The Cognitive Behavioral Treatment of Chronic Insomnia

with some sleep physiology for background
Hello

• Thanks for coming!
• Hope this is useful!
• What fields are represented here?
• What kinds of questions do you have?

• *Much material here so will move through based on time and group interest.*
Jeffrey Young, PHD, CBSM
Psychologist
Assistant Clinical Professor
UCLA Department of Psychiatry & Biobehavioral Science
Taught Sleep course among other classes
Certified in Behavioral Sleep Medicine by the American Academy of Sleep Medicine
Certified 2008
Founding Member Society of the Society of Behavioral Sleep Medicine
On Education Committee
Learn more about this organization
No Affiliations to Disclose
What is CBSM Certification

• Certified in Behavioral Sleep Medicine by the American Academy of Sleep Medicine

• Experience and Exam Requirements
  – Mini-Fellowship in Sleep Lab
    • Observed and participated in sleep interviews
    • Observed and participated in Polysomnograms
    • Treated Insomnia cases
    • Supervised by Sleep Medicine Physician in lab as well as sustained consultation with CBSM Psychologist.
    • Read wide range of sleep literature.
  – Passed the BSM exam (4 hour exam)-2008
  – 50% of practice is sleep.
Possible ABPP in Sleep

• Not sure what the “gold” standard, if any, will be (CBSM vs ABPP). Some disagreement about where it should go.

• The sleep board given by AASM was open to both Physicians and Psychologists until 2003 when the sleep board was taken over by the ABIM (internal medicine).

• Hence, the birth of the CBSM exam. See reading list on SBSM website.
My Interest in Sleep

• As an undergraduate, I was struck by fact that the understanding of sleep while better by that time was still, per my professor, hazy.
• We spend a third of our life in this state and at some level ignore it (actually good to ignore it unless you’re interested in its function).
• Remaining interested in the area since undergraduate days and decided to pursue it more seriously in 2007.
Overview of Talk

• What does sleep look like?
• What are the homeostatic and circadian influences on sleep?
• Why does sleep exist and what does sleep actually do for us.
• What is a sleep interview?
• What is insomnia?
• How do we treat insomnia with both behavioral and cognitive approaches?
• What is the Evidence that CBT works for Insomnia
• Pharmacologic Interventions

• More on Sleep and Circadian Physiology, if time and interest permits
Main Diagnostic Points

• Insomnia is a **24 hour per day** problem that includes daytime impairment (memory/concentration/fatigue) and daytime contributors such as napping/sleep extension (if can do) and decreases in activity to compensate.

• Insomnia implies that there is **adequate opportunity** for sleep to occur. This differentiates it from Sleep Deprivation.

• You will see a lot of **anxiety** about sleep and the perceived consequences of sleep loss and “sleep effort”

• Generally, SL>30, WASO>30, EMA>30, 3pms/wkX3months for Chronic Insomnia.

• SL=Sleep Latency, WASO=Wake After Sleep Onset, EMA=Early Morning Awakening
Insomnia DSM V (APA 2013)

Summary of Symptoms -- Adaptation

– Dissatisfaction with sleep quality or quality manifesting as difficulty with Initiating or Maintaining sleep or Early Morning Awakening.

– Causes clinically significant distress (impairment in social/occupational/behavioral functioning)

– Symptoms 3x/week for 3 months (minimum)

– Adequate opportunity for sleep

– Not better explained by or occurring exclusively during the course of another sleep/wake disorder (circadian delay causing sleep onset insomnia)

– Not attributable to physiologic effects of a substance

– Coexisting mental disorder/medical conditions do not adequately account for the primary complaint of insomnia
  • Specifiers
  • Comorbidities (non sleep mental, medical, other sleep d/o)
  • Episodic (1-3 mo.) Persistent (3 mo. +) Recurrent (2/yr. +)

– Other specified insomnia disorder
  • Brief Insomnia Disorder (<3 mo.), Only Nonrestorative Sleep
The ICSD-3rd Edition
International Classification of Sleep Disorders

– Insomnias (Chronic, Short-term, Psychophysiological)
– Circadian Rhythms Disorders (Delayed, Advanced)
– Hypersomnias (Narcolepsy)
– Sleep Related Breathing Disorders (OSA)
– Parasomnias (Sleep Walking, REM Behavior D/O)
– Sleep Related Movement Disorders (RLS, PLMD)
What Sleep Looks Like

- Sleep defined Behaviorally and Polysomnographically.
- Breakdown of Sleep Cycles and Staging
- Characteristics of NREM and REM sleep.
- Sleep onset and the first sleep cycle.
Sleep Defined Behaviorally

• *We know what this looks like...*
  • Postural Recumbrance (lying down)
  • Behavioral Quiescence (not moving much)
  • Closed Eyes

• *Also...*
  • Easily Reversible Behavioral State (vs. Hibernation)-Unless you’re 4 y/o
  • Relative *Perceptual Disengagement* from and Relative Unresponsiveness to the Environment

• *Sleep observed this way suggests that sleep is a unitary state. It is not as we will see.*
Perceptual Disengagement

- *Disengagement is on a continuum.*
- Lower threshold for one’s own name.
- Lower threshold for a baby’s crying
- That is, one can conceivably sleep through sirens and street noise but be quickly aroused by the examples above.
- Perceptual Disengagement may not be as sound in Persons with Insomnia (PWI)
Polysomnographic Sleep

• Based on the laboratory polysomnogram (PSG). Common diagnostic tool for sleep related disorders.

• Based on Electroencephalographic Tracings of Brain Wave Patterns

• Electromyographic patterns derived from the muscle tone of the Chin and the Movement of the Eyes.

Most referrals to sleep labs are for sleep disordered breathing. SDB hallmark is daytime sleepiness via sleep fragmentation (arousal from apneic events. Measures: Airflow, Respiratory Effort, O2 Sats, EEG (Staging, Seizures, Arousals) Chin Muscle tone (REM), Anterior Tibialis (PLMS)
Three States of Consciousness
Sleep States

• There are 2 Sleep States
• **REM** *(Rapid Eye Movement)*
• **NREM** *(Non Rapid Eye Movement)*
• These are as distinct from each other as they both are from WAKE
• Including WAKE, this translates into 3 states of consciousness in which we humans and all terrestrial animals can exist.
• Why NREM and not better name? Originally thought that REM (dreaming sleep) was the “Royal Road” so everything else was kind of superfluous.
Breakdown of Sleep Cycles

**Basic Breakdown**

- NREM (Percent)
  - Stage 1 (2-5)
  - Stage 2 (45-55)
  - Stage 3 (3-8) SWS
  - Stage 4 (10-15) SWS
- REM (Percent)
  - REM (20-25)
- Wake (Percent)
  - Wake (5)
- Most SWS First 1/3 of Night
- Most REM Third 1/3 of Night

**Sleep Cycle**

- 8 hours of sleep=480 min.
- Stage 1=10-24 min.
- Stage 2=216-264 min.
- Stage 3=14-38 min.
- Stage 4=48-72 min.
- SWS=62-110 min.
- Stage REM=96-120 min.
- Wake=24 min.
- About 5 NREM/REM Cycles 60-90 min apart per night
Characteristics of REM

- EEG activation - similar pattern as AWAKE.
- Vivid dreaming with generally detailed recall.
- Vivid dream recall in 80% aroused from REM
- Muscle Atonia with some twitching.
- Cardiac and Respiratory muscles intact.
- Cardiorespiratory irregularities.
- **Phasic REM**: Episodic bursts of REMS
- **Tonic REM**: Relative quiescence – the space between bursts of REMS
- **Shorthand**: Activated brain in a paralyzed body
Characteristics of NREM

• Associated with minimal or fragmentary mental activity.

• Dreams can occur in NREM but generally are reported as quite vague. “I think I was in someplace that looked like a bank…not much else”

• **Shorthand**: Relatively inactive but actively regulating (stuff is going on) brain in a moveable body.
Depth of Sleep

• Generally speaking moving from Stage 1 onward to Stage 4 sleep represents a continuum of sleep depth with *arousal thresholds* typically lowest in Stage 1 and highest in Stage 4.

• This may correlate with a patients complaint of “I never seem to get into a good deep sleep” or “I felt like I was hovering all night”.

• Boils down, at a level to *arousability*, which is part of actual and perceived sleep quality.
Overview of Function

**NREM**

- It appears that the lower metabolic rate and brain temperature occurring during NREM sleep provides an opportunity to deal with waking oxidative damage.

**REM**

- The interruption of monoamine release during REM may allow the receptor system to rest and regain full sensitivity and may be critical for mood regulation.

Two-Process Model of Sleep Regulation (Borbely, 1982)

• Process S: Homeostatic drive for sleep
  – More S=More Sleepiness

• Process C: Circadian variation in alertness
  – More C=More Alertness

• Process S and C interact to produce any given level of **Sleep Propensity**
How Much Sleep Does One Need?

- The average sleep need is about 7-8 hours
- Sleep need varies considerably from person to person and is likely genetically determined – So Non Pathologic Short (6hrs) & Long (9)
- However: Sleep<5-6 or >10 liked to poorer health
- 7 Hours, however, has been linked to greater health.
- **Overall, if you are alert, energetic, and feel good during the day, you are likely getting enough sleep**
Introduction to Behavioral Sleep Medicine
Behavioral Sleep Medicine
From Perlis & Lichstein *Treating Sleep Disorders* (Chap. 1 Stepanski & Perlis)

• Formally, behavioral sleep medicine, (BSM) refers to the branch of clinical sleep medicine and health psychology that:
  
  – 1. Focuses on the identification of the psychological (e.g., cognitive and/or behavioral) factors that contribute to the development and/or maintenance of sleep disorders.
  
  – 2. Specializes in developing and providing empirically validated cognitive, behavioral, and/or other non-pharmacologic interventions for the entire spectrum of sleep disorders.
Disorders Treated with BSM

• Insomnia: (CBT-I Package)
  • Primary, Secondary, Co-morbid
• Circadian Rhythm Disorder (Light, Melatonin)
  • Advanced, Delayed
• Narcolepsy (Scheduled Naps)
• Sleep Apnea (Exposure for Claustrophobia, Education to improve Adherence, Positional Therapy to Avoid Supine Position)
Historical Context

Insomnia
Brief History

• From the beginning there was an appreciation for the role of physiologic arousal. This was initially addressed by Edmond Jacobson who wrote *You Can Sleep Well* (1938) and who developed Progressive Muscle Relaxation as a way to decrease physiological arousal which was assumed to manifest, in part, by increased muscle tension.

• This is a common presentation in psychophysioptic insomnia where the patient is utterly exhausted but mentally and physically aroused. The role of physiologic arousal continues to have support (see neuroimaging) but arousal levels need not be off the chart to have an impact (as they once might have been)
Brief History-More Problems

- As the world became more industrialized there was also the growing understanding that the demarcation of night and day was to become increasingly blurred. The advent of electric lighting was a major advance that would extend light exposure, wakefulness, and the potential for both work and play.

- This could lead to both circadian dysfunction (poor entrainment) and/or poor stimulus control.

- Today, the plot thickens. 24 hour markets, internet, the ability to work and study late into the night very effectively.
Insomnia as an Independent Disorder

• This is a very important transition.
• Insomnia transitioned from being regarded as just a symptom secondary to a primary psychiatric or medical disorder to a focus of independent treatment.
• Influenced the Conceptual Modeling of insomnia and its treatment and the led to an appreciating of the importance of *treating the insomnia itself aggressively* even if it was arguably part of another disorder.
• Affirmed that insomnia could be a disorder that could develop “a life of its own” and outlast the primary disorder whether it be medical, psychiatric, or situational.
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Important Concepts

• Fatigue versus Sleepiness
• Rest versus Sleep
Fatigue versus Sleepiness

• **Fatigue**: You want to take a break, you experience some form or mental or physical weariness, you take a load off and feel better. Taking that load off doesn’t lead to a high probability of sleep.

• **Sleepiness**: All of the above but taking a load off does lead to a high probability of sleep.

• *Sometimes it’s hard to appreciate the difference, unless you’ve had the experience of being utterly exhausted and sleep will not come-* ”Wired but Tired”.*
Rest versus Sleep

- Resting and Sleeping are very different.
- Rest can feel good and can restore energy but only sleep can confer the benefits of sleep.
- Confusion regarding this concept often leads people to assume that laying in bed resting can confer some of the benefits of sleep – as if rest could be a good state of compromise between actual sleep and waking activity.
- Should address this directly with your patient to disavow the idea that resting in bed has some value. Should emphasize that not only does it have little or no value but leads to conditioning wakefulness in the bedroom.
What’s out there

• General estimate of 10% of adults having chronic insomnia. Unremitting.
• General estimate of 25% in older adults.
• Twice as common in women post age 30
• Major Comobidities: Depression, Anxiety, Substance Abuse, Dependence on Hypnotics.
Questions to Ask
Insomnia History

- Sleep Problems in Parents, Siblings
- Sleep as a Child, Adolescent, Young Adult
  - Solid sleep up to current episode a good sign
- Insomnia (Chronic, Episodic)
  - Precipitating Factors
    - Changes in health, medication, relationships
    - If forget precipitating event clue to chronic form
  - Coping (laying in bed, sleep extension)
  - Treatment (OTC and Rx), Psychological
  - Quality of sleep before
Current Sleep Schedule

- Time to Bed (TTB)
- Lights Out (LO)
- Sleep Onset Latency (SL)
- Number of Awakenings (NWAK)
- Wake after Sleep Onset (WASO)
- Time Out of Bed (TOB)
- Total Sleep Time (TST)
- Anything that predicts poor vs. good night
- Sleep better away from home (conditioning?)
- Night Owl vs. Morning Lark (by nature)
  - Work and other obligation aside when would be the best time for sleep for you? May find that current sleep schedule is out of phase (delayed or advanced) with preferred sleep schedule.
Sleep Hygiene

Caffeine (Coffee, Soda, Energy Drinks, Smoking?
Alcohol use other Drug use?
Exercise: What type and when? Regular?
Napping and when?
Eating regularly timed meals? Goes to bed hungry/too full?
Excessive liquids (nocturia)? Too many Rx meds before bed?
Bedroom activities
   - Sleep Effort, Worry, Tablet, TV, Reading, Work,
   Heavy discussions, Arguments?
Get up at the same time each day, 7 days a week?
Comfort: Bed, Bedroom, Light, Noise, Too hot/cold?
Clock Watching, Wake with alarm?
Other Sleep Disruption? Snoring, pets, kids?
Other Sleep Disorders

• Have you had a PSG?

• Snoring, Gasping, Morning Headache
  – Bed Partner Input

• Restless legs (look at movements of legs/feet)
  – Crawling sensations, pin & needles, urgency to move/rub to relieve (interfere with initiating sleep and associated PLMS can fragment sleep)?

• Excessive Daytime Sleepiness

• Sleepwalking, Sleep Eating, Acting out Dreams

• Attacks of Sleep, Sudden Muscular Weakness usually provoked by emotion (fear, laughter)
Daytime Function

- Poorer Memory, Concentration?
- Mood Changes: Depressed, Anxious, Irritable?
- Sleepiness (Epworth), Fatigue (Fatigue Scales)?
- Work, School Performance Lower?
- Worry about sleep during the day?
- Do symptoms vary with sleep quality/duration
  - *Ever fall asleep while driving?*
Medical and Psychiatric

• Medications currently: Rx, OTC, Recreational
• Review of Current Medical/Psychiatric d/o
• Depression, Anxiety, PTSD, Drug Abuse, Other Sleep d/o, Pain, Fibromyalgia, MS
  – Secondary or Comorbid
  – Vary with psychiatric or medical condition
Selected Models of Insomnia

Stimulus Control Model (Bootzin, 1972)
Stimulus-Response. Operant Conditioning

Spielman Model (Speilman, 1987)
Predisposing, Precipitating, & Perpetuating Factors

Four Factor Model (Perlis et. al 2005)
Extension of Spielman Model Classical Conditioning

These are the most common conceptualizations.
### Stimulus Control Model - Bootzin

<table>
<thead>
<tr>
<th>Good Stimulus Control</th>
<th>Poor Stimulus Control</th>
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<tbody>
<tr>
<td>Frustration</td>
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<tr>
<td>IPAD</td>
<td>Sleep</td>
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<td>Sleep</td>
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<td>Sex</td>
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<td>TV</td>
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<tr>
<td>Worry</td>
<td>Worry</td>
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<tr>
<td>Work in Bed</td>
<td>Work in Bed</td>
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</table>

**Basic Premise:** Elimination of nonsleep activities will tighten the association of bed, bedroom, bedtime with rapid sleep onset and satisfying sleep.

**Keep Conditioning History Simple!**

**Maintain Stimulus Value of the Bedroom!**
How to think about stimulus control

• It is clear that one stimulus can evoke a number of responses.
• However, if a particular stimulus is generally paired with only one response then that stimulus is likely to evoke the same response. (i.e., set of behaviors, emotions, physiologic reactions). This would be a discriminant stimulus.
• The opposite is true as well: A stimulus that is effectively muddied by having the capacity to evoke a number of responses is not very discriminating.
• Good stimulus control extends beyond conditioning principles by providing homeostatic and circadian structure (i.e., getting up at the same time). That is, eliminating resting in bed and associated Microsleeps increases homeostatic sleep pressure and eliminating sleep extension by enforcing a set wake time enhances circadian entrainment.
• Reinforcers are rapid sleep onset and consolidated, refreshing sleep.
The Spielman Model of Insomnia

**Predisposing:** Genetics, Family History, Propensity for Cognitive and/or Physiologic Arousal

**Precipitating:** Stressful life event, Illness/Injury, Pain, Bereavement, Psychiatric Disorder

**Perpetuating:** Extending Sleep Opportunity, Laying in bed awake, Napping, Variable Sleep/Wake Schedules, Diminished Daily Activity secondary to Fatigue - Drains Sleep Homeostat

**Acute Insomnia:** Initiated and Maintained by Predisposing & Precipitating Factors.

**Early Insomnia:** Maintained by Perpetuating Factors and in small part Precipitating Factors.

**Chronic Insomnia:** Precipitating Factors are effectively gone. Perpetuating Factors remain
Spielman Model

• Stress-Diathesis Model that explains how acute insomnia becomes chronic.

• **Insomnia occurs acutely in response to:**
  • **Predisposing** factors
    - Hyperarousal (increased startle and startle recovery)
    - Increased metabolic rate (increased cortisol & body temp.)
    - Propensity to rumination and worry
  
  **Precipitating** factors
  Medical, Psychiatric events, Novel and/or High Intensity Stressors

  **It is maintained by**
  
  **Perpetuating** factors
  *These are faulty strategies that are used to cope with sleep loss.*
  • Extending sleep opportunity, staying in bed awake “trying to sleep”, trying to recover sleep at times when sleep ability may be low due to homeostatic and circadian factors.
Classical Conditioning Effects

• Stimulus control model and the Spielman Model address the operant/instrumental side of the learning process; that is, the way that behaviors feed the insomnia.

• We need another mechanism that can explain why insomnia can lead to a life of its own; that is, why the insomnia remains long after the precipitant is gone. We also need a mechanism to explain why patients in CBT-I continue to improve several months post active treatment.

• The Model that explains this is classical conditioning. Laying in bed for long periods of time “trying to sleep” can be frustrating and activating and over time can lead to the bed and bedroom to become a conditioned stimulus for arousal.
This model is an extension of the Spielman Model but includes a 4th factor which is Conditioned Arousal. Conditioned Arousal is another overlay that maintains insomnia in its chronic form. It the Classical Conditioning side that is created by repeated pairing of the Bed and Bedroom with Sleep Effort and Frustration creating it into a conditioned stimulus for arousal. This arousal also diminishes with treatment and continues to diminish post-treatment.
How the Conditioning Works
Psychophysiological Insomnia

• Before you had insomnia, the bedroom was either a neutral stimulus or a stimulus that evoked relaxation and/or sleepiness.

• Then the stress hit. You worried in bed, laid in bed trying to sleep, or laid there hoping that the rest conferred would be better than nothing. Soon enough, you weren’t just worried about the stressor that started it all but were worried that you might have lost your ability to go to sleep naturally. This became compounded when you came to the horrible realization that if you didn’t sleep you would even have less chance of dealing effectively with that stressor. Maybe job performance would suffer as well adding into the mix of troubles.

• Some time has passed now. The original stressor has faded or may have completely resolved. That’s great, except for the fact that over the past couple of months the bedroom, once neutral or calming, has been a source of great stress. The bedroom (the stimulus) has been repeatedly paired with stress manifesting as cognitive arousal (worry) and physiologic arousal (muscle tension, increase metabolic rate). Now after all is much better, your still stuck with not sleeping well because the bedroom is now a conditioned stimulus for arousal in the absence of “real threat”. After a while Pavlov’s dogs didn’t need a “real” (unconditioned stimulus) to salivate – all is takes now is a bell – or in this case a bedroom.
Components of CBT-I

What to do

• Sleep Hygiene
• Stimulus Control
• Sleep Restriction
• Relaxation & Cognitive Restructuring
• Addressing Sleep Phase
Contraindications for CBT-I

- Patients unwilling/unable to comply
- Ongoing Drug and/or ETOH Abuse
- Acute Episode of an Illness
- Untreated/Unstable Bipolar d/o

- Specific modifications are often possible and SOME behavioral treatments may be appropriate.
First the Sleep Diary

• Sleep Diary is used to track sleep patterns to establish baseline and treatment progress.
• This is subjective at a level as compared to Actigraphy or PSG.
• You give your best estimation of the variables
  • Time to Bed (TTB)-Note Time
  • Lights Out (LO)-Note Time
  • Sleep Onset Latency (SL)
  • Number of Awakenings (NWAK)
  • Wake after Sleep Onset (WASO)
  • Time Out of Bed (TOB)
  • Total Sleep Time (TST)
  • No clock watching – Complete in the morning
Sleep Hygiene

Sleep only as much as you need to feel refreshed during the following day. Limit bed to sleep and sex.
Don’t bring problems to bed and don’t “try” to sleep”
Get up at the same time each day, 7 days a week.
Bedtime dictated by Sleepiness not Clock Time
Exercise regularly. Preferably 4-5 hours before bed.
Make sure your bedroom is comfortable and free from light and noise.
Make sure that your bedroom is at a comfortable temperature during the night. Cooler is often better.
Eat regular meals and do not go to bed hungry.
Avoid excessive liquids in the evening.
Avoid Alcohol, Caffeine, and Smoking, Naps, Clock-Watching
Wake with Alarm
More about Sleep Hygiene

Beware of patients getting sleep hygiene instructions from the PCP or internet, trying some ideas that did not work well and concluding that they failed behavioral treatment.

No evidence that Sleep Hygiene alone is an effective treatment.

Watch for perfectionism when it comes to Sleep Hygiene “rules” as it will have some regarding their sleep as so fragile that they dare not break a “rule”.
Stimulus Control Treatment

- **Rationale:** Establish and build the connection between bedtime, bedroom, and bed with rapid sleep onset and proper (desired) nighttime circadian entrainment.

- Bed only when sleepy not just fatigued.
- Leave bedroom if unable to sleep within 20 min or at first sign of frustration.
- Return to bed with first wave of sleepiness.
- Establish a fixed wake time that is independent of the amount of nighttime sleep obtained.
- Bed for sleep and sex only.

- **Bad habits develop as an initial response to insomnia and decrease the stimulus value of bedtime, bedroom, and bed. This can happen outside of awareness so the bedroom is insidiously transformed from sleep friendly to a cue for arousal, anxiety, and wakefulness.**

- **Needs very strict compliance in many cases.**
More about Stimulus Control

• Hardest thing to ask someone to do is get out of bed in the middle of the night. But must take sleeplessness out of the bedroom and avoid the microsleeps that come with staying in bed that will ultimately drain too much sleep pressure and lead to “thin sleep”.

• This is stimulus control but also an Exposure exercise as well. You need your patient be sleepless until really sleepy but you also need him to know that he can survive sleeplessness which is likely his major fear.

• When out of bed do something enjoyable but in low light.

• Beware of getting up once and concluding it doesn’t work. A limited trial only makes things worse and ruins compliance.

• Avoid the pressure of chasing sleep into the bedroom.
Sleep Restriction

- **Rationale**: Limiting time in bed to average time slept will increase homeostatic sleep pressure and consequently lead to deeper, less fragmented sleep.

- Time in Bed (TIB) = 9 hours
- Total Sleep Time (TST) = 5 hours
- Sleep Efficiency (SE) = TST/TIB = 5/9 = 55% so Time to Bed (TTB) is now Time out of Bed (TOB, say 7am) - 5 hours leaving a TTB of 2am instead of, say, 10pm (to 7am).

- We like to see sleep efficiency at 85%-90% and will bring bedtime earlier by 15-20 minutes q 4-7 days until target is reached.

- Very difficult “buy in” and will need to discuss safety and possible use of stimulants.
Sleep Restriction

• Want to assess for motivation and timing. That is: How determined is the patient to stick to the program and is this an opportune time to engage in a process that might lead to an increase in sleep deprivation and reduced performance.

• Want to have at least 1 week and preferably 2 weeks of sleep diaries.

• The clinician will need to calculate the relevant variables as averages. For example, Sleep Latency and Wake after Sleep Onset can be averaged over the period assessed. Similarly, the amount of sleep obtained is averaged over the period assessed.

• All of the variables are calculated in the service of determining how much time was spent in bed and how much of that time was spent sleeping.
Sleep Restriction Further Rationale

• If you are only restricting the patient to the actual time slept and no further, why would there be a concern about sleep deprivation and reduced daytime performance. After all, the patient is getting the same amount of sleep.
• Yes, in theory, the patient is getting the same amount of sleep but remember the amount of sleep is calculated via self-report and generally not via actigraphy and almost never via PSG (objective measures).
• Also, the likelihood of experiencing microsleeps while resting in bed or while waiting for sleep to come is quite high. That is, someone with a sleep latency of 2 hours may not be sleeping soundly at all during that time but may be getting short episodes of sleep that do add up and that do serve to reduce sleep pressure.

_People with insomnia tend to underestimate the amount of time actually slept. Sometimes the difference can be quite large so when you give them a sleep opportunity in line with their self-report, it is likely that this sleep opportunity will serve to actually restrict the amount of time available for sleep._
Sleep Restriction Example Pt. A

- 2 weeks of sleep diaries indicate the following averages:
  - Time to Bed (TTB)-11:00pm - Reading, TV, Trying to Relax
  - Lights Out (LO)-11:30
  - Sleep Onset Latency (SL): 1.00 Hour
  - Number of Awakenings (NWAK): 2
  - Wake after Sleep Onset (WASO): 1.50 Hours (for 2 awakenings)
  - Early Morning Wake (EMW): 1.00 Hour
  - Time Out of Bed (TOB): 8:00am

What this means: Of the 9 hours of Sleep Opportunity only (9hours-4hours) or 5 hours were spent sleeping. Sleep Efficiency is 5/9=55%

We like to see sleep efficiencies at 85% to 90%.

So, let’s restrict sleep opportunity to 5 hours and give a new TTB of 3am.
Sleep Restriction Example Pt. A

This person will have the opportunity to sleep roughly the amount he is sleeping now. A bedtime of 3am and a wake time of 8am will allow for a 5 hour opportunity for sleep. We would expect that the first night would provide a great amount of sleep pressure. If this patient complies, he will likely have enough sleep pressure to fall asleep fairly quickly and remain asleep fairly well until the 8am alarm. If he is able to maintain a sleep efficiency of 85% over 4 days, he can add another ½ hour of sleep opportunity (i.e., go to bed ½ hour earlier). The plan is to upwardly titrate sleep opportunity and to finally arrive at about 7-8 hours (depending on actual sleep need) with an efficiency of at least 85%.
More about Sleep Restriction

• We do this to press on the sleep homeostat.
• Like Stimulus Control you are likely to encounter at least some resistance.
• Your patient will feel worse before better.
• Contraindicated in Bipolar (Mania), Seizure Disorder, Parasomnias, and people who cannot avoid driving long distances.
• Continually assess for Sleepiness (Epworth)
Final Points on Behavioral Side

• It’s important to emphasize that what we are doing is not about tonight, tomorrow night, or even next week – it’s the big picture. You have had insomnia for 10 years. It does not need to get fixed in a week.

• Discuss with your patient if this is good time for behavioral treatment. Some prefer to engage in treatment on time off so that they don’t have to worry about driving or meeting obligations.
In the Beginning…

What do you do right at the onset of an episode of insomnia?
Best Answer

Nothing
The System Will Auto-Correct

• Do not engage in sleep extension.
• Do not lay there trying to sleep.
• Do not let worry creep in.
• Just let it be and there is a good chance that the increase in sleep pressure from a couple of bad nights will create some good nights and relieve worry and tension.
• *If the ship does not right itself*, if there is no correction, then attention from a sleep specialist should be sought. This is a time for stress management and/or the short term use of hypnotics or behavioral interventions.
Relaxation & Cognitive Restructuring

- Autogenic Training – Focus on Parts of Body as Both Heavy and warm - Can combine with Breathing Exercises
- Diaphragmatic Breathing – Mimics the kind of breathing that occurs at sleep onset. Abdomen should be moving and chest still. Induction of Relaxation. Reduction of Arousal
- Limit and challenge catastrophic thinking about sleep loss.
Examples of Cognitive Distortions

**All-or-Nothing Thinking:** “I need 8 hours each night or I won’t be fully rested.”

**Overgeneralization:** “I’ll never learn to relax”

Sleep difficulty is always followed by negative consequences

**Discounting the Positive:** “It doesn’t count that I was able to sleep well last weekend when I was away from home” or “that I slept really well before my insomnia”

**Catastrophizing:** “If I don’t get this insomnia fixed soon, I will lose my job – lose my house – family will be devastated.”

Can counter this by looking at how many nights a person has had insomnia and looking the Actual Consequences vs. Feared Consequences.

*Both Stimulus Control and Sleep Restriction can function as an Exposure Exercise. You can survive!*
Cognitive Pearls

People with insomnia tend to want to control the uncontrollable. **Sleep Effort** “Trying to sleep” is useless and counter productive

Often the most helpful thing to drive home is that “you cannot find sleep, sleep will find you (and it will always find you). Let go.

Paradoxical Intention: Try not to fall asleep—maybe not good for those with a lot of cognitive rigidity.
Cognitive Pearls

• Worry and Rumination can cause sleeplessness but being disengaged in bed in itself can be the perfect place to foster Worry and Rumination.
• It’s bad to awake when reason sleeps. That is, in this state ones ability to cope with stress is impaired so problems look bigger.
• Can’t stop your thinking but can disconnect it from the bed and bedroom.
Cognitive Pearls

• Give up Sleep Effort it cannot work. Laying in bed and finally getting to sleep after a long frustrating process is more happenstance and inefficient.

• PSG will show that laying in bed will produce microsleeps that will drain sleep pressure and reduce the possibility of good sleep (thin sleep). If your going to be effectively up for 3 hours then really be up for 3 hours and really build pressure (thick sleep).

• Don’t have to have your eyes closed (sets up tacit expectations). Sleep will close them
Cognitive Pearls

• Be wary of relaxation and meditation transforming itself into sleep effort. Do these things but possibly outside sleep window if it becomes problematic.

• Light stretching can help dissolve some physiologic tensions.

• Wake self identified good sleepers out of Stage 2- ”I was Sleeping”, Poor Sleeper “I was Awake: Evidence Increase in Perceptual Engagement- Maybe Cognitive Bias-Need to Tread Lightly on this so as not to diminish perception
Insomnia and the attribution process.


Subjects with Insomnia were given placebo pills to take a few minutes before going to bed.

-Condition 1: Subjects told that the pills would cause arousal.  
  **Arousal Condition**

-Condition 2: Subjects were told that the pills would reduce arousal.  
  **Relaxation Condition**

**Results**

Arousal Condition subjects got to sleep more quickly than usual. This group attributed their arousal to the pill and not to themselves and consequently became less emotionally activated. They could say:

“It’s just the pill – no need to worry”

Relaxation Condition subjects got to sleep less quickly than usual. This group likely felt that their emotions were unusually intense because their level of arousal was high even though they were given a pill to reduce arousal. They could say:

“I must really be out of sorts if I’m still screwed up even with this pill”.

The more you get caught up in your negative experience the more you are likely to suffer and the more you attribute this negative experience to something about the self the more likely you are to attend to it in an ineffective way.

Trying to sleep and trying to relax – **Sleep Effort** – instead of acting like a person who doesn’t care about sleep.
Woke subjects 10 minutes after the first sleep spindle (an indicator of unequivocal sleep) and asked if subject was awake. Poor sleepers were more likely to report that they were awake compared to good sleepers indicating that poor sleepers can misperceive sleep as wakefulness.

Again need to tread carefully on this in a clinical context so as not to come across as “it’s all in your head” even thought it may be.

Also, again, may have some physiologic basis in this so it may be “in their head” but out of their control.

- People with Insomnia versus Good Sleepers

- Woke 5 minutes after first Sleep Spindle

- People with Insomnia more likely to report having been awake the moment just before awakened compared to good sleepers.

- This Finding was confirmed at regular nighttime sleep periods as well as during afternoon naps.
Comorbid Insomnia and Secondary Insomnia
Comorbid Insomnia
Rybarczyk el al (2009, Sleep Med Clinics)

• Strong empirical support for regarding comorbid insomnia as a functional equivalent to primary insomnia.

• General understanding that relief from insomnia can improve coexisting medical and psychiatric conditions and daytime functioning (Disturbed sleep can lower pain threshold and can be a risk factor for depression).
Comorbid Insomnia

• There is a very strong argument for treating comorbid insomnia aggressively not only because of its potential to exacerbate an underlying condition but because it is clear that insomnia can often maintain “a life of its own”.

• This means that the insomnia can outlast the remission of the disorder or stressor due to the conditioning effects of bad habits (e.g., sleep extension, laying in bed awake).

• Research has shown Efficacy for CBT-I across many comorbid disorders including pain and cancer.
Secondary Insomnia

• If insomnia is found, let’s say, in Anxiety or Depression, or Pain then it can be reasonably considered secondary to the primary disorder.

• Of course, if the Insomnia improves in concert with the improvement of the primary disorder then the “secondary hypothesis” is upheld and all is well.

• However, concurrent treatment can be helpful and save someone from developing an Insomnia Disorder.

• Remember Perpetuating Factors and Insomnia having a life of its own.
Efficacy Research Findings for CBT-I
Effectiveness of CBT-I

Update on Insomnia 2006 (Richardson), PPSM 2010 (Morin)

• When effect sizes are translated into percentile ranks the data indicate that 70 to 80 percent of patients benefit from CBT-I.

• Effect sizes are comparable to those obtained for Benzodiazepine-Receptor Agonists with a modest advantage for CBT-I on measures of SOL and Sleep Quality. Pharmacotherapy demonstrated a modest advantage for TST.

• Efficacy is also demonstrated in older adults with associated medical and psychological comorbidities.

• CBT-I alters sleep perception as measured by sleep diary data and is improves objective EEG defined sleep continuity.

• Provides modest increase in stage 3,4 sleep with minimal other affect on sleep architecture.
Effectiveness of CBT-I
Update on Insomnia 2006 (Richardson), PPSM 2010 (Morin)

• General Expectations
  – The average patient with chronic insomnia (often exceeding 10 years duration) can expect to decrease SL and WASO by about 50% with absolute values of those parameters falling below or near the 30-minute cutoff criterion that was initially used to diagnose sleep onset and/or maintenance insomnia.
  – In addition to changes in sleep-wake parameters CBT-I demonstrates improvements on sleep quality, life quality, fatigue, psychological symptoms such as anxiety and depression. Active research area.

• CBT-I demonstrated that its most potent benefit was superior durability of clinical gains once treatment was terminated.

• This is a very robust finding and points to classical conditioning effects that are further reinforced post treatment. 4 Factor Model upheld
Practice Parameters for the Psychological and Behavioral Treatment of Insomnia: An Update.

- **3.6 Cognitive behavior therapy, with or without relaxation therapy, is effective and recommended therapy in the treatment of chronic insomnia.** [4.2, 4.6] (Standard)
- **3.1 Psychological and Behavioral Interventions are effective and recommended in the treatment of chronic primary insomnia.** [4.2] (Standard)
- **3.2 Psychological and Behavioral Interventions are effective and recommended in the treatment of secondary insomnia.** [4.3, 4.4] (Standard)
- **3.3 Stimulus Control therapy is effective and recommended therapy in the treatment of chronic insomnia.** [4.3, 4.4, 4.5] (Standard)
- **3.4 Relaxation Training is effective and recommended therapy in the treatment of chronic insomnia.** [4.6] (Standard)
- **3.5 Sleep Restriction is effective and recommended therapy in the treatment of chronic insomnia.** [4.2, 4.4, 4.6] (Guideline)
“When these cognitive methods have been added to the behavioral methods to compose a CBT package, it has been found to be as effective as prescription medications are for short-term treatment of chronic insomnia. Moreover, there are indications that the beneficial effects of CBT, in contrast to those produced by medications, may last well beyond the termination of active treatment. There is no evidence that such treatment produces adverse effects, but thus far, there has been little, if any, study of this possibility.”
Pharmacotherapy
Pharmacotherapy

- Most widely used treatment even though CBT-I has demonstrated similar efficacy and superior durability.
- Barbiturates gave way to the Benzodiazepines (GABA_A agonists) in the 1960’s and 1970’s as they were safer in overdose and less additive. They are effective in the short term but Benzos have problems including a substantial reduction in SWS (non nutritious sleep – Perlis), tolerance, rebound insomnia in shorter acting formulations, and carry-over sleepiness in longer acting formulations.
- The newest drugs are the BZRA (“Non-Benzo”) which appear to have negligible affects on sleep architecture and less potential for abuse as they do not confer the level of anxiolytic and myorelaxant effects as traditional benzodiazepines. More favorable side effect profiles except for reports of increased in Parasomnia activity.
Pharmacotherapy

• FDA guidelines indicate most Hypnotics for very short term use but you will find people who have been using them for years. Lunesta is approved for 6 months and may be longest time indicated.

• Hard to know how much is placebo vs. real clinical effects.

• Hard to get people to titrate down with their prescriber.
Pharmacotherapy Cont.

Michael Perlis, PH.D.

Basic Premise: We may not know the real capabilities of hypnotics because of the way they are used.

Infection Model vs. Chronics Pain Model

We prescribe hypnotics based on the Chronic Pain Model when we should be using the Infection Model. That is, we should dose QHS for a well-reasoned and specific period of time (e.g., one month) and then titrate down and evaluate. Instead, we tell people to just take it when you need it (which is very hard to determine) like we do generally in pain. Also, Variable Reinforcement Schedule can increase usage.
Melatonin

• Not really effective for insomnia if dosed at bedtime – not a potent hypnotic
• The function of melatonin is to tell an animal that it is dark and do what you do when it’s dark which may be to sleep (diurnal animals) or be active (nocturnal animals).
• Needs to be dosed in synchrony with preferred sleep period-about 3 hours before habitual bed.
• May have some placebo value.
• Not harmless-Can affect Reproductive System