ADJUVANT AND ALTERNATIVE TREATMENTS TO CBT-I

POSSIBLE ADJUVENTS AND ALTERNATIVES

BRIGHT LIGHT THERAPY

SLEEP COMPRESSION

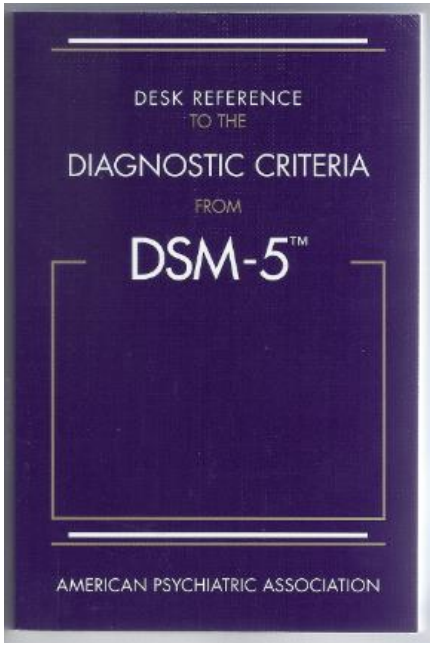
COUNTER CONTROL

ISR

MINDFULNESS

RATIONALE

The use of bright lights in the treatment of insomnia is based on the idea that the various subtypes of the sleep disturbance may be at least partially due to a circadian dysrhythmia.



Circadian Rhythm Sleep-Wake Disorders

A. A persistent or recurrent pattern of sleep disruption that is primarily due to an alteration of the circadian system or to a misalignment between the endogenous circadian rhythm and the sleep-wake schedule required by an individual's physical environment or social or professional schedule.

B. The sleep disruption leads to excessive sleepiness or insomnia, or both.

C. The sleep disturbance causes clinically significant distress or impairment in social, occupational, and other important areas of functioning.

Coding note: For ICD-9-CM, code **307.45** for all subtypes. For ICD-10-CM, code is based on subtype.

Specify whether:

307.45 (G47.21) Delayed sleep phase type: A pattern of delayed sleep onset and awakening times, with an inability to fall asleep and awaken at a desired or conventionally acceptable earlier time.

Specify if:

Familial: A family history of delayed sleep phase is present.

Specify if:

Overlapping with non-24-hour sleep-wake type: Delayed sleep phase type may overlap with another circadian rhythm sleep-wake disorder, non-24-hour sleep-wake type.

307.45 (G47.22) Advanced sleep phase type: A pattern of advanced sleep onset and awakening times, with an inability to remain awake or asleep until the desired or conventionally acceptable later sleep or wake times.

Specify if:

Familial: A family history of advanced sleep phase is present.

307.45 (G47.23) Irregular sleep-wake type: A temporally disorganized sleep-wake pattern, such that the timing of sleep and wake periods is variable throughout the 24-hour period.

307.45 (G47.24) Non-24-hour sleep-wake type: A pattern of sleep-wake cycles that is not synchronized to the 24-hour environment, with a consistent daily drift (usually to later and later times) of sleep onset and wake times.

307.45 (G47.26) Shift work type: Insomnia during the major sleep period and/or excessive sleepiness (including inadvertent sleep) during the major awake period associated with a shift work schedule (i.e., requiring unconventional work hours).

307.45 (G47.20) Unspecified type

Specify if:

Episodic: Symptoms last at least 1 month but less than 3 months.

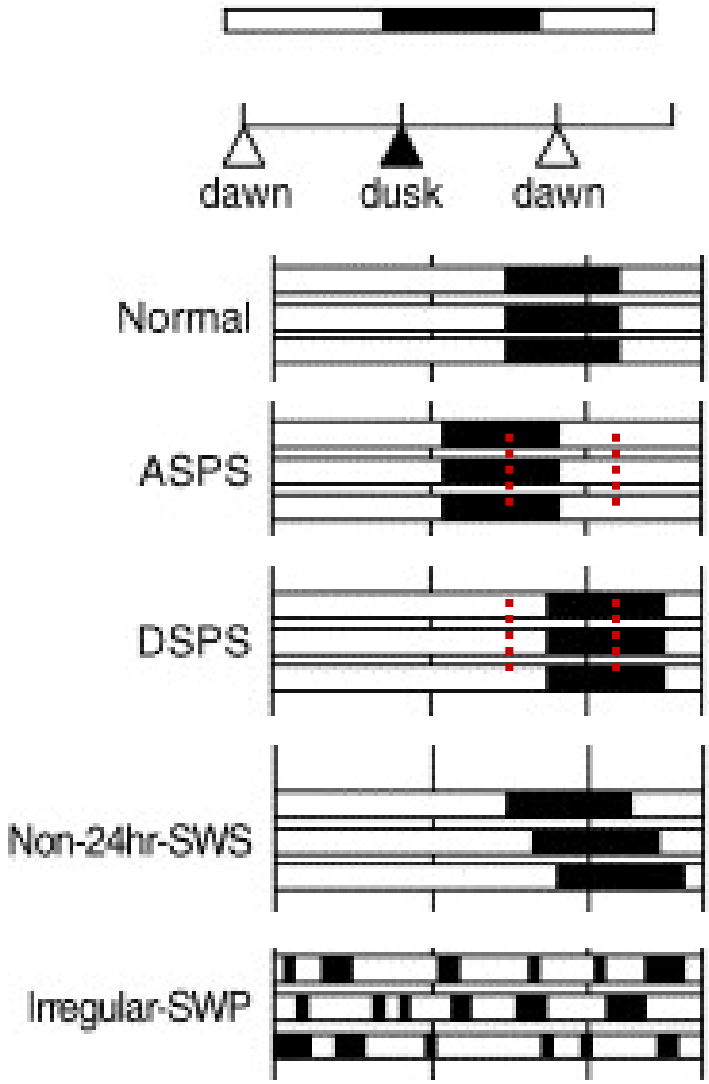
Persistent: Symptoms last 3 months or longer.

Recurrent: Two or more episodes occur within the space of 1 year.

CIRCADIAN RHYTHM DISORDERS

SIGNS AND SYMPTOMS

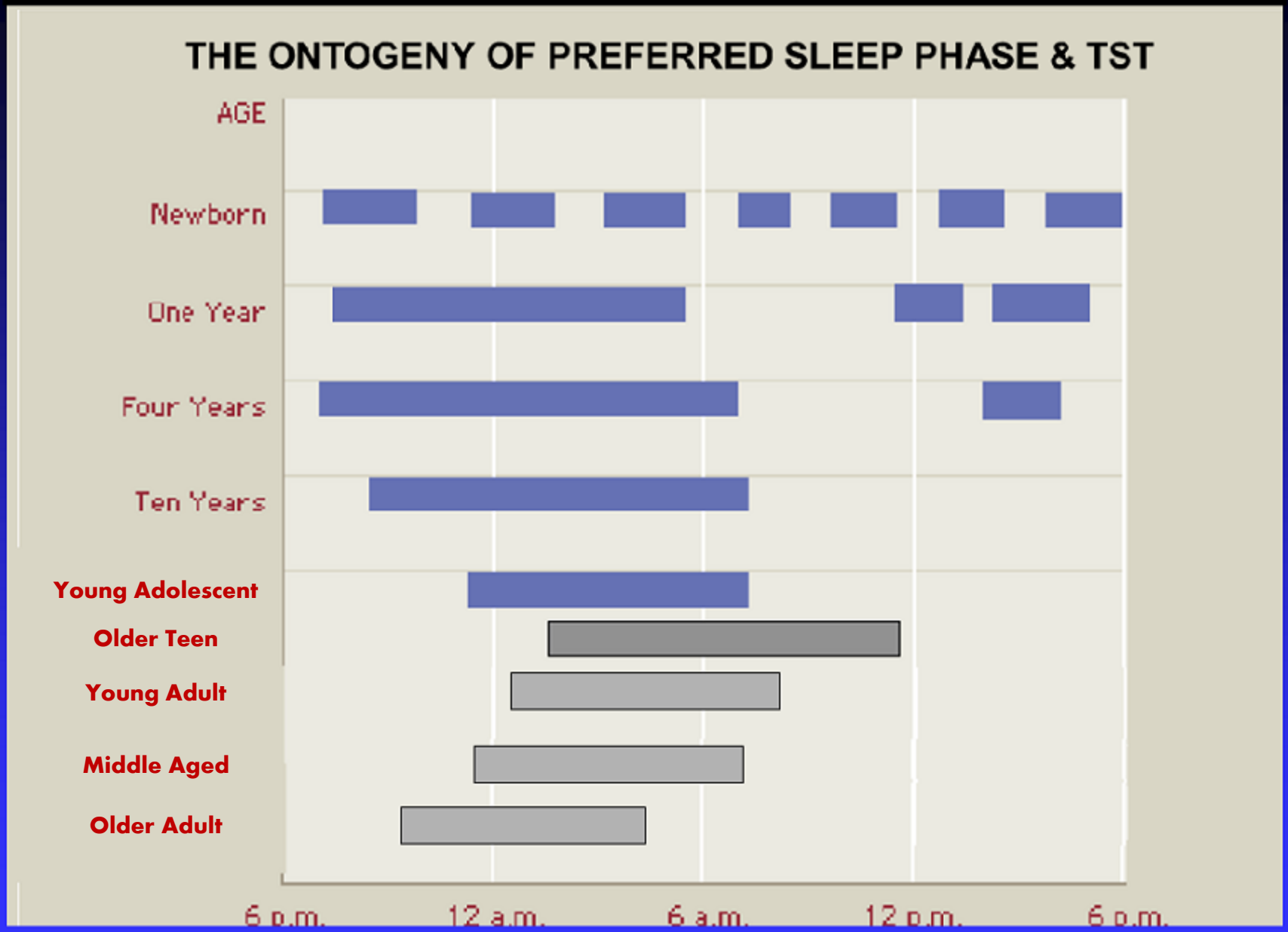
- COMPLAINT OF SLEEP ONSET/OFFSET INSOMNIA
  - DISCREPANT SLEEP SCHEDULES
  - NORMAL SLEEP WHEN SCHEDULE IS AD LIBITUM
  - AGE


dawn
dusk
dawn
Normal
ASPS
DSPS
Non-24hr-SWS
Irregular-SWP

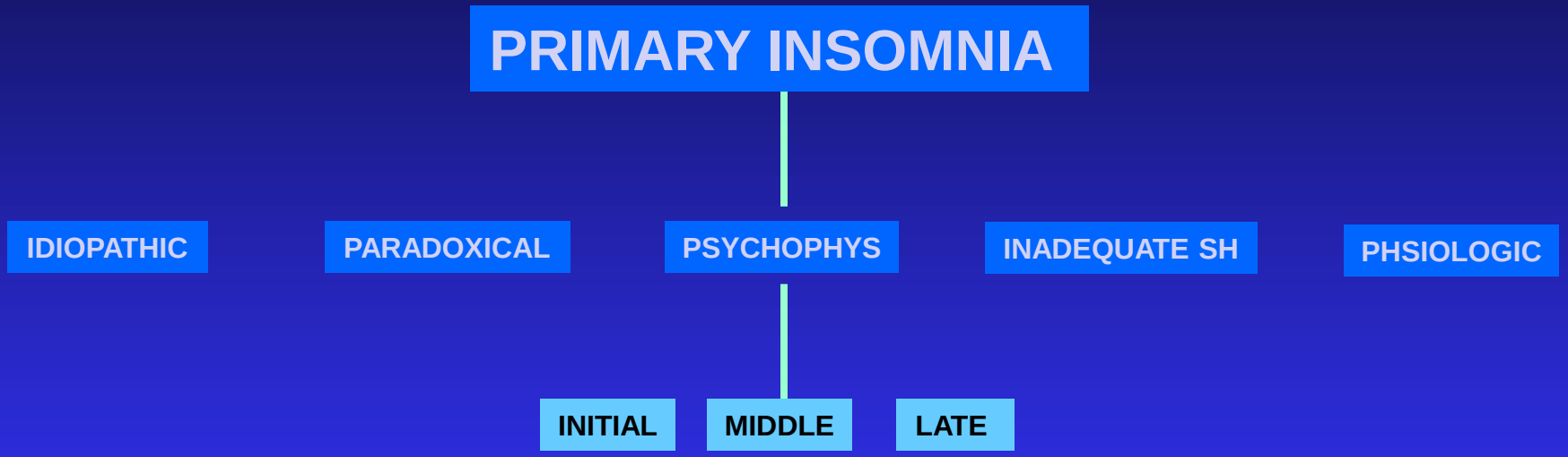
CIRCADIAN RHYTHM DISORDERS

SIGNS AND SYMPTOMS

- COMPLAINT OF SLEEP ONSET/OFFSET INSOMNIA
  - DISCREPANT SLEEP SCHEDULES
  - NORMAL SLEEP WHEN SCHEDULE IS AD LIBITUM
  - AGE


THE ONTOGENY OF PREFERRED SLEEP PHASE & TST
AGE
Newborn
One Year
Four Years
Ten Years
Young Adolescent
Older Teen
Young Adult
Middle Aged
Older Adult
6 p.m.
12 a.m.
6 a.m.
12 p.m.
6 p.m.

SUBTYPES OF INSOMNIA



WHICH SUBTYPE(S) MIGHT BE ASSOCIATED WITH CIRCADIAN DYSRHYTHMIA?

INITIAL INSOMNIA	→	DSPS
MIDDLE INSOMNIA	→	?
LATE INSOMNIA	→	ASPS

DSPS/ASPS
TREATMENT

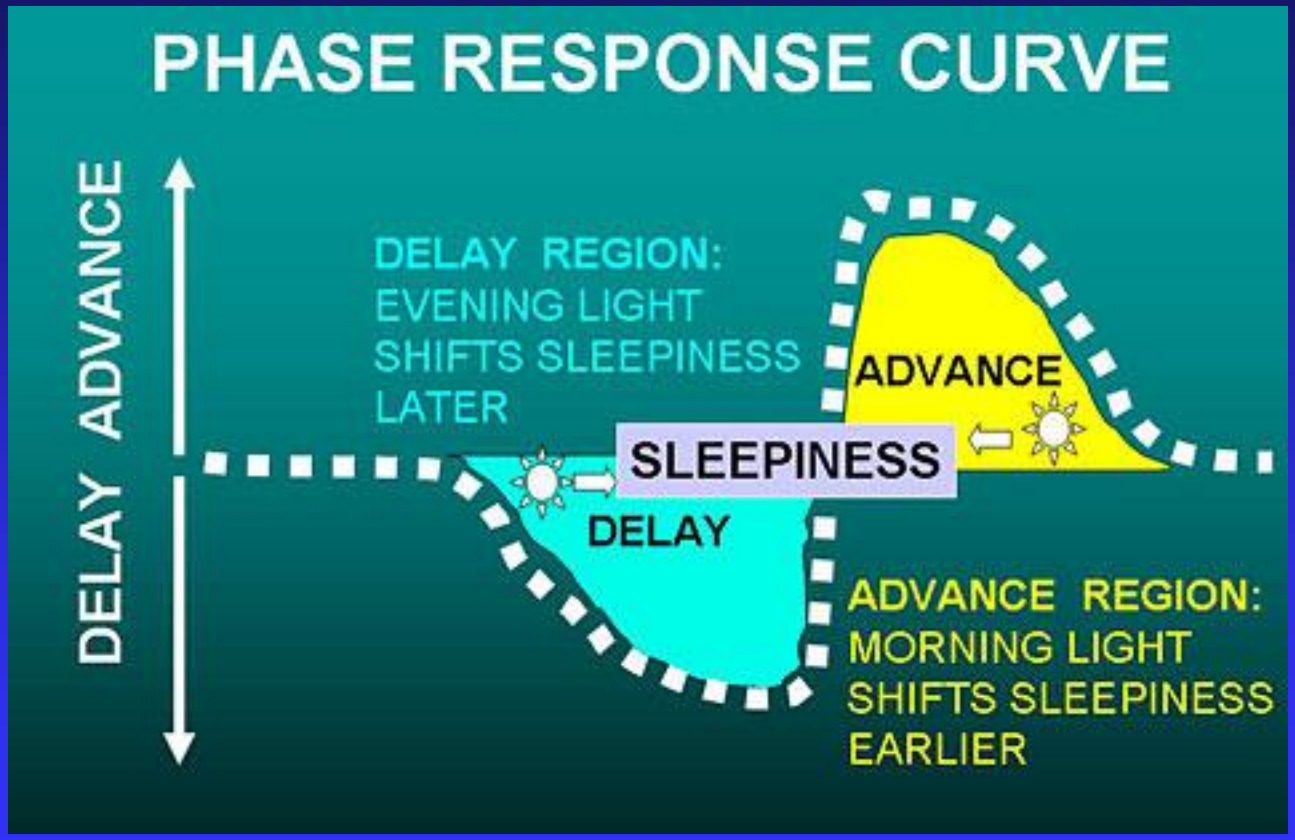
PHARMACOLOGIC / MEDICAL

- HYPNOTICS
- CHRONOBIOTICS

BEHAVIORAL

- CHRONOTHERAPY
- BRIGHT LIGHT

TRICKING THE BRAIN



WHAT THEY DO IN THE LAB

Throughout the 26-hour constant routines, rectal temperature was measured at 1-minute intervals using an indwelling Yellow Springs Instruments (Yellow Springs, Ohio) Series 400 temperature thermistor, inserted 10 to 15 cm into the rectum.

At approximately 7:30 AM, a rectal thermistor was inserted, and the 26-hour constant routine commenced at 8:00 AM until 10:00 AM the next morning. During the constant routine, participants remained in the near supine position, awake and engaging in minimal activity such as reading, listening to music, or watching video films. The ambient temperature was kept constant at 22°C, and room illumination was kept at less than 50 lux, as measured at the participant's head position. Snacks of approximate equal caloric value and a 200-ml glass of water or diluted juice were given at 2-hour intervals. Urine was collected every 2 hours. At 10:00 AM the following morning, subjects removed the thermistor and returned home. They were advised to remain active and try to stay awake as long as possible once at home in order to maintain their usual sleeping pattern.

LACK, et. al.., SLEEP 2005; 28(5): 616-623

WHAT IS SO IMPORTANT ABOUT FINDING T-MIN?

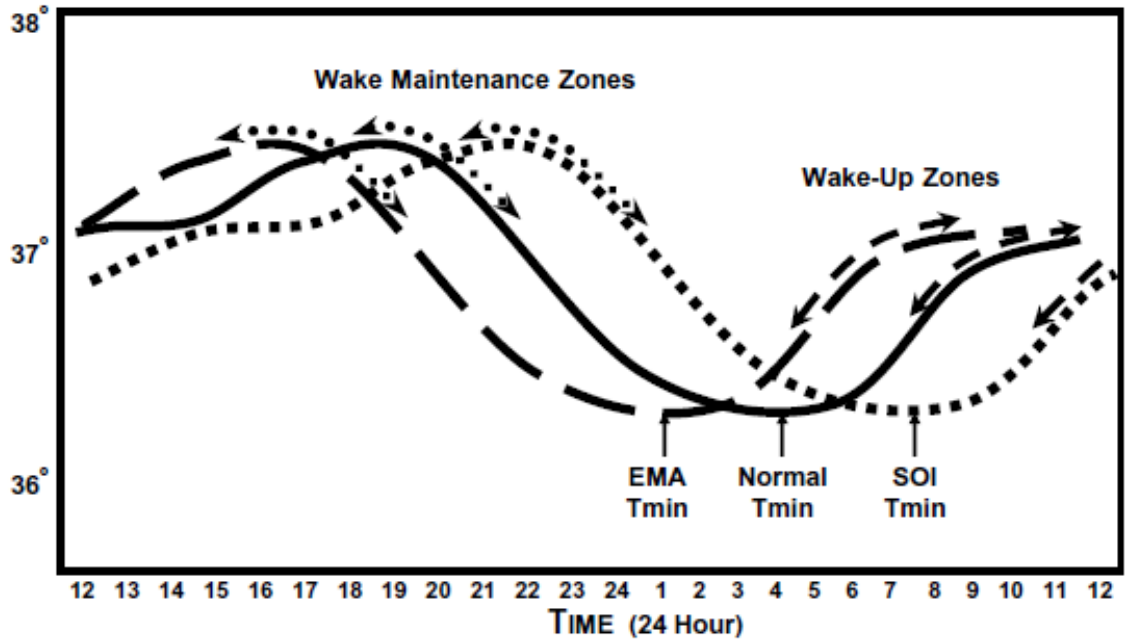
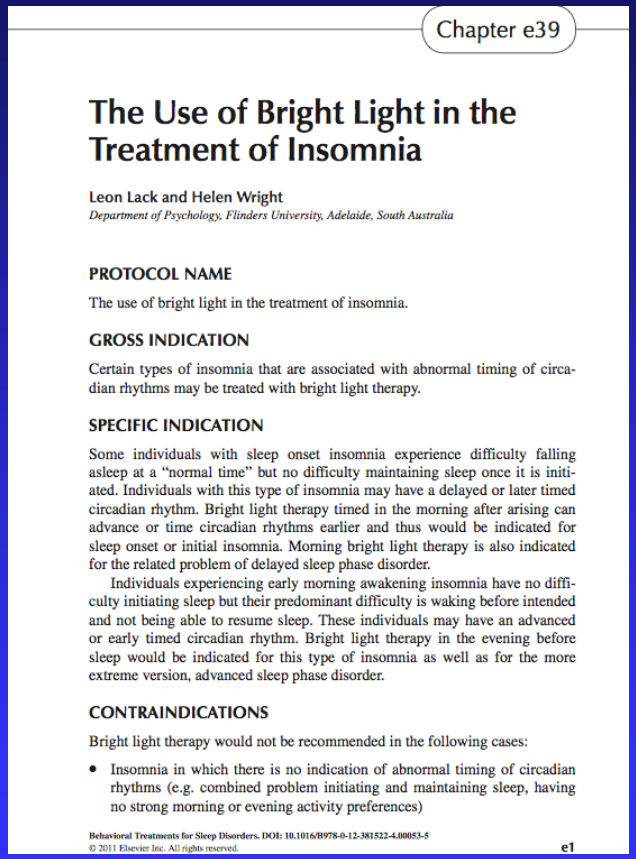


Fig. 1. Diagramatic representation of the circadian core body temperature rhythms of normally entrained good sleepers (solid line) and those of sleep onset insomniacs (SOI) (dotted line) and early morning awakening (EMA) insomniacs (dashed line) showing the wake maintenance zone (thick dotted line) and wake-up zones (thick dashed line).

STEPS FOR SLEEP ONSET INSOMNIA/DSPS TX



Chapter e39

The Use of Bright Light in the Treatment of Insomnia

Leon Lack and Helen Wright
Department of Psychology, Flinders University, Adelaide, South Australia

PROTOCOL NAME

The use of bright light in the treatment of insomnia.

GROSS INDICATION

Certain types of insomnia that are associated with abnormal timing of circadian rhythms may be treated with bright light therapy.

SPECIFIC INDICATION

Some individuals with sleep onset insomnia experience difficulty falling asleep at a "normal time" but no difficulty maintaining sleep once it is initiated. Individuals with this type of insomnia may have a delayed or later timed circadian rhythm. Bright light therapy timed in the morning after arising can advance or time circadian rhythms earlier and thus would be indicated for sleep onset or initial insomnia. Morning bright light therapy is also indicated for the related problem of delayed sleep phase disorder.

Individuals experiencing early morning awakening insomnia have no difficulty initiating sleep but their predominant difficulty is waking before intended and not being able to resume sleep. These individuals may have an advanced or early timed circadian rhythm. Bright light therapy in the evening before sleep would be indicated for this type of insomnia as well as for the more extreme version, advanced sleep phase disorder.

CONTRAINDICATIONS

Bright light therapy would not be recommended in the following cases:

- Insomnia in which there is no indication of abnormal timing of circadian rhythms (e.g. combined problem initiating and maintaining sleep, having no strong morning or evening activity preferences)

Behavioral Treatments for Sleep Disorders. DOI: 10.1016/B978-0-12-381522-4.00053-5
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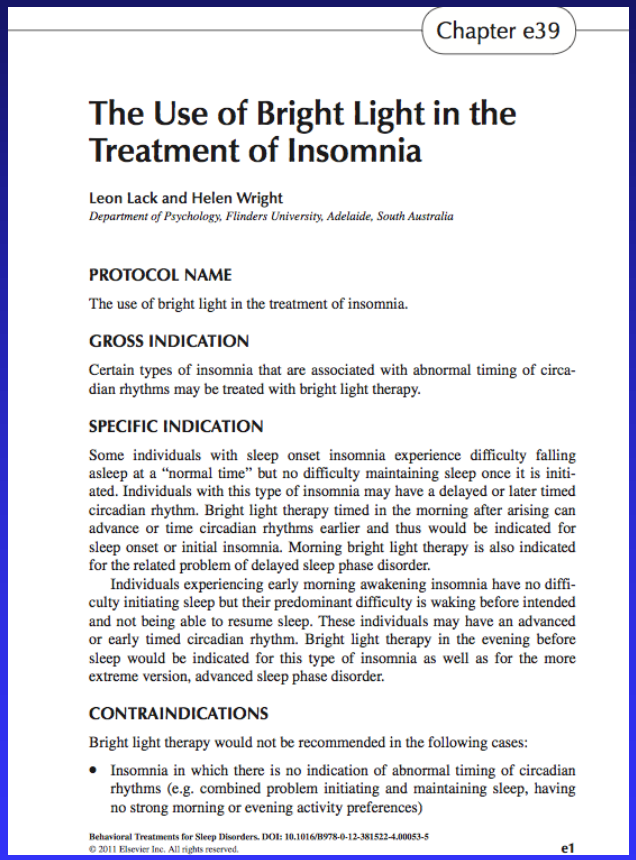
- DETERMINE HABITUAL BT/WT
- MAINTAIN ADHERENCE TO SCHEDULE
- BEGIN LIGHT THERAPY (60 mins.) AT HABITUAL WT
- ADVANCE BY 30 MINS EACH DAY UNTIL TARGET (Negotiable)
- DIM LIGHT FOR LAST 1-2 HOURS OF THE NIGHT (Consider blue blockers)
- MAINTAIN LIGHT AT TARGET SLEEP PHASE 1-2 WEEKS (May require boosters)
- MAINTAIN STIMULUS CONTROL INSTRUCTIONS

STEPS FOR LATE INSOMNIA/ASPS TX



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Behavioral Treatments for Sleep Disorders. DOI: 10.1016/B978-0-12-381522-4.00053-5
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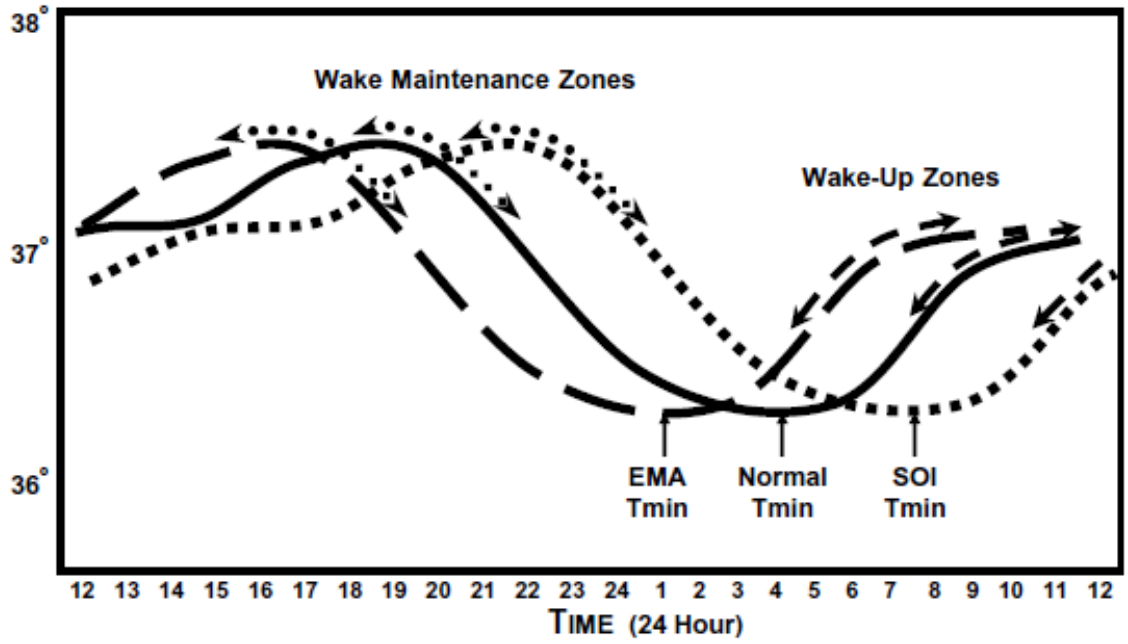
- **STAY ACTIVE AS LATE AS POSSIBLE**
- **BRIGHT LIGHT 120 MINS TO MIDNIGHT OR 1AM FOR 2 NIGHTS**
- **MAINTAIN BRIGHT LIGHT TO DESIRED BT FOR 5-7 MORE NIGHTS**
- **RESTRICT LIGHT EXPOSURE FIRST 1-2 HOURS IN AM (Consider blue blockers)**
- **MAY REQUIRE BOOSTERS**
- **MAINTAIN STIMULUS CONTROL INSTRUCTIONS**

WHAT'S UP WITH STAYING UP SO LATE?

WHAT IS SO IMPORTANT ABOUT FINDING T-MIN?



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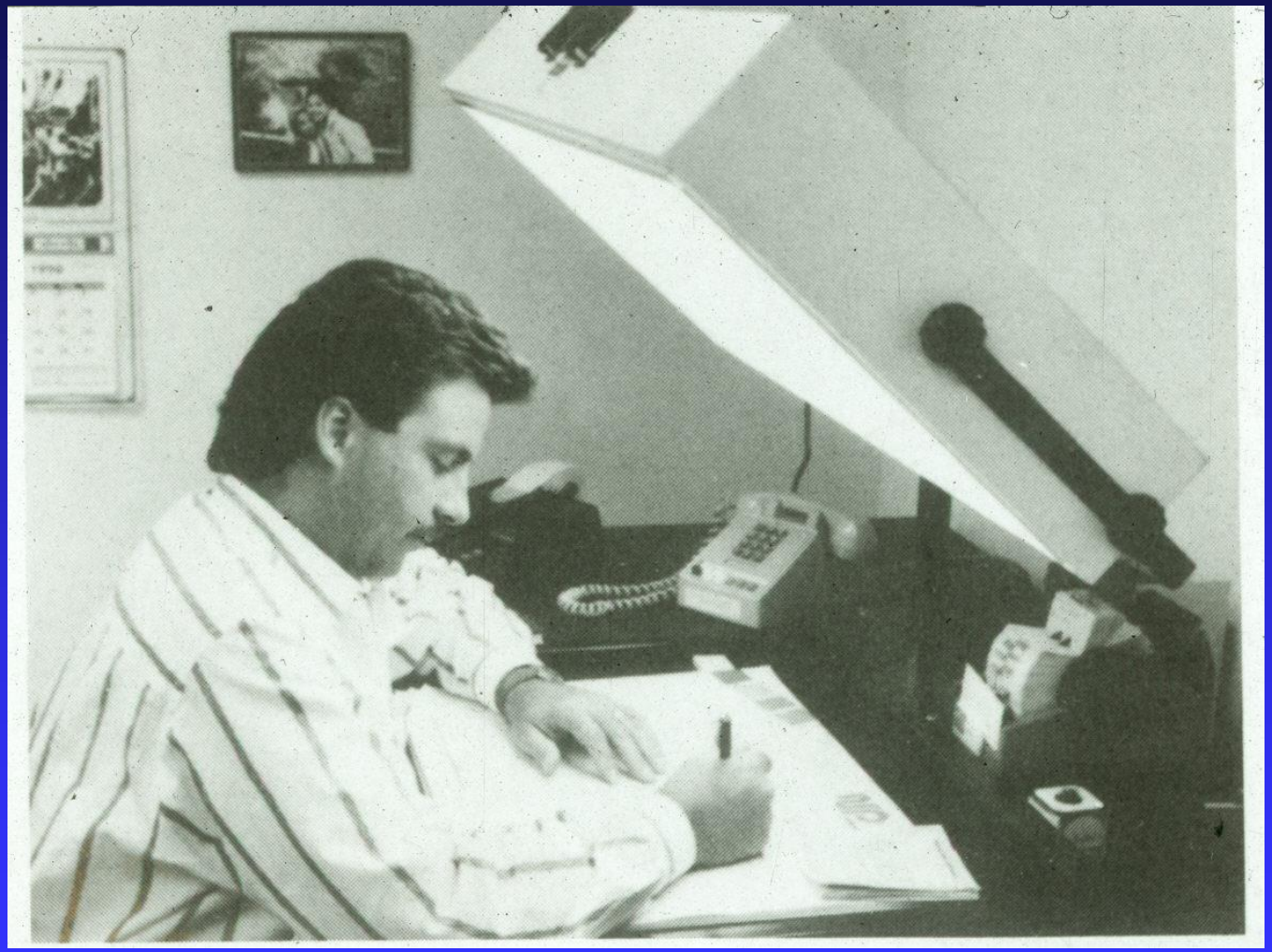
CONTRAINDICATIONS

- NO INDICATION OF CIRCADIAN DYSRYTHMIA
- OPTHAMALOGICAL ISSUES (e.g. cataracts, macular degeneration)
- MEDICAL OR MED INDUCED LIGHT SENSITIVITY

LIGHT SOURCE



THE PATIENT PERSPECTIVE

"MY psychiatrist is a fan of "happy lamps,"
lamps that use full-spectrum bulbs to treat wintertime seasonal depression.
A really big fan.
Opening his office door is like entering a scene in "Poltergeist":
I'm blinded by a powerful glow that emanates from every direction.
I must rely on his voice in the distance calling out to me in
order to orient myself. This is how our appointments begin:
I go toward the light."

http://www.nytimes.com/2014/03/28/opinion/its-no-substitute-for-the-sun.html?hp&rref=opinion

DATA

Adult Clinical Research

The Effect of Evening Bright Light in Delaying the Circadian Rhythms and Lengthening the Sleep of Early Morning Awakening Insomniacs

Leon Lack and Helen Wright

Sleep Laboratory, Flinders University of South Australia, Adelaide, Australia

Summary: Past studies have predicted that early morning awakening insomnia is associated with advanced or early circadian rhythms. Because bright light stimulation in the evening can delay the phase of circadian rhythms, we tested its effects on nine (4 females, 5 males) early morning awakening insomniacs. Their sleep was evaluated with wrist actigraphy and their temperature and melatonin circadian rhythms were measured in constant routine procedures. In the initial evaluation, the temperature rhythm phase positions of these insomniacs did appear to be earlier than normal. The subjects were then exposed to bright light stimulation (2,500 lux) from 2000 to 2400 hours on two consecutive evenings. Following the evening bright light treatment, temperature rhythm phase markers were delayed 2–4 hours and melatonin phase markers were delayed 1–2 hours. Sleep onset times were not changed but the mean final wake-up time was delayed from 0459 hours to 0611 hours, resulting in a mean increase of total sleep time of >1 hour. This pilot study suggests that evening bright light stimulation may be an effective nondrug treatment for early morning awakening insomnia. **Key Words:** Insomnia—Circadian rhythms—Bright light therapy—Body temperature—Early morning insomnia.

Sleep studies have shown the incidence of early morning awakening insomnia (EMA) ranges from 3% to 22% (1,2). Those who experience early morning awakenings also report midafternoon fatigue and have early sleep onsets, sometimes as early as 2100 hours. Usually there is no difficulty in initiating sleep. In fact, the tendency to fall asleep early in the night can be a social handicap. If the person does attempt to delay bedtime by remaining active in the evening, total sleep time is decreased since awakening still occurs at about the same time. They wake up much earlier than desired (e.g. 0300 hours) and because they are unable to get back to sleep, their total sleep time may range from only 5 hours to 7 hours (3). Excessive sleepiness may occur the following day due to the inability to "sleep in" in the morning (4).

It has been proposed that insomnia associated with sleep-scheduling disorders may be due to problems in the timing of a circadian pacemaker. Indeed, the clinical features described above are basically the same as those described for advanced sleep phase syndrome presumed to be due to an early or advanced circadian pacemaker (5). Normally people fall asleep 4–5 hours before their body temperature minima and wake up when their temperature is rising (6). Strogatz (7) and Zulley et al. (8) identified a wake-up zone 4–7 hours after the body temperature minimum, when sleeping subjects would wake, regardless of how long they had been asleep. If the body temperature rhythm is phase advanced in relation to the preferred sleep period, the wake-up zone would occur earlier (0300–0500 hours) and, predictably, terminate the sleep period before sufficient sleep is obtained (9).

A number of studies have shown bright light visual stimulation to be an effective manipulator of circadian rhythm phase. Exposure to bright light in the morning advanced the circadian rhythms of control subjects and subjects suffering delayed sleep phase syndrome (10–13). Conversely, evening bright light exposure has been shown to phase delay circadian body temperature rhythms in normal subjects (14–19).

Lewy et al. first suggested that delayed as well as advanced sleep phase syndrome can be helped by ap-

Accepted for publication March 1993.

Address correspondence and reprint requests to Dr. Leon Lack, School of Psychology, Flinders University, GPO Box 2100, Adelaide South Australia 5001, Australia.

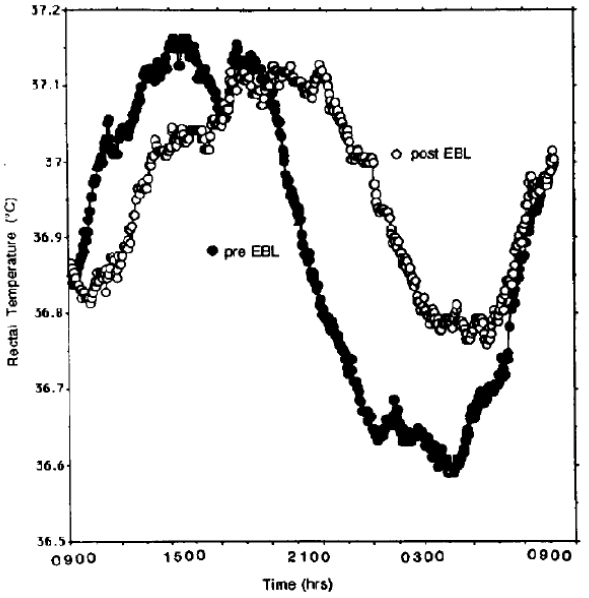


FIG. 1. Group mean rectal temperatures measured in the constant routines before (filled circles) and after (open circles) evening bright light (EBL) treatment.

TABLE 2. *Means of sleep onset, final wake-up time and total sleep time (hours and minutes) pre- and post-treatment with evening bright lights and separate bright light post-treatment days 1–5. Asterisks indicate a significant difference from the pre-treatment mean*

	Pre-treatment mean	Post-treatment mean	Post-treatment days 1	2	3	4	5
Sleep onset	2253	2300	2240	2315	2304	2304	2255
Wake-up time	0459	0611**	0630**	0558*	0556**	0620**	0608*
Total sleep time	5 hours 13 minutes	6 hours 26 minutes	7 hours 24 minutes	5 hours 43 minutes	5 hours 59 minutes	6 hours 36 minutes	6 hours 36 minutes

* $p < 0.05$.
** $p < 0.01$.

Lack & Wright, SLEEP. 1993; 16(5):436-443

WHAT ABOUT MELATONIN?

DATA

13 subjects (9 melatonin, 4 placebo)

2 doses (.3mg and 3mg combined)

Administered 6.5 hrs before DLMO

Administration advanced 1hr after 2weeks

Table 3—Effect of Treatment on Sleep Measures and Circadian Phase

	Baseline	Post-treatment	Change
DLMO			
Melatonin	23.67 ± 1.70 (9)	22.25 ± 0.80 (8)	1.75 ± 0.89***
Placebo	22.83 ± 1.44 (3)	23.17 ± 2.02 (3)	-0.33 ± 0.58
Tmin			
Melatonin	7.78 ± 2.29 (8)	6.15 ± 1.86 (8)	1.63 ± 1.79*
Placebo	N.A.	N.A.	N.A.
Sleep Onset			
Melatonin	1.88 ± 1.36 (8)	1.43 ± 1.56 (8)	0.45 ± 0.68
Placebo	1.71 ± 1.90 (3)	1.38 ± 1.72 (3)	0.33 ± 0.27
Sleep Offset			
Melatonin	9.36 ± 1.75 (8)	8.57 ± 1.15 (8)	0.79 ± 1.03#
Placebo	7.67 ± 0.60 (3)	8.14 ± 1.75 (3)	-0.47 ± 1.17
Total Sleep Time			
Melatonin	7.49 ± .68 (8)	7.13 ± .69 (8)	-0.36 ± 1.11
Placebo	5.97 ± 1.34 (3)	6.88 ± .09 (3)	0.91 ± 1.35
Sleep Efficiency			
Melatonin	81.75 ± 11.32 (8)	80.25 ± 10.07 (8)	-1.50 ± 7.56
Placebo	79.51 ± 2.33 (3)	81.32 ± 3.53 (3)	1.80 ± 2.14
Sleep Latency			
Melatonin	0.42 ± 0.51 (8)	0.30 ± 0.30 (8)	-0.11 ± 0.28
Placebo	0.39 ± 0.42 (3)	0.34 ± 0.14 (3)	0.05 ± 0.29

Average time in hours ± sd, (n) By convention, advances in phase are depicted as positive, and delays in phase are depicted as negative. (* $p < 0.05$, *** $p < 0.001$, # $p = 0.067$). N.A. Temperature data was not available for three of the four subjects in the placebo group.

Mundey, et. al. SLEEP 2005; 28(10):1271-1278

POSSIBLE ADJUVENTS AND ALTERNATIVES

BRIGHT LIGHT THERAPY

SLEEP COMPRESSION

COUNTER CONTROL

ISR

MINDFULNESS

SLEEP COMPRESSION PROTOCOL

Journal of Consulting and Clinical Psychology
2001, Vol. 69, No. 2, 227–239

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0022-006X/01/$5.00 DOI: 10.1037/0022-006X.69.2.227

Relaxation and Sleep Compression for Late-Life Insomnia:
A Placebo-Controlled Trial

Kenneth L. Lichstein
University of Memphis and Methodist Healthcare of Memphis

Brant W. Riedel and Nancy M. Wilson
University of Memphis

Kristin W. Lester and R. Neal Aguillard
Methodist Healthcare of Memphis

Older adults with insomni
sleep compression, and p
and 1-year follow-up a
following conclusions: A
Clinical significance an
suggested that sleep com
individuals with high day
in relaxation, and individ
sleep, as in sleep compr
treatment implementation

Chronic insomnia, referring to persisten
may have a pervasive impact on one's quality
of data identifies disturbed mood and anxiet
promised quality of life as common sequela
& Lichstein, 2000).
Insomnia in older adults is more common a
than it is in younger people. Insomnia preva
often exceeds 25% (e.g., Mellinger, Balter,
and these same surveys found, in sample
30–50% higher rate of insomnia than in
people. Older adults with insomnia (OAWI)
tion at a disproportionately high rate, risking
macy interactions, exacerbation of sleep ap
cessation lasting 10 s or longer), which is m
people, and multiple other side effects (Mell
Roth, Zorick, Wittig, & Roehrs, 1982).
The combination of high treatment need
ward side effects from hypnotic medications

Kenneth L. Lichstein, Department of Psycholo
phis, and Sleep Disorders Center, Methodist H
Memphis, Tennessee; Brant W. Riedel and Nancy
of Psychology, University of Memphis; Kristin
Aguillard, Sleep Disorders Center, Methodist Hea
This research was supported by Grant AG12
Institute on Aging, by Methodist Healthcare of
University of Memphis, Department of Psycholo
Psychological Research, part of the State of Tenn
lence grant program.
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L. Lichstein, Sleep Research Project, Department
sity of Memphis, 202 Psychology Building, Mem
3230. Electronic mail may be sent to lichstein@m

Chapter 5

Sleep Compression

Kenneth L. Lichstein, S. Justin Thomas
Department of Psychology, University of Alabama, Tuscaloosa, AL

Susan M. McCurry
Department of Psychosocial and Community Health, University of Washington, Seattle, WA

PROTOCOL NAME

Sleep compression.

GROSS INDICATION

Sleep compression is ideal for those who exhibit sleep continuity disturbance but not substantial daytime deficits.

SPECIFIC INDICATION

Poor sleep accompanied by little daytime impairment suggests that enough sleep has been obtained to satisfy biologic need. Decreasing wake time in bed, not increasing sleep, becomes the primary therapeutic goal.

There is insufficient experience with this method to recommend its preferred use with a type of insomnia (e.g., primary vs comorbid, midlife vs late life) or with a particular pattern of wakefulness (e.g., onset vs maintenance). However, sleep compression does use an incremental approach to decreasing time in bed, as compared to abrupt contraction in the method of sleep restriction, and sleep compression may be better tolerated by individuals who are experiencing daytime fatigue or mild sleepiness, or who may be sensitive to abrupt alteration of their time in bed pattern.

CONTRAINDICATIONS

There are no serious contraindications for sleep compression. Temporary, increased daytime sleepiness that sometimes occurs with the introduction of the similar procedure of sleep restriction has not been observed with sleep compression.

Behavioral Treatments for Sleep Disorders. DOI: 10.1016/B978-0-12-381522-6.00005-5
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DETERMINE AVERAGE SLEEP OPPORTUNITY AND SLEEP ABILITY USING 2 WEEKS OF DIARIES

DETERMINE THE DIFFERENCE BETWEEN TIB AND TST (DIFF)

DETERMINE AMOUNT OF SLEEP RESTRICTION (DIFF/ 5)

DELAY BEDTIME OR ADVANCE RISE TIME BY (DIFF/ 5) PER WEEK

TRACK SE% AND APPLY SRT TITRATION RULES

THE KINDER GENTLER SLEEP RESTRICTION?

FOR WHOM MIGHT THIS BE USEFUL ?

SHORT SLEEPERS

HIGHLY SLEEPY/ OSA

ELDERLY/ INFIRM

HIGHLY ANXIOUS

DATA

Sleep Diary Means and Standard Deviations Across Time

	Baseline		Posttreatment			Follow-up		
Measure	*M*	*SD*	*M*	*SD*	Effect size	*M*	*SD*	Effect size
Sleep latency (min)								
Relaxation	32.43	19.67	21.58	14.66	.18	27.40	18.50	.40
Sleep compression	32.82	29.80	21.30	16.44	.18	22.58	16.53	.63
Placebo	34.83	21.39	24.15	14.57		36.61	27.04	
No. of awakenings								
Relaxation	2.31	1.17	1.91	1.01	.28	2.00	0.82	.41
Sleep compression	2.39	1.14	1.70	1.08	.44	1.71	0.65	.67
Placebo	2.05	0.77	2.25	1.41		2.50	1.57	
WASO (min)								
Relaxation	66.47	37.05	42.57	26.39	.26	52.00	46.41	.16
Sleep compression	66.60	32.91	42.37	31.96	.24	38.25	27.77	.70
Placebo	72.00	36.07	49.70	28.15		58.19	29.40	
TST (min)								
Relaxation	345.01	78.44	397.79	87.15	.28	404.39	87.93	.43
Sleep compression	327.98	57.57	314.00	81.96	−.89	364.42	69.40	−.14
Placebo	332.25	71.09	376.80	54.92		372.90	53.01	
SE								
Relaxation	71.85	13.82	80.67	12.76	.16	79.62	13.28	.29
Sleep compression	71.10	12.41	78.61	14.83	−.02	81.47	11.83	.48
Placebo	69.22	12.43	78.86	8.76		76.14	10.48	
Sleep quality rating								
Relaxation	2.88	0.55	3.50	0.60	.36	3.38	0.50	.38
Sleep compression	2.80	0.61	3.38	0.57	.16	3.47	0.52	.54
Placebo	2.94	0.47	3.29	0.56		3.18	0.55	
Napping (min/day)								
Relaxation	12.12	13.99	7.12	10.98	−.11	12.49	21.77	−.13
Sleep compression	12.65	13.78	6.54	9.86	−.06	7.49	10.07	.25
Placebo	9.12	13.49	5.93	9.55		10.21	11.38	

Note. The effect sizes compare each treatment group with the placebo group. WASO = wake time after sleep onset; TST = total sleep time; SE = sleep efficiency.

Lichstein, et. al., J. Consul and Clin Psych. 2001; 69(2): 227-239

DATA

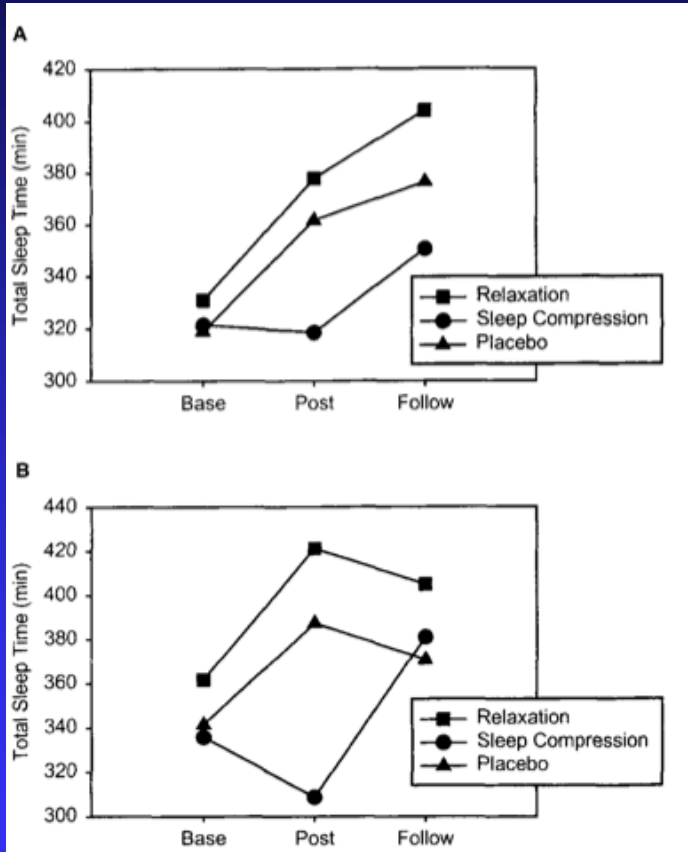


Figure 1. Treatment effects on total sleep time among (A) high-fatigue and (B) low-fatigue individuals. Base = baseline; Post = posttreatment; Follow = follow-up.

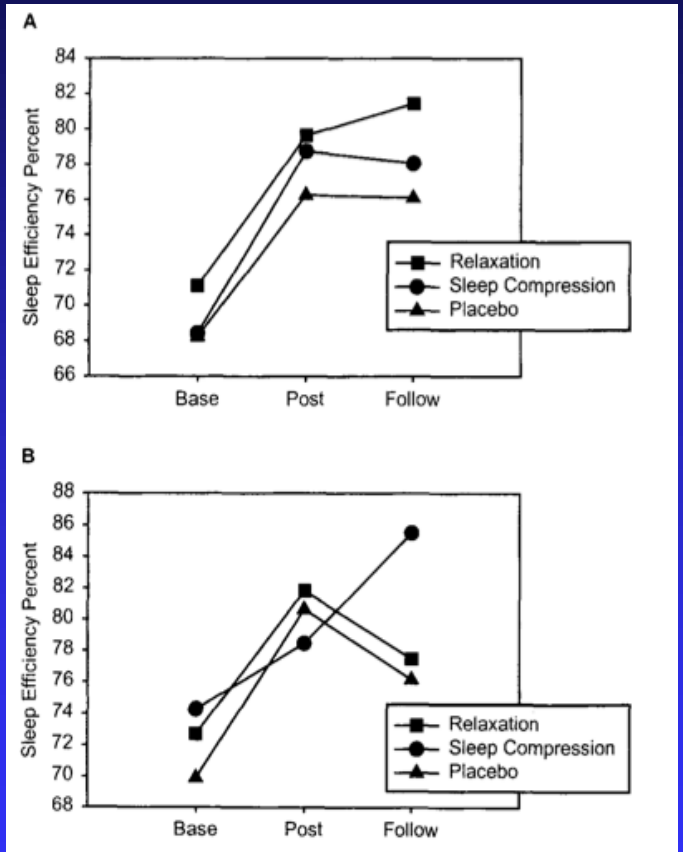


Figure 2. Treatment effects on sleep efficiency percentage among (A) high-fatigue and (B) low-fatigue individuals. Base = baseline; Post = posttreatment; Follow = follow-up.

Lichstein, et. al., J. Consul and Clin Psych. 2001; 69(2): 227-239

POSSIBLE ADJUVENTS AND ALTERNATIVES

BRIGHT LIGHT THERAPY

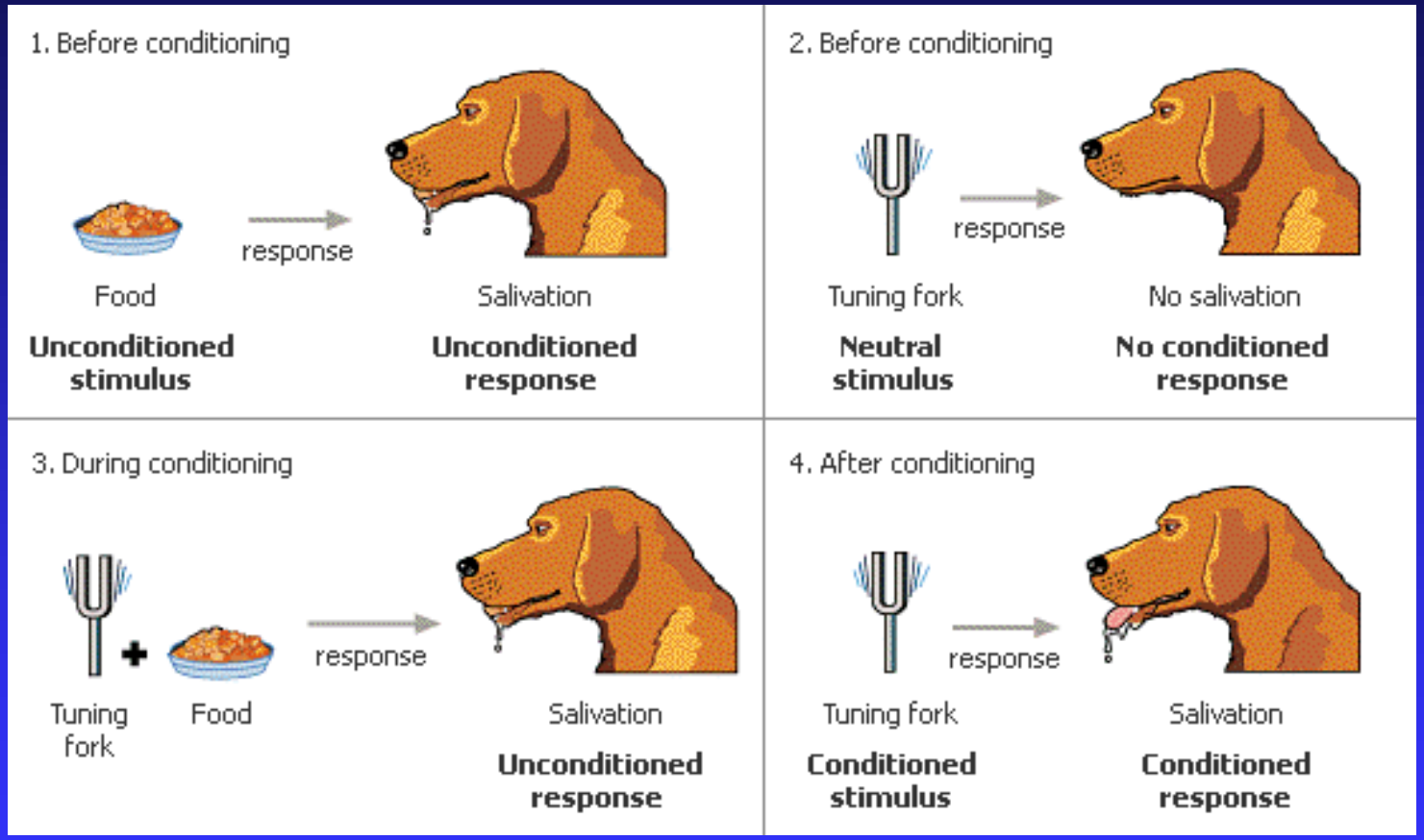
SLEEP COMPRESSION

COUNTER CONTROL

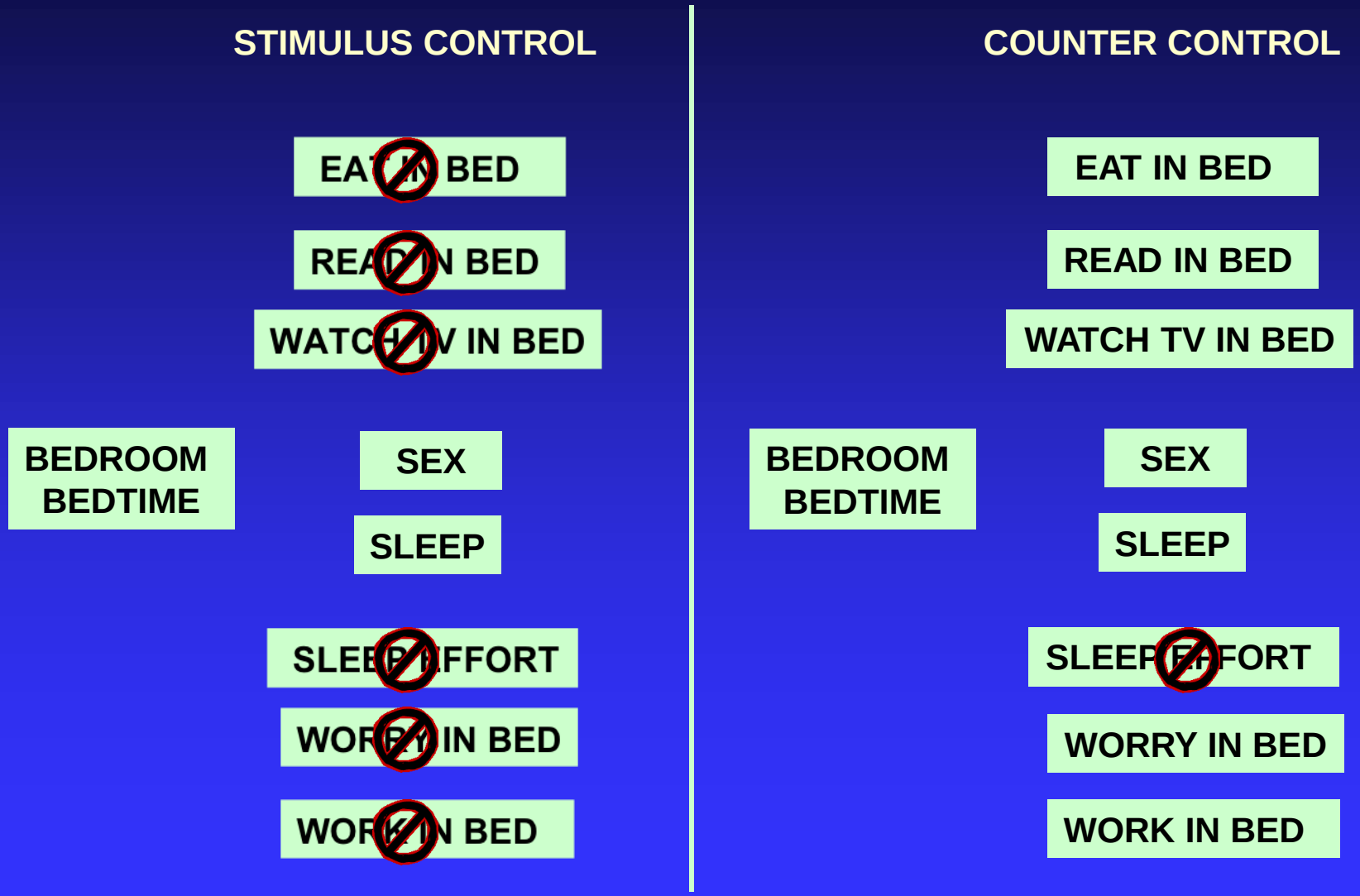
ISR

MINDFULNESS

AN ALTERNATIVE TO STIMULUS CONTROL



STIMULUS CONTROL VS COUNTER CONTROL



FOR WHOM MIGHT THIS BE USEFUL ?

ELDERLY/ INFIRM

HIGHLY ANXIOUS

COUNTER CONTROL

Psychology and Aging
1986, Vol. 1, No. 3, 233-238

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0882-7974/86/$00.75

Countercontrol Treatment of Sleep-Maintenance Insomnia in Relation to Age

Ruth Davies, Patricia Lacks, Martha Storandt, and Amy D. Bertelson
Washington University

We administered countercontrol behavioral therapy for sleep-maintenance insomnia to 34 insomniacs—ranging in age from 35 to 78 years—in small groups. Twenty-two subjects received immediate and 12 received delayed treatment. Three self-report measures of sleep disruption were collected on daily sleep diaries at baseline, termination of treatment, 1-month follow-up, and 12-month follow-up. Although amount of time awake at night was correlated with age ($r = .50$), response to treatment was not. Even though older people experienced more time awake after sleep onset prior to treatment, they were able to profit from therapy as well as the younger insomniacs. Countercontrol therapy reduced the sleep complaint for the total group by about 30% at the end of treatment, with gradual improvement continuing through a 4-week follow-up. Nevertheless, it appears that sleep-maintenance insomnia may be more difficult to treat than sleep-onset problems.

Older adults have a high rate of sleep-related complaints (Miles & Dement, 1980; Webb, 1983), which are most commonly treated with hypnotics and other sedating drugs. Use of sleeping pills is very common among older adults, even though they are known to have only short-term effectiveness and many detrimental side effects. The latter include development of tolerance, next-day drowsiness and lowered performance, toxic drug interactions, compromised cortical activity, and the possibility of impaired respiratory, hepatic, or renal functioning because of drug accumulation (Bootzin, Engle-Friedman, & Hazelwood, 1983; Miles & Dement, 1980; Regestein, 1980). Many studies have explored the usefulness of a variety of nondrug, behavioral treatments for insomnia. Borkovec (1982) concluded that stimulus control strategies have demonstrated the highest overall success for young and middle-aged adults. Only one study has examined the effectiveness of the stimulus control treatment with older sleep-onset insomniacs (Puder, Lacks, Bertelson, & Storandt, 1983). Older adults showed improvements that were equivalent or slightly better than those obtained by individuals under age 60 in a previous study (Lacks, Bertelson, Gans, & Kunkel, 1983).

Almost all of the studies of the behavioral treatment of insomnia have focused on sleep-onset difficulties (i.e., problems in getting to sleep). Older adults, however, more frequently suffer from sleep-maintenance insomnia (more wake time after sleep onset and more arousals; Webb & Campbell, 1980). Very little has been published on the treatment of sleep-maintenance insomnia for any age group (for several case studies, see Coates & Thoresen, 1979; Thoresen, Coates, Kirmil-Gray, & Rosekind, 1981). The one controlled group study of younger individuals with sleep-maintenance problems showed that stimulus control reduced wakefulness significantly, but no more so than did a credible placebo (Lacks, Bertelson, Sugerman, & Kunkel, 1983). Stimulus control treatment consists of using the bed only for sleep and establishing a regular sleep schedule. Many of the subjects in that study complained about the stimulus control requirement of getting out of bed after every 10-min period of sleeplessness during the night. It is possible that this requirement may have adversely influenced the sleep-maintenance insomniacs' response to treatment.

In looking more closely at the stimulus control procedure, several researchers (e.g., Borkovec, 1982) suggested that it may owe most of its effectiveness to the disruption of sleep-incompatible behaviors such as cognitive activity or restless tossing. If the crucial component to stimulus control is the disruption of these activities rather than reestablishing the bed as a discriminative stimulus for sleep, then the requirement to leave the bed and bedroom should be largely superfluous. By removing the out-of-bed requirement, perhaps effectiveness would be improved for the more-difficult-to-treat sleep-maintenance problem. A treatment that can be done in bed may also be more feasible for the sometimes less ambulatory older adult.

An intervention developed by Zwart and Lisman (1979) seems suited for this goal. The treatment, called *countercontrol*, was designed to disrupt sleep-incompatible activities without introducing novel procedures (such as leaving the bed) that might be responsible for any observed improvement. Participants were instructed to deliberately engage in a nonarousing activity (e.g., dull reading) in bed whenever they were unable to

This research was supported in part by Grant BRSG SO7 RR07054-17, awarded by the Biomedical Research Grant Program, Division of Research Resources, National Institutes of Health, and by National Institute on Aging, Training Grant AG 00030. The results were presented at the annual meeting of the Gerontological Society of America, San Antonio, Texas, in November 1984.

The authors would like to express their appreciation to Dan Cody, Ellen Levy, Kimberly Powlishta, Claude Rabinowitz, Angela Rosenberg, Monique Rotert, and Wendy Zeppelin for their assistance. Thanks are also extended to three anonymous reviewers for their helpful comments on an earlier draft of this article.

Correspondence concerning this article should be addressed to Patricia Lacks, Department of Psychology, Washington University, St. Louis, Missouri 63130.

34 SUBJECTS

MEAN AGE 58.5

WASO ONLY (79.91)

DURATION 13.4YRS

IMMEDIATE CONDITION

DELAYED CONDITION

Davies, et. al., Psychology and Aging.1986; 1(3): 233-238

DATA

Means of Three Indices of Insomnia for Two Treatment Groups Throughout the Course of the Study

Treatment group	Time of measurement					
	Week 0	Week 3	Week 4	Week 7	Week 8	Week 12
Minutes awake after sleep onset (WASO)						
Immediate						
M	79.91	65.91	58.00		54.90[a]	
SD	48.17	50.40	48.69		45.31	
Delayed						
M	73.92		78.42	48.42	54.75	31.64[a]
SD	42.68		34.45	51.27	54.69	34.21
Average no. of awakenings per night						
Immediate						
M	2.54	1.66	1.82		1.92	
SD	1.02	0.80	0.89		0.77	
Delayed						
M	1.27		1.37	1.32	1.22	1.04
SD	0.53		0.83	1.05	0.66	0.81
Average no. of awakenings of 10 min or more						
Immediate						
M	2.11	1.50	1.59		1.68	
SD	0.84	0.84	0.90		0.86	
Delayed						
M	1.09		1.31	1.06	1.04	0.81
SD	0.61		0.86	0.77	0.58	0.84

Note. Treatment occurred between Week 0 and Week 4 for the immediate group ($n = 22$) and between Week 4 and Week 8 for the delayed group ($n = 12$).

[a] These follow-up data are based on 19 people in the immediate group and 11 in the delayed group.

POSSIBLE ADJUVENTS AND ALTERNATIVES

BRIGHT LIGHT THERAPY

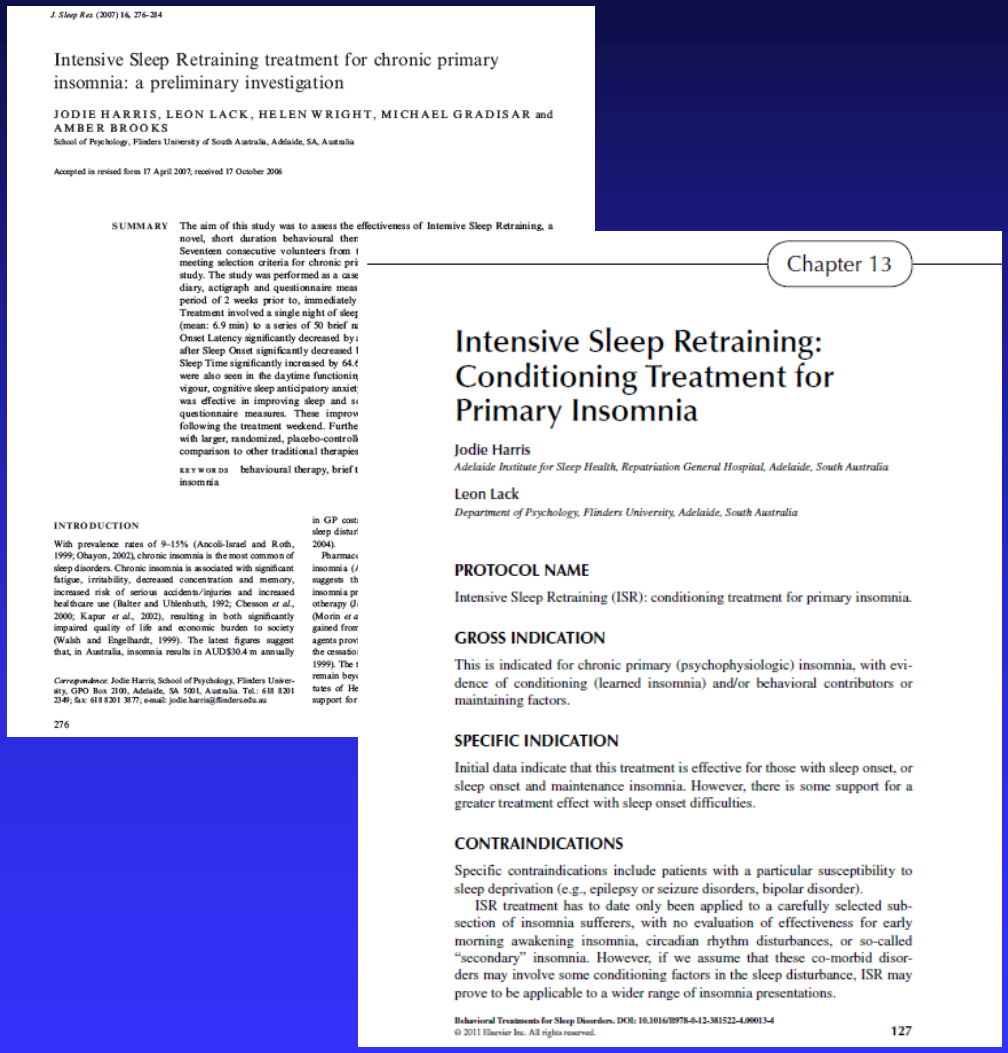
SLEEP COMPRESSION

COUNTER CONTROL

ISR

MINDFULNESS

ISR PROTOCOL



J. Sleep Res. (2007) 16, 276–284

Intensive Sleep Retraining treatment for chronic primary insomnia: a preliminary investigation

JODIE HARRIS, LEON LACK, HELEN WRIGHT, MICHAEL GRADISAR and AMBER BROOKS
School of Psychology, Flinders University of South Australia, Adelaide, SA, Australia

Accepted in revised form 17 April 2007; received 17 October 2006

SUMMARY The aim of this study was to assess the effectiveness of Intensive Sleep Retraining, a novel, short duration behavioural ther
Seventeen consecutive volunteers from
meeting selection criteria for chronic pri
study. The study was performed as a case
diary, actigraph and questionnaire meas
period of 2 weeks prior to, immediately
Treatment involved a single night of sleep
(mean: 6.9 min) to a series of 50 brief
Onset Latency significantly decreased by
after Sleep Onset significantly decreased
Sleep Time significantly increased by 64.
were also seen in the daytime functionin
vigour, cognitive sleep anticipatory anxiet
was effective in improving sleep and s
questionnaire measures. These improv
following the treatment weekend. Furthe
with larger, randomized, placebo-controll
comparison to other traditional therapies

KEYWORDS behavioural therapy, brief
insomnia

INTRODUCTION

With prevalence rates of 9–15% (Ancoli-Israel and Roth, 1999; Ohayon, 2002), chronic insomnia is the most common of sleep disorders. Chronic insomnia is associated with significant fatigue, irritability, decreased concentration and memory, increased risk of serious accidents/injuries and increased healthcare use (Balter and Uhlenhuth, 1992; Chesson *et al.*, 2000; Kapur *et al.*, 2002), resulting in both significantly impaired quality of life and economic burden to society (Walsh and Engelhardt, 1999). The latest figures suggest that, in Australia, insomnia results in AUD$30.4 m annually in GP cost
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Correspondence: Jodie Harris, School of Psychology, Flinders University, GPO Box 2100, Adelaide, SA 5001, Australia. Tel.: 618 8201 2149; fax: 618 8201 3877; e-mail: jodie.harris@flinders.edu.au

Chapter 13

Intensive Sleep Retraining: Conditioning Treatment for Primary Insomnia

Jodie Harris
Adelaide Institute for Sleep Health, Repatriation General Hospital, Adelaide, South Australia

Leon Lack
Department of Psychology, Flinders University, Adelaide, South Australia

PROTOCOL NAME

Intensive Sleep Retraining (ISR): conditioning treatment for primary insomnia.

GROSS INDICATION

This is indicated for chronic primary (psychophysiologic) insomnia, with evidence of conditioning (learned insomnia) and/or behavioral contributors or maintaining factors.

SPECIFIC INDICATION

Initial data indicate that this treatment is effective for those with sleep onset, or sleep onset and maintenance insomnia. However, there is some support for a greater treatment effect with sleep onset difficulties.

CONTRAINDICATIONS

Specific contraindications include patients with a particular susceptibility to sleep deprivation (e.g., epilepsy or seizure disorders, bipolar disorder).

ISR treatment has to date only been applied to a carefully selected subsection of insomnia sufferers, with no evaluation of effectiveness for early morning awakening insomnia, circadian rhythm disturbances, or so-called "secondary" insomnia. However, if we assume that these co-morbid disorders may involve some conditioning factors in the sleep disturbance, ISR may prove to be applicable to a wider range of insomnia presentations.

Behavioral Treatments for Sleep Disorders. DOI: 10.1016/B978-0-12-381522-4.00013-4
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CONDUCT AN IN-LAB 24 HR PSG

PROVIDE A NAP OPP ONCE EVERY 30 MIN

MONITOR FOR 3-5 MIN SLEEP AND AWAKEN SBJ

30 MINUTES LATER CONDUCT THE NEXT NAP OPP

FOR WHOM MIGHT THIS BE USEFUL ?

PATIENTS WITH EDS

CASES WHEN CBT-I CONTRAINDCATED

RCT FOR ISR

INTENSIVE SLEEP RETRAINING FOR CHRONIC INSOMNIA

http://dx.doi.org/10.5665/sleep.1584

A Randomized Controlled Trial of Intensive Sleep Retraining (ISR): A Brief Conditioning Treatment for Chronic Insomnia

Jodie Harris, PhD[1,2]; Leon Lack, PhD[1,2]; Kristyn Kemp, PhD[3]; Helen Wright, PhD[3]; Richard Bootzin, PhD[4]

[1]School of Psychology, Flinders University, South Australia; [2]Adelaide Institute for Sleep Health, Repatriation General Hospital, South Australia; [3]Adelaide Insomnia Clinic, Adelaide, South Australia; [4]University of Arizona, Tucson, AZ

Study Objective: To investigate the effectiveness of intensive sleep retraining in comparison and combination with traditional behavioral intervention for chronic primary insomnia.

Participants: Seventy-nine volunteers with chronic sleep-onset insomnia (with or without sleep maintenance difficulties) were randomly assigned either to intensive sleep retraining (ISR), stimulus control therapy (SCT), ISR plus SCT, or the control (sleep hygiene) treatment condition.

Intervention: ISR treatment consisted of 50 sleep onset trials over a 25-h sleep deprivation period.

Measurements and Results: Treatment response was assessed with sleep diary, activity monitoring, and questionnaire measures. The active treatment groups (ISR, SCT, ISR+SCT) all resulted in significant improvements in sleep onset latency and sleep efficiency, with moderate to large effect sizes from pre- to post-treatment. Wake time after sleep onset decreased significantly in the SCT and ISR+SCT groups. Total sleep time increased significantly in the ISR and ISR+SCT treatment groups. Participants receiving ISR (ISR, ISR+SCT) experienced rapidly improved SOL and TST during treatment, suggesting an advantage of rapid improvements in sleep in response to ISR. Although there were few statistically significant differences between groups on individual variables, ISR+SCT resulted in consistently larger effect sizes of change than other treatments, including questionnaire measures of sleep quality, sleep self-efficacy, and daytime functioning. The combination treatment group (ISR+SCT) showed trends to outperform other active treatment groups with fewer treatment dropouts, and a greater proportion of treatment responders with 61% reaching "good sleeper" status. Treatment gains achieved at post-treatment in the active treatment groups were largely maintained throughout follow-up periods to 6 months.

Conclusion: This 25-hour intensive conditioning treatment for chronic insomnia can produce rapid improvements in sleep, daytime functioning, and psychological variables. Adding ISR to traditional interventions seems to result in a superior treatment response.

Keywords: Chronic insomnia, classical conditioning, behavioral treatment, intensive sleep retraining

Citation: Harris J; Lack L; Kemp K; Wright H; Bootzin R. A randomized controlled trial of intensive sleep retraining (ISR): a brief conditioning treatment for chronic insomnia. *SLEEP* 2012;35(1):49-60.

INTRODUCTION

Chronic insomnia is a pervasive, unrelenting sleep disorder, and is associated with considerable consequences for sufferers. Various conceptual models contribute an understanding of the development and maintenance of insomnia, in addition to helping inform treatment choice. Of those, a conditioning model offers a theoretical basis for the efficacy of behavioral intervention. This model suggests that the wakeful state associated with insomnia may be learned through a process of conditioning.[1] According to this model, transient sleep disturbance may be triggered by acute periods of stress or heightened arousal. With repeated episodes of wakefulness and distress in bed, an individual may rapidly associate the bedroom, bedtime, and other associated cues with anxiety and sleeplessness. Thus both temporal and contextual stimuli may become cues for apprehension, worries, and fear of being unable to sleep, in addition to the sleeplessness itself. Research suggests that once insomnia is established, it may persist over many years.[2,3]

Although a significant body of research supports cognitive behavioral treatments for insomnia, this common sleep disorder often remains untreated, or treated only with pharmacotherapy.[4] Of the behavioral interventions, stimulus control therapy (SCT) is the most widely studied and endorsed single component treatment method. Indeed, SCT is currently recommended with the highest standard of support by the American Academy of Sleep Medicine.[5] SCT is considered effective for both sleep onset and sleep maintenance insomnia symptoms.[6,7] The assumed mechanism involved in successful SCT implementation is the eventual conditioning of a rapid sleep onset, countering the learned psychophysiologic arousal (or absence of de-arousal[8]) associated with the insomnia response.

Nevertheless, the administration of behavioral treatment for insomnia is typically associated with a lag in treatment response, some early treatment sleepiness and/or fatigue, and some difficulties with treatment compliance. Consequently, the rapid treatment response associated with intensive sleep retraining (ISR)[9] may improve the response to and compliance with non-drug interventions. ISR is a brief 25-h conditioning treatment, involving the use of acute sleep deprivation to facilitate a series of rapid sleep onsets in an effort to counteract the conditioned insomnia response. In a case series study, this brief conditioning treatment has shown rapid and sustained improvements in sleep variables.[9]

The current study aims to compare the treatment response to both ISR and SCT treatments alone, in addition to evaluating a combination of the 2 treatment methods in comparison to a control (sleep hygiene intervention only) group.

A commentary on this article appears in this issue on page 11.

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79 SUBJECTS RANDOMIZED
TO
1 OF 4 CONDITIONS

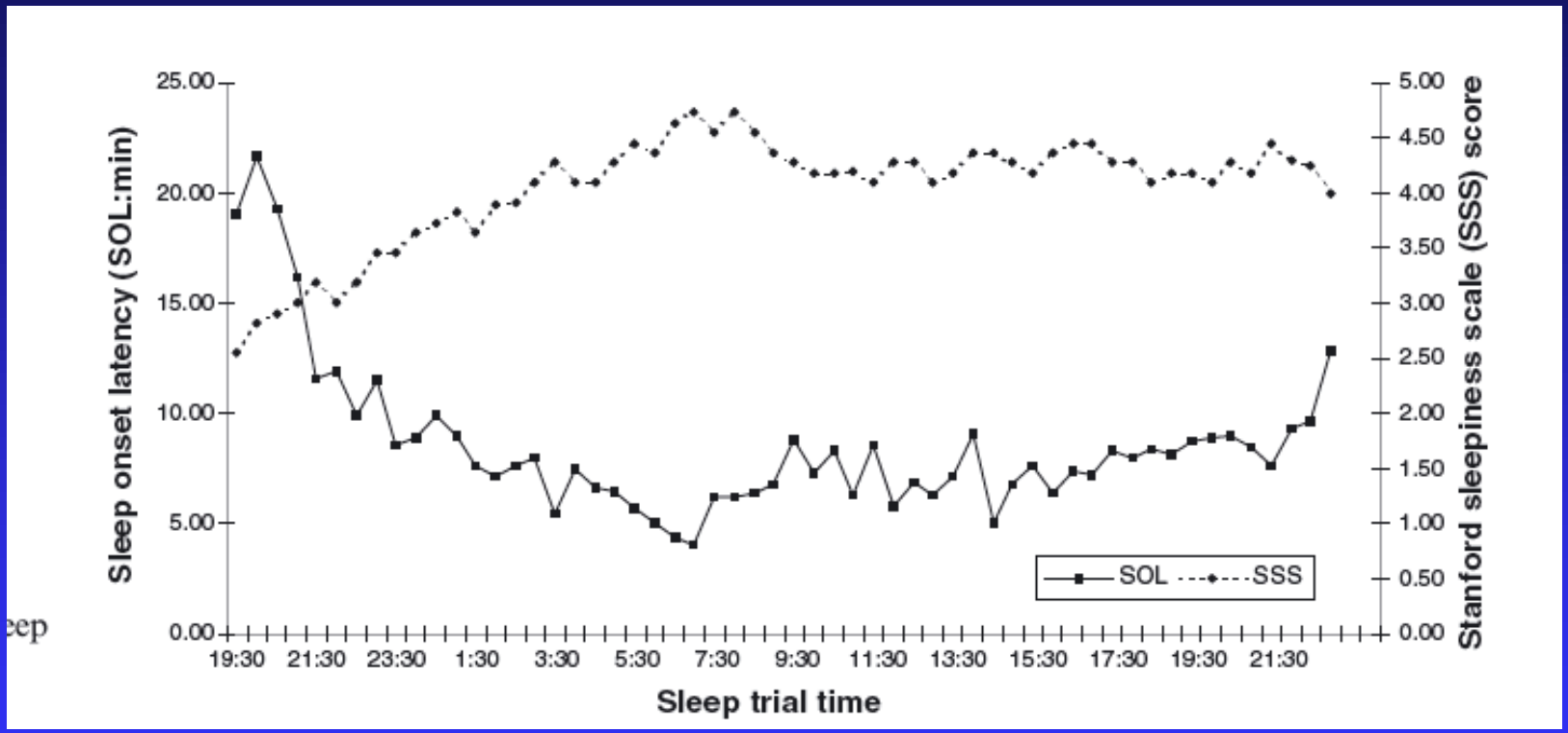
ISR (25h; 50 naps; 3m/nap)

SCT

ISR + SCT

SH ?

DATA



Harris, et. al., *SLEEP* 2012;35(1):49-60

DATA

Table 2—Mean (SD) sleep diary, actigraphy and questionnaire values from Pre-treatment (Pre-T) to Post-treatment (Post-T), and change effect sizes (*d*)

	ISR			SCT			ISR+SCT			CONTROL		
Variable	**Pre-T**	**Post-T**	***d***	**Pre-T**	**Post-T**	***d***	**Pre-T**	**Post-T**	***d***	**Pre-T**	**Post-T**	***d***
Sleep Diary												
SOL	61.41 (25.21)	38.41** (16.24)	0.61	68.33 (44.04)	38.94*** (29.39)	0.78	60.79 (42.79)	24.70*** (12.83)	0.96	71.87 (37.23)	68.65 (37.72)	0.09
TST	368.59 (62.71)	403.23** (55.37)	0.53	370.65 (54.13)	387.95 (57.36)	0.26	357.41 (65.61)	411.91*** (50.91)	0.83	348.76 (78.83)	350.26 (76.76)	0.02
WASO	75.57 (56.00)	60.71 (59.01)	0.26	72.34 (44.47)	42.66** (37.53)	0.52	87.24 (53.30)	42.42*** (25.32)	0.79	99.25 (70.48)	80.31* (57.38)	0.33
SE	70.02 (11.08)	79.85*** (8.84)	0.65	70.64 (9.40)	81.57*** (7.34)	0.91	68.08 (11.89)	84.20*** (5.68)	1.34	65.96 (14.89)	68.24 (14.14)	0.24
Actigraphy												
SOL	38.53 (17.18)	28.05 (15.29)	0.35	34.17 (23.73)	28.36 (15.60)	0.19	40.53 (26.46)	31.89 (32.04)	0.29	47.54 (45.70)	43.01 (42.64)	0.15
TST	386.61 (69.92)	392.38 (62.54)	0.09	375.86 (55.32)	365.12* (65.98)	0.17	388.84 (58.22)	378.24 (71.41)	0.17	379.40 (73.84)	368.50* (72.74)	0.17
WASO	90.04 (66.90)	90.31 (69.04)	0.01	93.85 (49.46)	92.47 (44.00)	0.03	85.37 (50.48)	84.20 (50.90)	0.02	99.47 (43.11)	100.19 (46.49)	0.01
SE	75.32 (12.27)	76.57 (11.91)	0.11	74.33 (10.96)	75.61 (9.82)	0.11	75.55 (10.03)	77.59 (11.63)	0.18	72.26 (12.58)	71.92 (11.91)	0.03

SOL, sleep onset latency; TST, total sleep time; WASO, total wake time overnight after sleep onset; SE, sleep efficiency (total sleep time/time in bed × 100). Data is derived from participants within each condition with available data at both assessment points. ANOVA (P) statistics for Pre-Treatment to Post-Treatment change, *** < 0.001, ** < 0.01, * < 0.05.

VARIATIONS ON A THEME

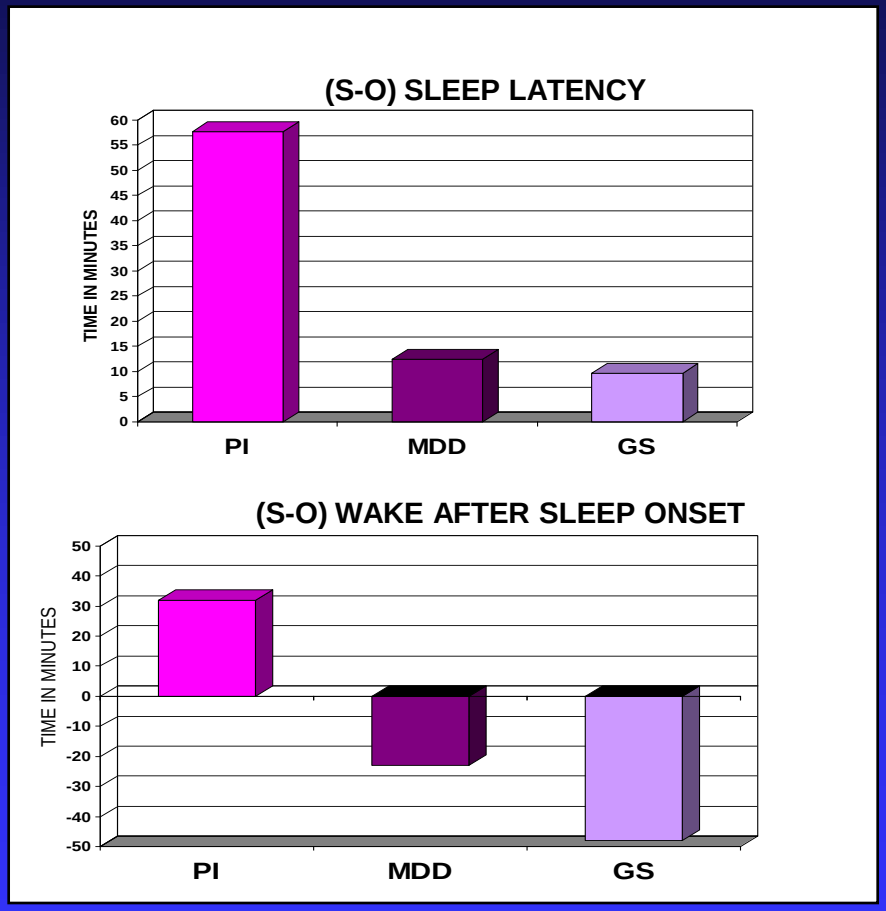
PATIENTS WITH EDS

CASES WHEN CBT-I IS CONTRAINDCATED

PARADOXICAL INSOMNIA

“SLEEP STATE MISPERCEPTION”

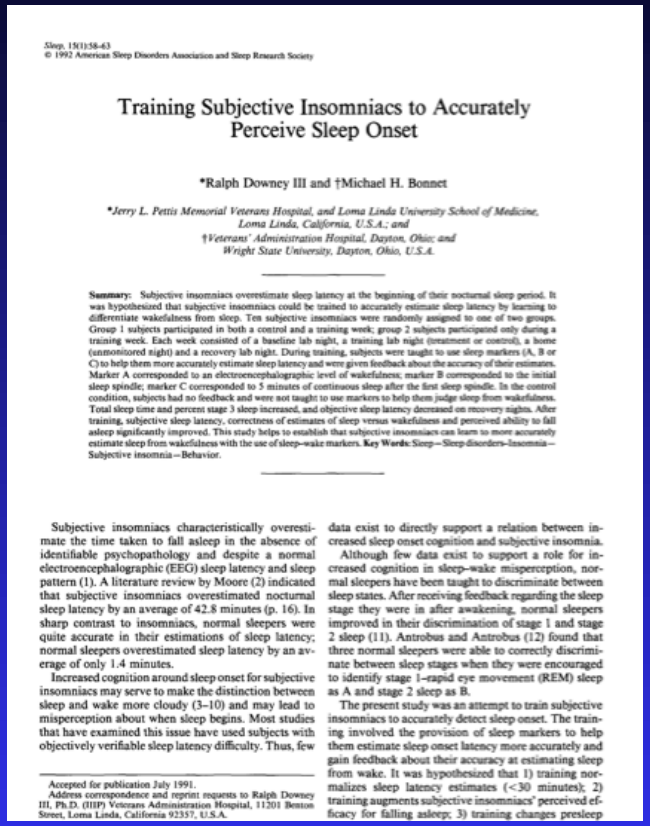
SUBJECTIVE-OBJECTIVE DISCREPANCY



“SLEEP STATE MISPERCEPTION”

SUBJECTIVE-OBJECTIVE DISCREPANCY

SLEEP LAB TRAINING



Sleep, 15(1):58-63
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Training Subjective Insomniacs to Accurately Perceive Sleep Onset

*Ralph Downey III and †Michael H. Bonnet

*Jerry L. Pettis Memorial Veterans Hospital, and Loma Linda University School of Medicine, Loma Linda, California, U.S.A.; and
†Veterans' Administration Hospital, Dayton, Ohio; and Wright State University, Dayton, Ohio, U.S.A.

Summary: Subjective insomniacs overestimate sleep latency at the beginning of their nocturnal sleep period. It was hypothesized that subjective insomniacs could be trained to accurately estimate sleep latency by learning to differentiate wakefulness from sleep. Ten subjective insomniacs were randomly assigned to one of two groups. Group 1 subjects participated in both a control and a training week; group 2 subjects participated only during a training week. Each week consisted of a baseline lab night, a training lab night (treatment or control), a home (unmonitored night) and a recovery lab night. During training, subjects were taught to use sleep markers (A, B or C) to help them more accurately estimate sleep latency and were given feedback about the accuracy of their estimates. Marker A corresponded to an electroencephalographic level of wakefulness; marker B corresponded to the initial sleep spindle; marker C corresponded to 5 minutes of continuous sleep after the first sleep spindle. In the control condition, subjects had no feedback and were not taught to use markers to help them judge sleep from wakefulness. Total sleep time and percent stage 3 sleep increased, and objective sleep latency decreased on recovery nights. After training, subjective sleep latency, correctness of estimates of sleep versus wakefulness and perceived ability to fall asleep significantly improved. This study helps to establish that subjective insomniacs can learn to more accurately estimate sleep from wakefulness with the use of sleep–wake markers. **Key Words:** Sleep—Sleep disorders—Insomnia—Subjective insomnia—Behavior.

Subjective insomniacs characteristically overestimate the time taken to fall asleep in the absence of identifiable psychopathology and despite a normal electroencephalographic (EEG) sleep latency and sleep pattern (1). A literature review by Moore (2) indicated that subjective insomniacs overestimated nocturnal sleep latency by an average of 42.8 minutes (p. 16). In sharp contrast to insomniacs, normal sleepers were quite accurate in their estimations of sleep latency; normal sleepers overestimated sleep latency by an average of only 1.4 minutes.

Increased cognition around sleep onset for subjective insomniacs may serve to make the distinction between sleep and wake more cloudy (3–10) and may lead to misperception about when sleep begins. Most studies that have examined this issue have used subjects with objectively verifiable sleep latency difficulty. Thus, few data exist to directly support a relation between increased sleep onset cognition and subjective insomnia.

Although few data exist to support a role for increased cognition in sleep–wake misperception, normal sleepers have been taught to discriminate between sleep states. After receiving feedback regarding the sleep stage they were in after awakening, normal sleepers improved in their discrimination of stage 1 and stage 2 sleep (11). Antrobus and Antrobus (12) found that three normal sleepers were able to correctly discriminate between sleep stages when they were encouraged to identify stage 1–rapid eye movement (REM) sleep as A and stage 2 sleep as B.

The present study was an attempt to train subjective insomniacs to accurately detect sleep onset. The training involved the provision of sleep markers to help them estimate sleep onset latency more accurately and gain feedback about their accuracy at estimating sleep from wake. It was hypothesized that 1) training normalizes sleep latency estimates (<30 minutes); 2) training augments subjective insomniacs' perceived efficacy for falling asleep; 3) training changes presleep

Accepted for publication July 1991.
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10 SUBJECTS

2 GROUPS

	Night[a]													
Group	1	2	3	4	5	6	7	8	9	10	11	12	13	14
1	A	H	BL	D	H	R	H	H	H	H	BL	DT	H	R
2										A	BL	DT	H	R

[a] A = adaptation night; H = night spent at home unmonitored; BL = baseline, undisturbed sleep; D = disruption night without feedback as to accuracy of subject's sleep–wake estimations (control); R = recovery night, undisturbed sleep; DT = disruption night with feedback as to accuracy of subject's sleep–wake estimations (treatment). Control week (group 1 only) is nights 2–6 shown above. Treatment week (groups 1 and 2) is nights 10–14 above.

DATA

Variable	G1C		G1T		G1C vs. G1T		G2T		G1T vs. G2T	
	Baseline	Recovery	Baseline	Recovery	*F* (cond)	*F* (nt)	Baseline	Recovery	*F* (cond)	*F* (nt)
Total sleep	289.0 (66.1)	339.4 (28.6)	325.2 (44.9)	342.0 (34.0)	1.31	3.21	296.8 (57.6)	390.7 (42.6)	0.22	5.80[b]
Recording time	359.7 (50.2)	404.4 (31.4)	396.9 (58.0)	381.4 (42.9)	5.91	0.05	415.5 (53.2)	428.9 (39.9)	1.11	0.01
Sleep efficiency	83.3 (13.3)	90.7 (6.5)	85.8 (8.5)	93.7 (3.4)	0.24	2.76	78.1 (19.4)	93.3 (5.2)	0.90	4.12
% stage 1	7.0 (3.5)	6.5 (4.7)	8.2 (5.1)	7.0 (3.6)	1.89	0.38	8.2 (4.2)	7.9 (3.2)	0.02	0.29
% stage 2	40.8 (17.9)	45.7 (6.9)	49.3 (12.6)	53.4 (1.2)	6.50	0.78	34.8 (9.9)	43.3 (7.6)	8.58[b]	2.18
% stage 3	4.2 (3.2)	3.4 (2.6)	3.9 (1.4)	3.5 (2.5)	0.50	12.07[b]	4.7 (2.8)	4.7 (3.8)	0.28	0.09
% stage 4	17.4 (6.7)	17.7 (8.2)	12.9 (9.5)	15.8 (8.5)	1.80	0.96	17.9 (5.4)	16.9 (9.7)	0.38	0.13
% stage REM	14.7 (4.2)	17.8 (5.2)	13.3 (6.2)	13.9 (3.4)	2.55	1.00	12.4 (6.6)	19.9 (8.0)	0.41	5.11
Latency REM	172.8 (76.7)	126.3 (126.4)	120.6 (56.5)	131.0 (59.5)	3.08	0.15	213.8 (113.1)	171.1 (78.4)	1.89	0.37
Wake in sleep	58.3 (46.7)	35.0 (25.0)	45.7 (25.7)	23.1 (13.9)	0.39	3.38	92.3 (89.5)	28.3 (21.3)	1.12	3.42
No. awakenings	16.7 (7.1)	20.2 (6.9)	20.2 (5.8)	16.8 (4.0)	9.09[b]	0.09	12.4 (2.6)	18.2 (9.0)	0.34	0.62
% movement	00.0 (0.0)	0.1 (0.2)	00.2 (0.2)	0.0 (00.0)	0.00	3.64	0.0 (0.0)	0.3 (0.4)	0.45	0.45
Stage changes	90.7 (18.2)	120.0 (45.4)	111.2 (20.2)	104.2 (31.7)	1.40	2.47	89.4 (31.0)	108.4 (26.5)	0.26	0.67
Sleep latency	17.6 (15.5)	14.5 (7.7)	25.7 (18.0)	16.0 (13.0)	1.36	5.49	26.4 (12.4)	9.9 (1.2)	0.16	6.65[b]
Latency estimate	82.0 (66.0)	64.0 (55.8)	67.0 (81.9)	27.4 (19.6)	7.80[b]	4.21	81.0 (80.3)	23.0 (4.0)	0.10	2.68
Sleep estimate ratio	5.5 (2.4)	3.9 (1.6)	2.5 (1.3)	2.1 (0.9)	11.81[b]	9.57[b]	2.9 (1.8)	2.4 (0.6)	0.40	0.67

[a] G1C = group 1 control; G1T = group 1 treatment; G2T = group 2 treatment; standard deviations in parentheses. Wake in sleep measured in minutes.

[b] $p \leq 0.05$.

DOWNEY & BONNET, SLEEP. 1992; 15(1): 58-63

POSSIBLE ADJUVENTS AND ALTERNATIVES

BRIGHT LIGHT THERAPY

SLEEP COMPRESSION

COUNTER CONTROL

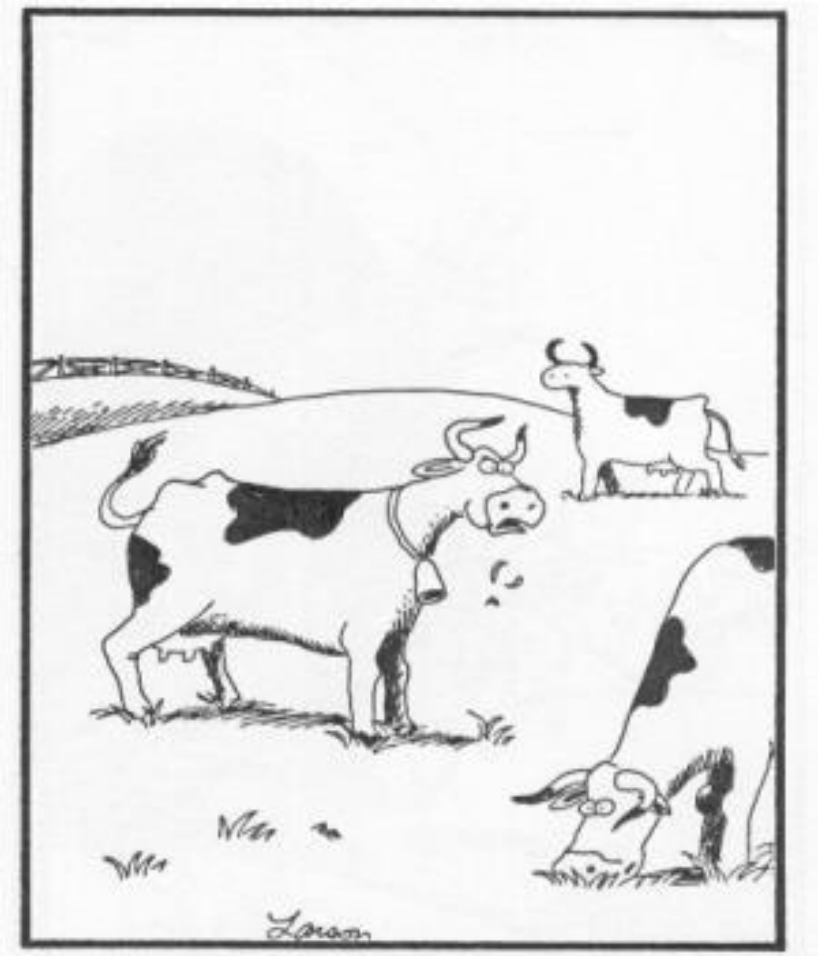
ISR

MINDFULNESS

APPLYING MINDFULNESS

- Beginners Mind
- Non-Striving
- Letting Go
- Non-Judging
- Acceptance
- Trust
- Patience

THE PROBLEM WITH NOT BEING MINDFUL



"Hey, wait a minute! This is grass! We've been eating grass!"


I...
AM...
DETERMINED...
TO...
GO...
TO...
SLEEP...

"Sleep (is like) a dove which has landed near one's hand and stays there as long as one does not pay any attention to it; if one attempts to grab it, it quickly flies away"

Viktor E. Frankl (1965, p. 253) cited in Ansfield et al. Behav.Res.Ther. 1996;34:523-531

SLIDE PROVIDED BY COLIN ESPIE

DATA

Table 2

Sleep and Waking Measures at Pretreatment and Posttreatment

Measures	Pretreatment	Posttreatment
SLEEP PARAMETERS AND NOCTURNAL SYMPTOMS		
Total wake time (min)	320.00	120.00
Sleep-onset latency	41.43	10.00
Wake time after sleep onset	278.57	110.00
Total sleep time (min)	142.89	280.71
Time in bed (min)	462.86	400.71
Sleep efficiency (%)	30.87	69.87
Number of awakenings at night	2.57	1.71
Sleep quality	1.14	4.57
Insomnia Severity Index (Bastien, Vallieres, & Morin, 2001)	22.00	12.00
Dysfunctional beliefs and attitudes about sleep	135.00	112.00

Ong & Sholtes, J Clini Psychol. 2010; 66(11): 1175-1184

QUESTIONS

BREAK