

Sleep Restriction Therapy

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PROTOCOL NAME

Sleep Restriction Therapy (SRT).

GROSS INDICATION

Sleep restriction therapy is indicated for the treatment of insomnia, including trouble sleeping during the beginning, middle or end of the time spent in bed [1].

SPECIFIC INDICATION

SRT is indicated for sleep difficulties in which the subjective sleep efficiency (sleep time/time in bed \times 100%), based on a 1- to 2-week sleep log or retrospective report, is less than 85 percent (or less than 80 percent in older individuals).

There are also individuals who exhibit relatively high sleep efficiency and yet remain amenable to SRT. For example, there are those who do not get enough sleep on weekdays because they wake up too early. Having learned through experience that if they do resume sleep, it will be right before their alarms ring, they just end the night and get out of bed after, say, 5 hours. However, on non-workdays these individuals tend to stay in bed long enough to fall back asleep and sleep late into the day. Averaged across the entire week their sleep efficiency may still be above 85 percent; nonetheless, such sleepers can benefit from SRT.

There is no systematic evidence that SRT is the treatment of choice for a particular insomnia diagnosis (e.g., psychophysiological vs idiopathic vs paradoxical insomnia).

CONTRAINDICATIONS

The increased sleep propensity produced by SRT (especially at the start of treatment) will make patients sleepy. Therefore, individuals who need to maintain optimal vigilance to avoid serious accidents should not engage in SRT. For example, long-haul truck drivers, long-distance bus drivers, air traffic controllers, operators of heavy machinery, and some assembly-line workers would be placed at unacceptably increased risk because of the sleepiness produced by SRT. Similarly, individuals with conditions that are exacerbated by sleepiness or deep sleep, such as epilepsy, parasomnias, and sleep disordered breathing, should not engage in SRT.

Individuals who fall asleep quickly and have short, compact sleep prior to a terminal early morning awakening (even on non-workdays and holidays) are unlikely to benefit from SRT. In these cases restricting time in bed will (1) not reduce sleep latency, (2) not reduce the number or duration of awakenings, and (3) not likely increase the duration of sleep. Judgment will be necessary when patients report that they stay in bed “completely awake” just to rest. The ability to perceive sleep is imperfect (sometimes to a significant degree, as in paradoxical insomnia, previously called sleep-state misperception), and individuals may be unaware that they are getting some sleep after the major sleep period. In cases where some light or unappreciated sleep does occur at the end of the night, SRT may be of benefit.

Some individuals may be very sensitive to the side effects of SRT and therefore find the restrictions too demanding. Intolerance may develop to even a short period of fatigue, sleepiness, memory impairment, irritability, or diminished concentration. Despite the likelihood of improved sleep depth and efficiency at the start of SRT, this intolerance of daytime deficits may preclude adherence to SRT for a sufficient duration to consolidate gains.

RATIONALE FOR INTERVENTION

One of the most reliable ways to strengthen the homeostatic sleep drive and thereby increase the propensity for sleep during upcoming nights is to limit the amount of sleep currently being accumulated [2,3]. Restricting time in bed over a number of nights is a simple way to limit sleep accumulation. Sleep restriction also redresses such indicators of poor sleep as elevated amounts of light Stage N1 sleep, prolonged sleep latencies, and excessive wakefulness after sleep onset. Rapid sleep onset and a well-consolidated night of quality sleep, core goals of insomnia treatment, are achieved rapidly and reliably at the start of SRT. However, other treatment objectives are deferred, such as accruing sufficient

sleep to function well during the day. As treatment proceeds through adjustments in time in bed, a balance is sought whereby better daytime functioning is restored while sufficient sleep quality is maintained.

According to the 3P model of insomnia [4,5], behavioral practices and cognitive tendencies that perpetuate sleep disturbance are often the most promising targets for intervention. Many of these perpetuating factors, such as spending too much time in bed, anticipatory anxiety about the prospects for sleep, and inordinate concern about daytime performance deficits, are addressed by SRT. As noted above, SRT quickly changes the experience of insomnia, replacing sleep that has been haphazard and light with deeper, more consolidated sleep. There is little doubt that it is an “active treatment,” even if it does have significant side effects. Patients can rest assured that their sleep problem is being addressed, and this translates into less worry about what the night will bring. While they can no longer expect a luxuriously long night of sleep, there is also less likelihood of virtually sleepless nights. Patients learn that they can at least muddle through the day on what sleep they reliably accumulate, lowering the stakes regarding sleep loss. Finally, hyperarousal (whether a trait-like predisposing factor or reactive to events) is directly dampened by sleep loss.

Another reason SRT is effective is that it tightens regulatory control of sleep by the endogenous circadian pacemaker. Patients with chronic insomnia often display widely varying times of retiring to and rising from bed, with consequent variability in the timing of light exposure, social interaction, physical activity, and other stimuli that entrain the circadian system. The output of the endogenous oscillator is weakened as it continually responds to these shifting patterns, leading in turn to increased variability in the propensity to fall asleep and wake up. By closely regulating the time of “lights out” and “lights on” SRT gradually returns sleep regulation to effective circadian control, resulting in more reliably timed phases of sleep and wakefulness.

STEP BY STEP DESCRIPTION OF PROCEDURES

In the original SRT study we followed a rigid protocol with few procedures [1]. After a 2-week sleep log we set the initial TIB equal to the average estimated sleep time. Regardless of reported sleep time, no individuals were assigned less than 4 hours and 30 minutes of TIB. Time to get up in the morning was set to the time subjects needed to be up on work days. Based on information provided to a telephone answering machine for 8 weeks, a 5-day window was analyzed for sleep efficiency and changes were made to TIB according to the following rules:

- If SE was ≥ 90 percent (85 percent in seniors), TIB was increased by 15 minutes
- If SE was < 85 percent (80 percent in seniors), TIB was decreased to the average estimated sleep time
- If TIB was between 85 and < 90 percent, no changes were made

- In addition, subjects were not permitted to lie down or nap at times other than the assigned TIB.

We have modified our approach to SRT, as will be detailed below, for the following reasons. In order to limit the sleep deprivation at the start of treatment, we set the lower limit of TIB at 5 hours and allow for a 30-minute increase in TIB to quickly forestall severe sleepiness. To promote the most and best quality sleep at the start of treatment, we no longer set the wake-up time no later than the earliest time subjects need to be up (usually for work) on any day of the week. As will be seen in the examples below (see I, Example 3), the timing of the sleep period now takes into account the time of the night when sleep is the most likely to be experienced as deep and refreshing. In later clinical applications we did not end treatment after 8 weeks but when TIB is sufficient to sustain daytime functional capacity without leaving the individual too vulnerable to a recurrence of insomnia (see III below).

Initiation of SRT

SRT begins by estimating three key features of sleep: (1) typical sleep duration; (2) workday wake-up time; and (3) the portion of the night likely to contain the best sleep. These features are best assessed via a representative 1- to 2-week graphic sleep diary (by averaging estimated total sleep times and logged workday wake-up times, and perusal of the patterning of sleep segments within a night) along with a clinical interview. TIB at the start of treatment is set equal to the average sleep duration. (The minimum amount of TIB should not be less than 5 hours.) Wake-up time on SRT should be no later than the average logged workday wake-up time. However, the specific bedtime period assigned will depend on the individual's sleep pattern. If, for example, a patient's verbal report and sleep log show that the best sleep is obtained in the first two-thirds of the night, with erratic sleep thereafter, the assigned wake-up time should be earlier than the average logged workday wake-up time.

Example 1

For example, the 1-week sleep diary reveals the following:

Average bedtime to rising time	11 pm to 6:30 am
Average TIB	7 hours and 30 minutes
Average sleep latency	15 minutes
Average nightly sleep time	5 hours and 45 minutes
Average workday wake-up time	6:15 am

The latter 2 hours of sleep are described as “light” and interrupted frequently on the log.

The SRT prescription for the initial sleep schedule is as follows:

Allowed TIB	5 hours and 45 minutes
Bedtime	11 pm
Wake-up time	4:45 am, 7 days per week

The patient is asked to continue logging sleep on the new schedule and follow-up in 1 week. The prescribed TIB, 5 hours and 45 minutes, is determined from the calculated average nightly sleep time. The decision to have the patient wake-up at 4:45 am is based on the report of poor sleep in the latter 2 hours of the night.

Example 2

Suppose the sleep latency in the above example had been 1 hour and 15 minutes, with 30 minutes of wakefulness after sleep onset typically distributed in two awakenings. The initial SRT sleep schedule would then be:

Allowed TIB	5 hours and 45 minutes
Bedtime	12:30 am
Wake-up time	6:15am, 7 days per week

In this case, TIB is assigned later in the night to reduce sleep onset latency. Wake-up time on SRT is set equal to the average workday wake-up time; working back 5 hours and 45 minutes yielded the assigned bedtime.

Example 3

Suppose the sleep log in the above examples revealed wakefulness interspersed fairly evenly throughout the night, with a moderately long sleep latency of 35 minutes, a couple of awakenings of 15–20 minutes' duration, and a terminal awakening of about 30 minutes. The initial SRT sleep schedule would then be:

Allowed TIB	5 hours and 45 minutes
Bedtime	12:00 am
Wake-up time	5:45 am

Now, the nightly average of 1 hour and 45 minutes of wakefulness in bed (calculated from the sleep log) is addressed by assigning both a later bedtime and an earlier wake-up time, with the aim of consolidating sleep in between.

SRT Procedures During the “Middle Phase” of Treatment

According to the standard SRT procedure, sleep efficiency (SE) is calculated each week from a sleep diary:

- If SE is ≥ 90 percent, then TIB is increased by 15 or 30 minutes. The clinician/researcher uses judgment (or a rule) to decide on whether TIB is increased by 15 or 30 minutes. In older individuals, the SE cut point for increasing TIB is ≥ 85 percent.
- If SE is between 85 percent and < 90 percent, then TIB is not changed. (In older individuals, the range is between 80 percent and < 85 percent.)
- If SE is < 85 percent, then TIB is reduced by 15 or 30 minutes. (In older individuals, the cut point is < 80 percent.)

Example 4

A 74-year-old woman presents with sleep maintenance insomnia. Her 2-week sleep log shows accumulation of an average of 6 hours of sleep within an average TIB of 8 hours and 30 minutes. She is started on SRT, with bedtime set between midnight and 6 am. She returns with a 1-week sleep log showing an average subjective sleep time of 5 hours and 15 minutes, with relatively short sleep latencies but a persistent tendency to awaken too early in the morning. Her new assigned SRT sleep schedule would be:

Allowed TIB	6 hours and 15 minutes
Bedtime	11:45 pm
Wake-up time	6:00 am

This elderly patient’s sleep efficiency of 87.5 percent ($5.25/6.00 \times 100\%$) qualifies her for an extra 15 minutes of sleep, which were added to the beginning of the night given that she has more difficulty maintaining sleep toward morning.

In cases of paradoxical insomnia, patients may report very limited sleep time – for example, 3 hours per night on average. As SRT does not permit reduction of TIB below 5 hours, these patients may not progress in SRT. A decision must be made in such cases to persist with TIB set at 5 hours or to discontinue SRT.

Completing SRT

We have previously shown that SRT is amenable to analysis within a cost/benefits model [6]. This model is useful in determining when to end treatment. Maximizing sleep efficiency cannot be the sole endpoint, since sleep efficiency will tend to be highest when time in bed is cut to the prescribed

minimum of 5 hours, yielding very sleepy and likely noncompliant patients. In our model, satisfying nocturnal sleep and good daytime functioning are seen as primary “benefits” whereas the time spent in bed in order to accumulate sleep and vulnerability to insomnia are “costs”.

Prior to treatment, costs are high and benefits low, in that patients are spending a lot of time in bed, only to garner broken, unreliable and non-refreshing sleep. Daytime functioning is poor, marred not only by the effects of sleep loss but also by anticipatory anxiety over what the next night will bring. At the start of SRT there is a dramatic lowering of costs, as much less time is spent in bed, and susceptibility to very poor nights of sleep is reduced due to an increased homeostatic sleep drive. There is often a concurrent reduction in benefits, however, in that typically less sleep is accumulated, leading to increased sleepiness and deficits in mood, attention, and other aspects of daytime functioning.

As treatment progresses, there is generally an increase in both costs and benefits (e.g., less time is spent in bed, but more sleep is obtained, with only slightly increased susceptibility to insomnia). The increase in benefits rises at a greater rate than the increase in costs. As the titration proceeds, a point of maximal net benefit is reached. Time in bed is restricted enough to maintain a reliable pattern of well-consolidated sleep, but not so much as to yield significant daytime deficits.

This is the desired endpoint of treatment, and the schedule the patient should strive to maintain on his or her own. If time in bed were to be further increased – perhaps approaching its baseline value – there would likely not be much additional benefit in terms of extra sleep, since the bulk of the patient’s homeostatic sleep need has now been addressed, whereas the risk of reintroducing variable, broken sleep would be elevated.

Example 5

The 74-year-old woman introduced in Example 4 qualified for three additional 15-minute increments to TIB, which were added to the beginning of the night to yield the following SRT schedule:

Allowed TIB	7 hours
Bedtime	11:00 pm
Wake-up time	6:00 am

Subsequent logs showed estimated sleep time hovering near 5 hours and 45 minutes, yielding sleep efficiencies between 80 percent and 85 percent and therefore not qualifying for either an increase or a decrease in time in bed. The patient reported good daytime functioning. She was getting nearly as much sleep as prior to treatment, but in a much more efficient and predictable

manner, with less worry about what would happen each night. Therefore, SRT was ended, and she was advised to maintain an 11 pm to 6 am bedtime schedule going forward.

POSSIBLE MODIFICATIONS/VARIANTS

Investigators and clinicians have modified the original SRT procedure or created treatments that share essential features with SRT. The initial prescribed TIB in the SRT approach may be experienced by the patient as a severe deprivation and result in significant daytime sleepiness. In addition, given that sleep efficiency is rarely 100 percent, the initial TIB prescribed within SRT introduces some degree of sleep deprivation. One modification of the SRT procedure that takes this into account sets the initial TIB equal to the average amount of sleep reported plus 30 minutes [7]. This is a sensible and modest change to SRT; it eases the patient into a restricted bedtime schedule. Similarly, allowing or prescribing a daytime nap has been used to limit daytime sleepiness at the start of treatment [8]. Another change from the original SRT procedure that we wholeheartedly endorse is basing all changes in TIB on 7 days of data rather than the 5 days used in the original study.

Another approach that avoids the shock of a radically reduced TIB is called sleep compression [9–11]. While sharing the assumption that individuals with insomnia benefit from a reduction of TIB, this intervention starts with a modest reduction in TIB compared to SRT. During the first session, patients were advised to reduce TIB by half of the difference between baseline TIB and baseline TST. During the second and third sessions, TIB was further reduced by one-quarter of the difference between baseline TIB and baseline TST. For a complete description of sleep compression, please see Chapter 5. This approach is consistent with the clinical wisdom to “start where the patient is”. We have frequently suggested that when SRT is used clinically it is important to negotiate initial TIB with the patient so that resistance is minimized [12].

We have been impressed with how rarely we have had to reduce TIB after the initial restriction. Therefore, we have implemented a modification of SRT in which, following the initial restricted schedule (determined as usual from the average reported sleep time across a baseline week), we do not further reduce TIB. Instead, we increase TIB by either 15 or 30 minutes on a weekly basis regardless of reported sleep efficiency [12,13]. This approach is necessary in individuals with paradoxical insomnia who do not report sufficiently greater sleep efficiency on a significantly restricted schedule to trigger extra allotments of TIB.

PROOF OF CONCEPT

The treatment efficacy of SRT has been tested in a number of studies with different patient populations. There are only a few studies in which the

effectiveness of SRT is assessed as a stand-alone treatment. Spielman and colleagues [1] administered SRT to 35 adults with psychophysiological insomnia and insomnia co-morbid with psychiatric disorders in eight weekly individual sessions. Treatment outcomes, as measured by subjective sleep onset latency (SOL), total sleep time (TST), sleep efficiency (SE), and ratings on insomnia symptoms were all improved after treatment as well as at a 36-week follow-up. Although initially restricted, TST eventually increased from 320 minutes to 343 minutes and SE improved from 67 percent to 87 percent. This study did not employ a control group; therefore, its findings cannot be attributed directly to the treatment.

Subsequently, several studies modified the SRT procedures for elderly patients with insomnia, with more flexibility in the prescription of TIB [14–16], gradual sleep compression instead of abrupt cut-down of TST [8,10,11], and/or installing a mandatory or optional daytime nap [8,15]. These procedures were intended primarily to enhance the patients' tolerance of sleep restriction and/or to reduce daytime sleepiness due to partial sleep deprivation. In comparison to various control treatments (waiting-list control, relaxation training, sleep hygiene education, and a placebo desensitization procedure), SRT in these studies was found to increase SE and TST, and decrease wake after sleep onset (WASO). The outcome on SOL was not as consistent as the other measures. Treatment effects were at least partially maintained at short-term (2-month) and long-term (up to 2 years) follow-up.

For example, Friedman and colleagues [14] compared modified SRT with progressive muscle relaxation training. Subjects' tolerance was taken into consideration in the assignment of the prescribed TIB at the start of treatment to increase compliance. Also, TIB was not reduced for failure to reach criterion to avoid drop-out. The results showed that both treatments were effective in increasing TST, and reducing SOL and WASO. However, SRT was more effective in increasing SE and TST than relaxation training. Lichstein and colleagues [11] compared sleep compression (see above for the shared assumptions with SRT) that reduced TIB gradually in 5 weeks with relaxation training and placebo desensitization procedures; treatment effects were significant for sleep-log derived variables but not for those derived from a polysomnogram. It was found that both treatments produced significant improvement in SOL and WASO at post-treatment, but WASO benefits were maintained only in the sleep compression group at 1-year follow-up. Relaxation did better than sleep compression in enhancing TST.

The effective deployment of SRT in an inpatient setting was described in a case report. A 49-year-old woman with insomnia co-morbid with major depression and chronic pain was treated with SRT with the assistance of nursing staff. The SRT was administered in a modified form by starting with 4 hours in bed for 3 nights, increasing to 5 hours for the subsequent 3 nights. TIB was further increased to 6 hours per night after SE achieved 85 percent, and increased to 7 hours for 2 weeks prior to discharge. TST was improved

from 2 hours 30 minutes to over 6 hours after about 2 weeks of treatment, and the improvements were maintained at 4-month follow-up [17]. Sleep compression has also been found effective when delivered by self-help video, although additional meetings with a therapist did enhance the treatment outcomes [10].

Based on a review of the research evidence, the Practice Parameters for the Psychological and Behavioral Treatment of Insomnia [18] and the Clinical Guideline for the Evaluation and Management of Chronic Insomnia in Adults [19] published by the American Academy of Sleep Medicine rated SRT as a Guideline treatment, meaning that SRT is a patient-care strategy with a moderate degree of clinical certainty [20]. Although SRT is effective as a stand-alone treatment, clinically it is typically incorporated into the multi-component treatment known as cognitive behavior therapy for insomnia (CBT-I). The most common combination involves an educational component (sleep hygiene), a behavioral component (stimulus control, SRT, relaxation), and a cognitive component (cognitive restructuring). One study comparing different components of CBT-I reported that while relaxation was more effective for sleep onset problems, a combination of stimulus control and SRT had more benefits for sleep maintenance variables [21]. Both the 2006 Practice Parameter [18] and the 2008 Clinical Guideline [19] papers that rated CBT as a Standard explicitly include SRT as one of the three behavioral components of this approach. The review paper accompanying the Practice Parameters paper [20] showed that SRT was more commonly a part of CBT than relaxation therapy. In all twelve of the studies cited, SRT was a component of CBT.

REFERENCES

- [1] A.J. Spielman, P. Saskin, M.J. Thorpy, Treatment of chronic insomnia by restriction of time in bed, *Sleep* 10 (1) (1987) 45–56.
- [2] W. Webb, H. Agnew, Sleep: Effects of a restricted regime, *Science* 150 (1965) 1745–1747.
- [3] W. Webb, H. Agnew, The effects of a chronic limitation of sleep length, *Psychophysiology* 11 (1974) 265–274.
- [4] A.J. Spielman, Assessment of insomnia, *Clin. Psychol. Rev.* 6 (1986) 11–25.
- [5] A.J. Spielman, L. Caruso, P. Glovinsky, A behavioral perspective on insomnia treatment, *Psychiatr. Clin. N. Am.* 10 (4) (1987) 541–553.
- [6] A.J. Spielman, C.M. Yang, P.B. Glovinsky, in: Buysse, Sateia, (Eds.), *Sleep Restriction Therapy*, Informa, 2010 (in press).
- [7] J.D. Edinger, C.E. Carney, *Overcoming Insomnia: A Cognitive-Behavioral Therapy Approach – Therapist Guide*, Oxford University Press, Inc, New York, NY, 2008.
- [8] J.O. Brooks, III, L. Friedman, D.L. Bliwise, J.A. Yesavage, Use of the wrist actigraph to study insomnia in older adults, *Sleep* 16 (2) (1993) 51–55.
- [9] B.W. Riedel, K.L. Lichstein, W.O. Dwyer, Sleep compression and sleep education for older insomniacs: self-help versus therapist guidance, *Psychol. Aging* 10 (1995) 54–63.
- [10] B.W. Riedel, K.L. Lichstein, Strategies for evaluating adherence to sleep restriction treatment for insomnia, *Behav. Res. Ther.* 39 (2001) 201–212.
- [11] K.L. Lichstein, B.W. Riedel, N.M. Wilson, et al., Relaxation and sleep compression for late-life insomnia: a placebo-controlled trial, *J. Consult. Clin. Psychol.* 69 (2) (2001) 227–239.

- [12] P.B. Glovinsky, A.J. Spielman, Sleep restriction therapy, in: P. Hauri, (Ed.), *Case Studies in Insomnia*, Plenum, New York, NY, 1991, pp. 49–63.
- [13] M.L. Rubinstein, S.A. Rothenberg, S. Maheswaran, Modified sleep restriction therapy in middle-aged and elderly chronic insomniacs, *Sleep Res.* 19 (1990) 276.
- [14] L. Friedman, D.L. Bliwise, J.A. Yesavage, S.A. Salom, A preliminary study comparing sleep restriction and relaxation treatment for insomnia in older adults, *J. Gerontol.* 46 (1991) 1–8.
- [15] L. Friedman, K. Benson, A. Noda, et al., An actigraphic comparison of sleep restriction and sleep hygiene treatments for insomnia in older adults, *J. Geriatr. Psych. Neurol.* 13 (1) (2000) 17–27.
- [16] T.J. Hoelscher, J.D. Edinger, Treatment of sleep-maintenance insomnia in older adults: sleep period reduction, sleep education, and modified stimulus control, *Psychol. Aging* 3 (3) (1988) 258–263.
- [17] C.M. Morin, R.A. Kowatch, G. O’Shanick, Sleep restriction for the inpatient treatment of insomnia, *Sleep* 13 (2) (1990) 183–186.
- [18] T. Morgenthaler, M. Kramer, C. Alessi, et al., Practice parameters for the psychological and behavioral treatment of insomnia: an update. An American Academy of Sleep Medicine report standards of practice, *Sleep* 29 (11) (2006) 1415–1419.
- [19] S. Schutte-Rodin, L. Broch, D. Buysse, et al., Clinical guideline for the evaluation and management of chronic insomnia in adults, *J. Clin. Sleep Med.* 4 (5) (2008).
- [20] C.M. Morin, R.R. Bootzin, D.J. Buysse, et al., Psychological and behavioral treatment of insomnia: update of the recent evidence (1998–2004), *Sleep* 29 (11) (2006) 1398–1414.
- [21] W.F. Waters, M.J. Hurry, P.G. Binks, et al., Behavioral and hypnotic treatments for insomnia subtypes, *Behav. Sleep Med.* 1 (2) (2003) 81–101.

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