

CONTROLLED SLEEP RESTRICTION FOR RHYTHMIC MOVEMENT DISORDER

TAMAR ETZIONI, BSC, NERI KATZ, MD, ELI HERING, MD, SARIT RAVID, MD, AND GIORA PILLAR, MD, PHD

Rhythmic movement disorder is a parasomnia that is difficult to treat. In our study, 3 weeks of controlled sleep restriction with hypnotic administration in the first week resulted in almost complete resolution of the movements in 6 children. This therapeutic success suggests that rhythmic movement disorder results from a voluntary self-soothing behavior. (*J Pediatr* 2005;147:393-5)

Rhythmic movement disorder (RMD) is defined as a group of stereotyped, repetitive movements (RM), involving large muscles, usually of the head and neck (head banging), that typically occur immediately before sleep onset and are sustained into light sleep. Occasionally it may be seen in deep or rapid eye movement sleep.^{1,2} Although several studies reported that RMD may spontaneously alleviate and even disappear with age, it may be important to treat it earlier because it may be harmful and may persist to adulthood.²⁻⁵

Treatment of RMD may be difficult. In some cases benzodiazepines were beneficial,^{4,6} but in other cases they failed.³ In several case reports, antidepressants such as imipramine produced good clinical outcome.³ Behavioral intervention⁷ or hypnosis⁸ has also been suggested, but they have not been sufficiently studied.⁹ Most of these studies reported the effects of short-term treatment, and there are only few data regarding long-term effects.

One potential explanation of the mechanism underlying RMD is that these patients voluntarily generate RM to help them fall asleep. Thus we planned a controlled sleep restriction program, with usage of hypnotics in the first part of the program, to rebuild faith in the ability to fall asleep. We hypothesized that once good sleep initiation is established, RM would reduce in frequency.

METHODS

Six children and adolescents (3 females) aged 7.3 ± 2.9 years (range 3.5 to 12 years), with a diagnosis of RMD (2 of them with a previous treatment failure) were studied. They (or the parents) completed a questionnaire, underwent a 1-week actigraphic study (primarily to objectively determine total sleep time), and commenced a treatment protocol (see below). Parents were asked to observe their children while falling asleep every night during the first month of treatment and quantitatively score the existence of RM (0 = no RM, 1 = rarely RM, 2 = occasional RM, 3 = frequent RM, 4 = continuous intense RM until asleep). Parents scored sleep latency and RM every night during the first month of treatment. Additional assessment took place at 1-year follow-up.

The Actigraph (Mini Monitor Actigraph, Ambulatory Monitoring, New York, NY) is a self-contained microcomputer housed in a $2.5 \times 3.5 \times 0.75$ -inch light-weight case. It is placed on the nondominant hand and translates movements into electrical signals that identify wake or sleep by use of a validated scoring program.¹⁰ The children wore it for 7 consecutive days and nights in their native environment. Actigraphy was obtained before any therapeutic intervention, and the important variables derived from it were sleep latency (the time from "lights out reported by the parents to falling asleep defined by the actigraph), total sleep time (TST, time of actual sleep excluding periods of wakefulness) and sleep efficiency (percentage of TST of total time in bed).

From the Sleep Lab, Meyer Children's Hospital, Rambam Medical Center and Technion-Israel Institute of Technology, and Pediatric Neurology Unit, Meyer Children's Hospital, Rambam Medical Center, Haifa, the Pediatric and Premature Departments, Wolfson Hospital, Holon, and the Pediatric Department, General Health Care, Child Care Center, Tirat Hacarmel, Israel.

Submitted for publication Feb 28, 2005; last revision received May 5, 2005; accepted Jun 22, 2005.

Reprint requests: Giora Pillar, MD, PhD, Sleep Lab, Rambam Medical Center, Haifa 31096, Israel. E-mail: gpillar@tx.technion.ac.il.

0022-3476/\$ - see front matter

Copyright © 2005 Elsevier Inc. All rights reserved.

10.1016/j.jpeds.2005.06.045

RM	Repetitive movement	TST	Total sleep time
RMD	Rhythmic movement disorder		

Table. Characteristics and findings of the children

Child	Age (years)	Sex	Previous treatment	Reported HSD (h)	Actigraph SL (min)	Actigraph TST (h)	Actigraph SE (%)
1	5	m	None	11	32	10.2	84
2	12	m	Clonazepam	9	47	7.8	81
3	7	f	None	10.5	11	10	93
4	8	f	Psychological	8.5	36	8.1	86
5	8	m	None	9.5	14	9.8	94
6	3.5	f	None	10	15	9.5	93
Mean ± SD	7.3 ± 2.9			9.8 ± 0.9	25.8 ± 14.6	9.2 ± 1.0	88.5 ± 5.5

Reported HSD, Reported habitual sleep duration (hours); SL, sleep latency (min); SE, sleep efficiency (%).

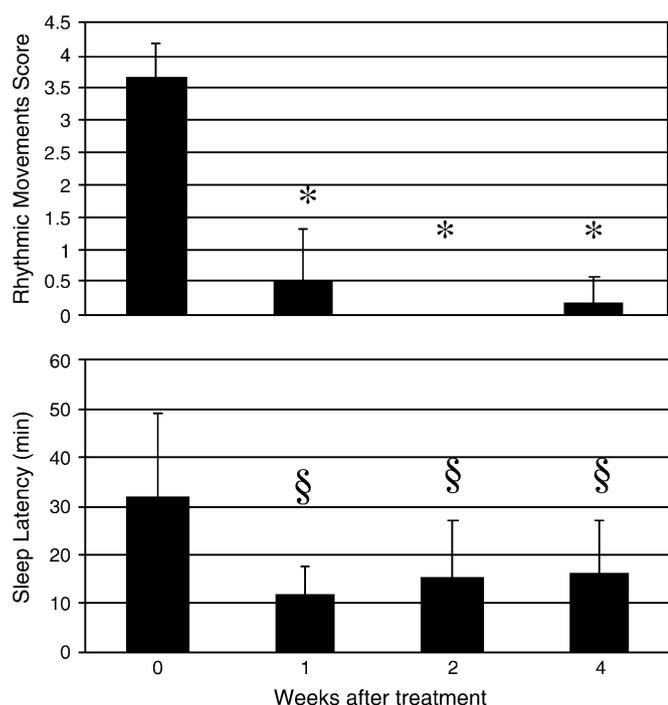


Figure. Rhythmic movement score (upper panel) and sleep latency (lower panel) before treatment (= 0), and at 1, 2, and 4 weeks after treatment protocol. All data are mean ± SD. All changes were significant compared to baseline values (paired *t* test), for RMD score in *P* < .005 level (*), and for sleep latency in *P* < .01 level (§).

Habitual total nocturnal sleep time was determined as the average between the reported and the actigraphic measures. A 3-week sleep restriction regimen was determined for each individual: first week a reduction of 1 hour from nocturnal TST, and administration of chloral-hydrate 0.5 mL/kg before bedtime (no change in nap duration), second week hypnotic was ceased but total nocturnal sleep time was still restricted for 1 hour less than baseline, and third week a gradual return (increment of 10 minutes every night) to the baseline schedule.

RESULTS

Characteristics of the 6 children are presented in the Table. Body weight, number of children in the family or their

place within the family did not have an effect on the existence of RMD or their response to treatment. Two of the children had a previous failure of a therapeutic trial before the current regimen was imposed. Two children had no sleep complaints, whereas 4 reported difficulties in falling asleep, and one of them also reported about frequent awakenings from sleep. Families and children reported a very good compliance with the treatment, with almost no side effects (parents reported bearable nervousness and mild sleepiness or agitation). Both sleep latency and rhythmic movements dramatically improved in all children (Figure), although there was no correlation between them (*r* = 0.1). Although rhythmic movements were completely abolished at 2-week follow-up, at 4 weeks one of the children re-experienced rare RM, which persisted at 1-year follow-up.

DISCUSSION

Our study suggests that the combination of controlled mild sleep deprivation, with usage of hypnotics at treatment initiation, may abolish RM and cure RMD. In addition, the resolution of RMD with primarily sleep deprivation supports the hypothesis that it can be classified as a type of voluntary movement that serves as a self-soothing behavior in the process of falling asleep, rather than as an involuntary movement disorder. Most of these latter movements such as those from epilepsy usually aggravate with sleep deprivation.¹¹

Although usually a self-limiting disorder, we believe RMD should be treated on diagnosis because treatment can prevent secondary social/psychological consequences, physical damage,^{5,8} and persistent into adulthood.^{2-4,12} Hypnotics alone may fail,³ and movements can re-occur after treatment cessation. We do not know whether the treatment should be longer or shorter than our 3-week regimen, which was arbitrarily chosen. Our rationale was to build a mild sleep deprivation protocol for 3 weeks (and not dramatic sleep restriction for shorter period) to allow the child to build confidence in his or her ability to fall asleep without the assistance of the RM. We speculate that this allowed the success to continue long term. The optimal timing and usage of hypnotic treatment will have to be determined on a larger group of children. However, we believe our findings of long-term success in 5 of 6 children,

long after cessation of medication and therapeutic intervention, is an important observation.

REFERENCES

1. Hoban TF. Rhythmic movement disorder in children. *CNS Spectr* 2003;8:135-8.
2. Thorpy MJ, Glovinsky PB. Parasomnias. *Psychiatr Clin North Am* 1987;10:623-39.
3. Alves RS, Aloe F, Silva AB, Tavares SM. Jactatio capitis nocturna with persistence in adulthood. Case report. *Arq Neuropsiquiatr* 1998;56:655-7.
4. Chisholm T, Morehouse RL. Adult headbanging: sleep studies and treatment. *Sleep* 1996;19:343-6.
5. Mackenzie JM. "Headbanging" and fatal subdural haemorrhage. *Lancet* 1991;338:1457-8.
6. Reimao R, Lefevre AB. Evaluation of flurazepam and placebo on sleep disorders in childhood. *Arq Neuropsiquiatr* 1982;40:1-13.
7. Ross RR. Treatment of nocturnal headbanging by behavior modification techniques: a case report. *Behav Res Ther* 1971;9:151-4.
8. Rosenberg C. Elimination of a rhythmic movement disorder with hypnosis—a case report. *Sleep* 1995;18:608-9.
9. Kuhn BR, Elliott AJ. Treatment efficacy in behavioral pediatric sleep medicine. *J Psychosom Res* 2003;54:587-97.
10. Sadeh A, Hauri J, Kripke DF, Lavie P. The role of actigraphy in the evaluation of sleep disorders. *Sleep* 1995;18:288-302.
11. Wills L, Garcia J. Parasomnias: epidemiology and management. *CNS Drugs* 2002;16:803-10.
12. Kohyama J, Matsukura F, Kimura K, Tachibana N. Rhythmic movement disorder: polysomnographic study and summary of reported cases. *Brain Dev* 2002;24:33-8.