Sleep Medicine 16 (2015) 908–909

Contents lists available at ScienceDirect

Sleep Medicine

journal homepage: www.elsevier.com/locate/sleep

Editorial

Sleep and fibromyalgia: it is time to think big

There is no doubt that sleep disruption is a distinct clinical feature of fibromyalgia (FM). As noted by Diaz-Piedra and colleagues [1], there is a robust literature documenting the presence of sleep problems among patients with FM. Almost all women with FM self-report poor sleep quality and objective measures of sleep have frequently documented evidence of disordered sleep. This line of research is consistent with the Sleep and Pain Diathesis (SAPD) [2] model which posits that a syndrome of generalized widespread pain (the cardinal symptom of FM) can develop with onset of any form of sleep disruption.

The purpose of Diaz-Piedra and colleagues' study was to demonstrate that FM patients have objective sleep disturbances that correspond to their subjective complaints. To this end they were successful. Women with FM reported poor subjective sleep quality and poor sleep quality was related to percent wake time. Moreover, individuals with FM differed from healthy controls in terms of lower sleep efficiency (primarily due to excessive time in bed), greater time spent in N1 sleep, and a greater number of arousals. These data are consistent with a substantial body of literature documenting a heterogeneous set of sleep abnormalities in patients with FM.

Diaz-Piedra's study contributes to a growing body of literature comparing individuals with FM to healthy controls or other pain populations. This literature is composed of small studies that have used very different selection criteria. As noted by Diaz-Piedra et al. in a comprehensive review, there are substantial differences among studies for a number of important parameters [3]. The studies reported in Table 1 of their article in this issue of the journal reflect this heterogeneity [1]. For instance, several studies excluded patients based on bed-time and/or time in bed [4–6]. Studies differed on wash-out duration for sleep related medications, ranging from two days [7] to two weeks eg [4,5]. Perhaps the biggest difference was in age. At the low end, the mean age was 42.5 [4,5] and at the high end, the average age was 52 [7]. Moreover studies differed in how participants were matched, with all studies matching for age, while several matched on age, menopause status and body mass index (BMI) [4,5], and one matched for age, BMI, sex, and respiratory function [8]. Still another study reported a high degree of discordance in menopause status between individuals with FM and controls [9]. Many of these limitations were also noted by Diaz-Piedra et al. in their review. Although as a whole these data suggest the presence of disordered sleep, it is difficult to determine the nature of FM-related sleep disruption given the inclusion of many variables in the data.

Another layer of heterogeneity relates to referral source. The patients studied by Diaz-Piedra et al. and those in the studies reviewed by Diaz-Piedra et al. were selected primarily for the presence of FM, and excluded if there were indications of a primary sleep disorder [1,3]. However, there exists another body of small studies that have assessed sleep in FM patients referred to sleep clinics [10,11]. Patients in these studies have a high likelihood of having a primary sleep disorder such as obstructive sleep apnea or periodic limb movement disorder and differ from patients in the studies reviewed by Diaz-Piedra et al. on other patient characteristics such as BMI. It is difficult to gain a complete picture of sleep in FM patients because of the disconnection of these two bodies of research.

The small study approach is limiting in yet another regard: it reduces our ability to determine whether patients with similar sleep profiles are similar in other regards. Although the SAPD model posits that sleep is a causal factor, it does not specify that all sleep disorders have the same etiology or would have a similar response to treatment. For instance, it may be the case that FM patients with primary sleep disorders have different characteristics than those with more diffuse sleep complaints. Or, it may be that there is a unifying sleep-related pathology that cuts across all groups, as is suggested by Rosenfeld and Stern [11]. Regardless of which of these suppositions is true, identifying subgroups of patients with similar sleep complaints could be a useful first step in understanding the range of etiologies that result in an FM phenotype, which would aid in the development of more effective treatments and perhaps prevention strategies. It is time to move beyond small studies. It is time for a large epidemiological study of sleep in patients with FM that purposefully includes patients with a wide range of BMI; a study that documents rather than excludes comorbid medical and psychological comorbidities, and carefully documents menopause status. It is time for a clear picture, rather than a series of snapshots of very different patient populations.

Conflict of interest

The ICMJE Uniform Disclosure Form for Potential Conflicts of Interest associated with this article can be viewed by clicking on the following link: http://dx.doi.org/10.1016/j.sleep.2015.05.001.

References

[1] Diaz-Piedra C, Catena A, Sánchez AI, et al. Sleep disturbances in fibromyalgia syndrome: the role of clinical and polysomnographic variables explaiing poor sleep quality in patients. Sleep Med 2015 (in press).
[2] Hamilton NA, Atchley RA, Karlson CW, et al. The role of sleep and attention in the etiology and maintenence of fibromyalgia. Cognit Ther Res 2012;36(1):81–93.
[3] Diaz-Piedra C, Di Stasi LL, Baldwin CM, et al. Sleep disturbances of adult women suffering from fibromyalgia: a systematic review of observational studies. Sleep Med Rev 2015;21:86–99.
[4] Burns JW, Crofford L, Chervin RD. Sleep stage dynamics in fibromyalgia patients and controls. Sleep Med 2008;9:689–96.

http://dx.doi.org/10.1016/j.sleep.2015.05.001
1389-9457/

[5] Chervin RD, Teodorescu M, Kushwaha R, et al. Objective measures of disordered sleep in fibromyalgia. J Rheumatol 2009;36:2009–16.
[6] Roehrs T, Diederichs C, Gillis M et al. Nocturnal sleep, daytime sleepiness and fatigue in fibromyalgia patients compared to rheumatoid arthritis patients and healthy controls: a preliminary study. Sleep Med 2013;14:109–15.
[7] Riva R, Mork PJ, Westgaard RH, et al. Fibromyalgia syndrome is associated with hypocortisolism. Int J Behav Med 2010;17:223–33.
[8] Sergi M, Rizzi M, Braghiroli A, et al. Periodic breathing during sleep in patients affected by fibromyalgia syndrome. Eur Respir J 1999;14:203–8.
[9] Landis CA, Lentz MJ, Rothermel J et al. Decreased nocturnal levels of prolactin and growth hormone in women with fibromyalgia. J Clin Endocrinol Metab 2001;86:1672–8.
[10] Gold AR, Dipalo F, Gold MS, et al. Inspiratory airflow dynamics during sleep in women with fibromyalgia. Sleep 2004;27:459–66.
[11] Rosenfeld VW, Stern RDN. Polysomnography with quantitative EEG in patients with and without fibromyalgia. J Clin Neurophysiol 2015;32:164–70.

Nancy A. Hamilton
Department of Psychology, University of Kansas, 1415 Jayhawk Blvd, Lawrence, KS 66045, USA.
E-mail address: nancyh@ku.edu

Available online 14 May 2015