FAST Behavioral Therapy May Defeat Insomnia

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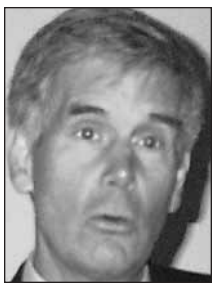
DENVER — A novel behavioral therapy conducted over just 26 hours in a single weekend shows promise for the treatment of chronic primary insomnia, Leon Lack, Ph.D., reported at the annual meeting of the Associated Professional Sleep Societies.

"Even though stimulus control therapy and other cognitive behavioral therapies have been shown [to be] very effective in clinical trials, we sometimes wonder whether they're as effective in real-world situations where we give instructions to patients and have them carry them out in the home environment. We don't know much about that. And there could be difficulties with people complying with the instructions. If they do, we think they're very effective, but some of these instructions are rather rigorous," noted Dr. Lack of Flinders University in Adelaide, Australia.



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DR. LACK

In contrast, Flinders Accelerated Sleep Therapy (FAST) is a modified sleep-deprivation regimen involving constant bed rest during the treatment weekend, the psychologist explained. Patients arrive at the sleep laboratory at about 8 p.m. Saturday and get hooked up to the EEG machine. From then until 11 p.m. Sunday they're encouraged to fall asleep—but every time they do, they are awakened after 4 minutes. This typically happens at least 50 times.

During this period patients get very sleepy. Their sleep latencies start dropping. Starting at 11 p.m. Sunday, patients are permitted to have uninterrupted recovery sleep until Monday morning. Then they go home.

Dr. Lack reported on 17 patients (mean age 39 years) with primary insomnia. Their chief sleep problem involved delayed onset, although some also had sleep maintenance difficulties. They were assessed using sleep diaries, actigraphy, and psychological questionnaires for 2 weeks at baseline, immediately after FAST therapy, and again 6 weeks after treatment.

Sleep-onset latency as reflected in diaries dropped from 70 minutes at baseline to 40 after treatment, rebounding slightly to 47 at follow-up. Sleep latency as measured by actigraphy also showed significant improvement: 45 minutes at baseline, 33 post-treatment, and 38 at follow-up, he said.

Mean total sleep time from patient diaries was 5.29 hours at baseline, 6.36 hours right after FAST, and 6.0 hours at 6 weeks. Sleep efficiency rose from 62% to 76% immediately after treatment and was 74% at 6 weeks. Actigraphy also showed significant improvements in total sleep time and sleep efficiency as a consequence of FAST. Results of the Profile of Mood States documented significant improvements in fatigue, vigor, and sleep anticipatory anxiety. There were trends toward reductions on measures of depression, anxiety, and stress, none of which reached significance.

Speculating on FAST's possible mechanism of action, Dr. Lack said that the therapy retrains patients to fall asleep quickly, presumably reducing the psychophysiologic conditioned response of insomnia. It could be viewed as a form of desensitization to sleep deprivation.

"Insomniacs typically are very frightened to put themselves into situations where they might lose some sleep. Here they lose sleep over a 25-hour period and find they don't die from it—they just get very, very sleepy. And sleepiness is actually an experience that many of them don't have much of the time. It may be therapeutic," he said.

It's also conceivable that the clinical benefit is attributable to a placebo effect, since there was no control group. To remedy this shortcoming, Dr. Lack and his associates are currently conducting a randomized controlled trial involving 100 patients with primary sleep-onset insomnia. The patients are being randomized to a wait-list control group, stimulus control therapy, FAST alone, or FAST followed by stimulus control therapy. Dr. Lacks hopes that it achieves a greater, more durable treatment effect than FAST alone. ■