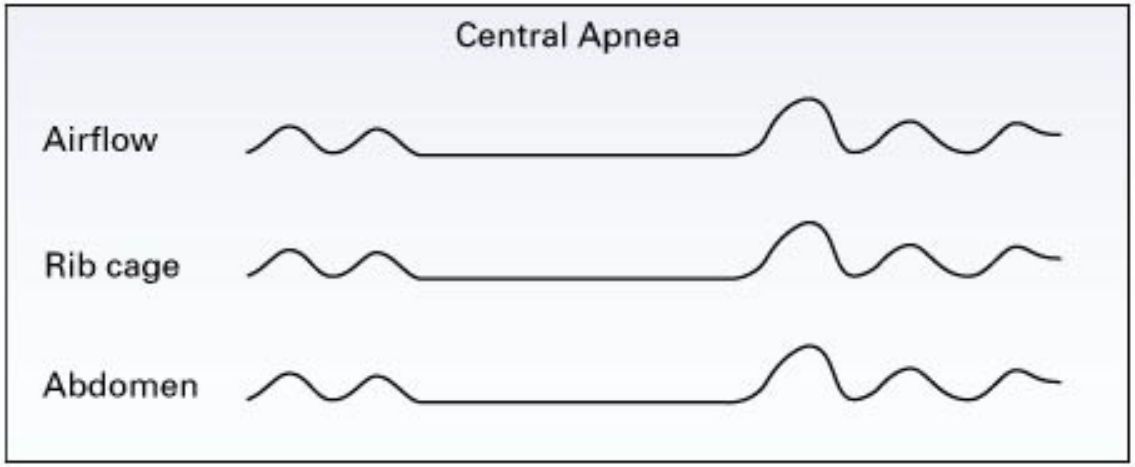
Subjects	*Methods*	*Outcomes*
10 persons with OSA and mild to moderate AD who were initially enrolled in a 6-week RCT of CPAP therapy. 5 subjects continued CPAP and 5 subjects discontinued CPAP. • Female: 30% • AHI: ≥ 10 • MMSE score: ≥ 18 9 caregivers.	Subjects were followed for a mean of 13.3 ± 5.2 months, and assessments included PSG (for CPAP users), PSQI, ESS, FOSQ, CSD, NPI and neuropsychological test battery.	Compared to non-CPAP use, sustained CPAP use was associated with: • Less cognitive decline • Stable depressive symptoms (CSD) and daytime somnolence (ESS) • Improved subjective sleep quality (PSQI) • Improved psychopathological behavior (NPI) • Improved sleep of caregivers (PSQI)

AD: Alzheimer's disease; AHI: apnea-hypopnea index; CPAP: continuous positive airway pressure; CSD: Cornell scale for depression in dementia; ESS: Epworth sleepiness scale; FOSQ: Functional outcomes of sleep questionnaire; MMSE: Mini mental status examination; NPI: Neuropsychiatric inventory; PSQI: Pittsburg Sleep Quality Index; OSA: obstructive sleep apnea; RCT: randomized clinical trial

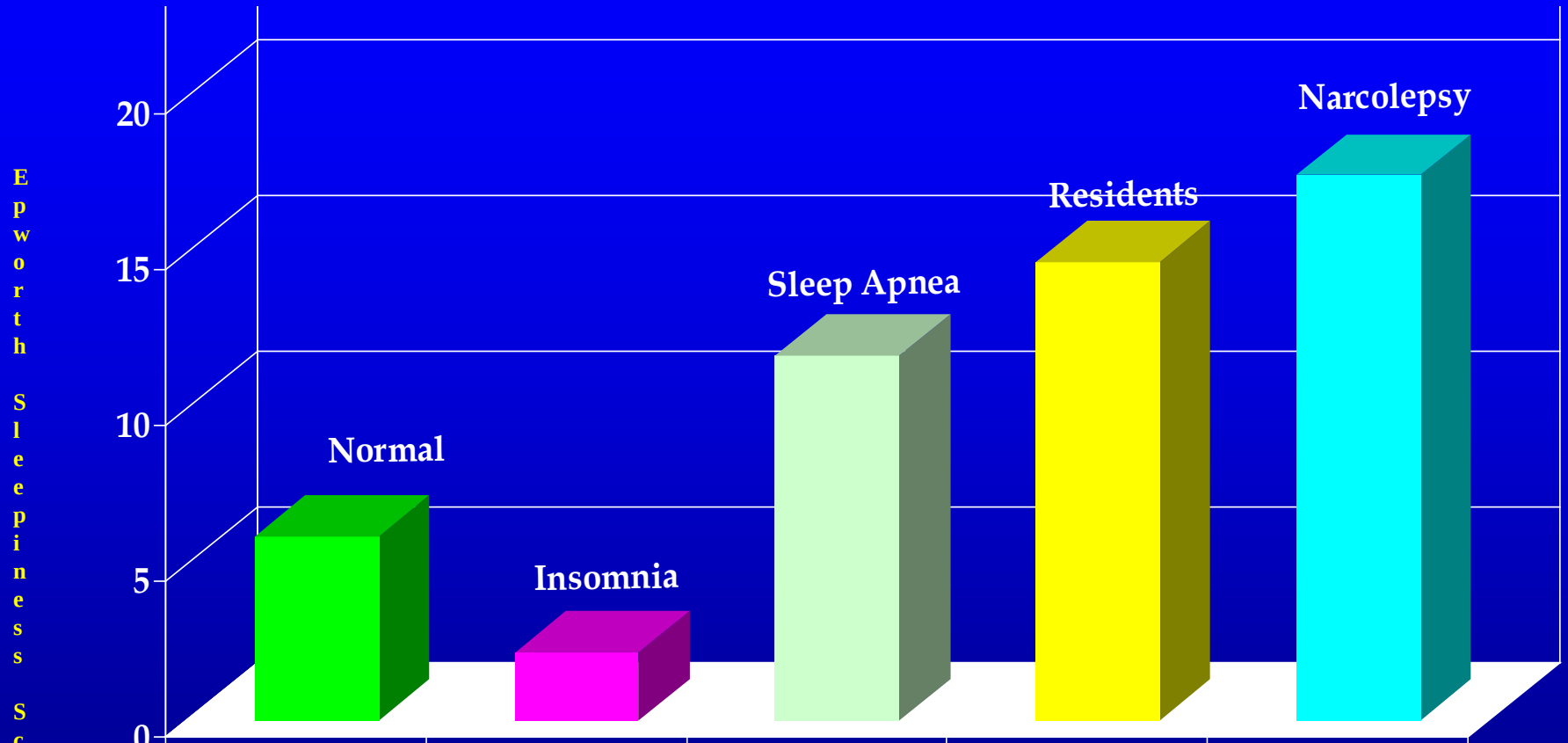
Conclusion Long-term use of continuous positive airway pressure for obstructive sleep apnea produced lasting improvements in mood and slowed cognitive decline in persons with Alzheimer's disease.

PSG of OSA vs. CSA



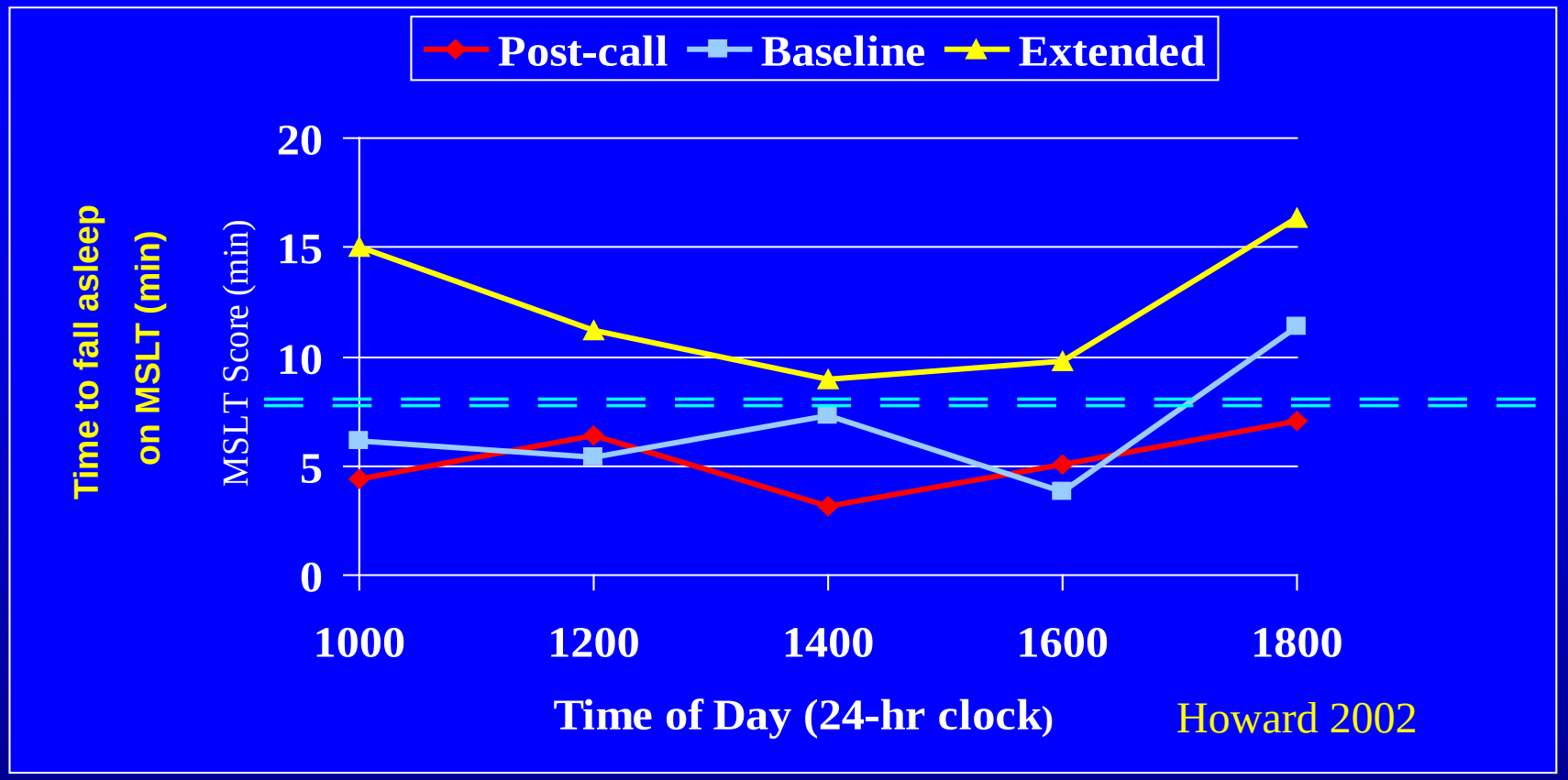


L’ échelle de somnolence de Epworth



	Normal	Insomnia	Sleep Apnea	Residents	Narcolepsy
Mean	5.90	2.20	11.70	14.70	17.50

Papp et al. *Sleep*, 2002.



Sleepiness level post-call ◆ vs on a normal (baseline) schedule ■ was equivalent in anesthesia residents. A period of extended sleep ▲ (over 4 nights) normalized post-call sleepiness levels.

A quels aspects du sommeil faut-il prêter le plus d'attention lors de la prise en charge du patient psychiatrique ?

- 1) Durée *(n'aggravons pas ce qui est déjà fait)*
- 2) Qualité *(insomnie <=> dépression)*
- 3) Régularité *(aspects circadiens & luminothérapie)*
- 4) Cauchemards / troubles REM *(suicide – Parkinson^{+})*
- 6) Acathisie *(RLS – Fer – Dopamine – PLMS - SSRIs)*
- 7) Apnées *(parce que nous avons une approche hollistique du patient psychiatrique)*
- 1, 2, 3 => clinique
- 4, 5, 6 => labo du sommeil
- 7 => labo du sommeil ou maison

Un grand merci aux Pr.

- D. Saravane
- A Krystal
- P Linkowski

& Mme. Mazzia