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RESEARCH ARTICLE

A comparative study of sleep architecture in non-institutionalized senior citizens of Bengaluru city and normative data of sleep

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ABSTRACT

Background: Sleep disturbances are particularly common among even the apparently healthy aging population, and its role as a cause or consequence of chronic illness makes it an issue worth addressing. **Aims and Objectives:** (i) To record and score the sleep architecture in familial senior citizens using polysomnography, (ii) to compare the score with the normative data provided by American Academy of Sleep Medicine for the same age group, and (iii) to compare the polysomnographic recordings of male and female subjects. **Materials and Methods:** The study was done on apparently healthy 30 non-institutionalized senior citizens (15 males and 15 females) residing with their families in residential areas of Bengaluru city. Eligible subjects underwent overnight polysomnography. The recorded sleep parameters included total sleep time (TST), sleep latency (SL), rapid eye movement (REM) latency (RL), wake after sleep onset, sleep efficiency (SE), and stages N1%, N2%, N3%, and REM%. The data were manually scored, tabulated, and compared with normative data using the appropriate statistical tools. **Results:** On statistical analysis, TST was not significantly altered. The SL, RL, wake time after sleep onset, N1%, and N2% were significantly ($P < 0.05$) increased in the study group compared to normative data. SE, N1%, and N2% are significantly decreased compared to the normative data. Furthermore, males had longer RL and females had greater percentage of N1% compared to each other. **Conclusion:** The sleep architecture of the senior citizens under study is significantly deviate from the normative data and the causes for this may be altered circadian rhythm, nutritional, hormonal, or psychosocial factors.

KEY WORDS: American Academy of Sleep Medicine; Non-institutionalized; Polysomnography; Senior Citizens

INTRODUCTION

India has around 100 million elderly and the number is expected to increase to 323 million, constituting 20% of the total population by 2050.[1] The occurrence of chronic diseases increases with ageing. In 2005, chronic diseases accounted for 20 million deaths worldwide among >70 years old.[2] Hence, geriatric medicine is a health sector priority. Sleep disturbance is a common complaint among the senior citizens. The deteriorating quality of sleep and chronic illness, in fact, forms a vicious cycle. Keeping in view the rejuvenating and restorative effects of good quality sleep, the objective of this study is to find out the extent of derangement of sleep architecture in familial senior citizens of Bengaluru. The data will be recorded using polysomnography and compared with the normative data provided by American Academy of Sleep Medicine (AASM)[3] for that age group.

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Objectives

- To record and score the sleep architecture in non-institutionalized familial senior citizens using polysomnography.

- To compare the score with the normative data provided by AASM for the same age group.
- To compare the polysomnographic parameters of male and female subjects.

MATERIALS AND METHODS

This was an observational study done on 30 senior citizens of residential areas of Bengaluru city selected based on inclusion and exclusion criteria from the general population. The study period was from February 2014 to September 2016, and it was done at the Lifestyle lab, Department of Physiology, Victoria Hospital, Bangalore Medical College and Research Institute (BMCRI), Bengaluru.

The study included apparently healthy 15 males and 15 females who were 60 years or older and was residing with their families, i.e., not under the care of any institution like old age homes. We excluded cases of any cardiopulmonary, renal, gastroenterological, or neurological disorders. Individuals on antidepressants, antipsychotics, beta-blockers, theophylline derivatives, or glucocorticoids were excluded. All the subjects were non-smokers, non-alcoholics, and not addicted to any form of psychoactive substances.

The study was approved by the Ethical Committee of BMCRI. A written informed consent was taken followed by relevant history taking and general physical examination. General Health Questionnaire 12[4] was administered to those selected after screening to exclude individuals with psychological distress. The selected subjects were instructed to visit the Lifestyle lab for two consecutive nights. The 1st day was meant for habituation and the following day for actual sleep recording. Overnight polysomnography recording was done under all precautions, for at least 8 h during which the variables were monitored by Recorders and Medicare System Polysomnography Software (RMS PSG Version 1.0.0.0). The data were analyzed and scored manually according to Rechtschaffen and Kales criteria,[5] and it was compared to the normative data provided by AASM for that age group. The sleep parameters of male and female subjects were also compared.

IBM SPSS Statistics (Version 20.0.0) software was used for the analysis of the data. Descriptive statistical analysis has been carried out in the present study. Results on continuous measurements are presented as mean ± standard deviation. Significance is assessed at 5% level of significance. Student's *t*-test (two-tailed, independent) has been used to find the significance of study parameters on continuous scale between the study group and the normative data.

RESULTS

In our study, we found that in comparison to the normative data there is significantly increased mean values of sleep latency (SL), rapid eye movement (REM) latency (RL), and wake time after sleep onset (WASO) in the subjects. This is reflected in the significant reduction in the sleep efficiency (SE) compared to the normative data [Table 1]. Furthermore, the stages N1% and N2% are significantly increased while the stages N3% and REM% are significantly reduced compared to normative data [Table 2]. We also found that male subjects had a significantly longer RL compared to female subjects while female subjects had significantly greater stage N1% compared to males [Table 3].

Table 1: Comparison of mean values of TST, SE, SL, and RL of all the 30 subjects with the normative data

Study variables	Values	Normative data	*P* value
TST (in min)	342.57±30.14	348.8	0.27
SL (in min)	13.22±4.12	8.2	<0.0001**
RL (in min)	126.8±22.13	90	<0.0001**
WASO (in min)	50.28±7.5	42.9	<0.0001**
SE (%)	83.41±3.8	85.8	0.001**

TST: Total sleep time, SE: Sleep efficiency, SL: Sleep latency, RL: REM latency. ** P <0.05

Table 2: Comparison of mean values of different stages of sleep (N1, N2, N3, R, and WASO) as a percentage of TRT of the study group with the normative data

Study variables	Values	Normative data	*P* value
N1 (% TRT)	4.44±0.7	4	0.001**
N2 (% TRT)	59.67±1.98	57.6	<0.0001**
N3 (% TRT)	5.6±2.39	7.7	<0.0001**
REM (% TRT)	14.76±2.2	16.4	0.0002**
WASO (% TRT)	12.27±1.76	12.3	0.93

TRT: Thoracic radiation therapy, REM: Rapid eye movement. ** $P < 0.05$

Table 3: Comparison between study variables of male and female subjects

Study variables	Males (15)	Females (15)	*P* value
Age (in years)	66.13±6.4	68.2±7.1	0.42
Total recording time (min)	406.13±35.2	415.2±25.2	0.44
TST (min)	341.65±37.33	343.49±20.5	0.87
SE (%)	84.0±3.9	82.8±3.6	0.41
SL (min)	12.87±2.86	13.6±5.1	0.66
RL (min)	140.27±20.8	113.3±13.6	0.0003**
Wake after sleep onset (min)	49.59±5.4	50.96±9.1	0.63
N1 (% TRT)	4.03±0.58	4.85±0.55	0.0006**
N2 (% TRT)	59.76±2.23	59.58±1.69	0.81
N3 (% TRT)	5.24±2.0	5.98±2.6	0.41
R (% TRT)	15.46±2.1	14.07±2.1	0.09
WASO (% TRT)	12.29±1.6	12.25±1.9	0.95

TST: Total sleep time, SE: Sleep efficiency, SL: Sleep latency, TRT: Thoracic radiation therapy, RL: REM latency. ** $P < 0.05$

DISCUSSION

In our study, we noted significant deviation of the sleep parameters from the normative data. SL, RL, and WASO were found to be increased and SE was found to be decreased. Percentage of N1 and N2 was increased while the percentage of N3 and REM sleep was decreased compared to normative data. Males had longer RL and females had increased percentage of N1 compared to each other.

The study group had significantly increased WASO and decreased SE [Table 1]. These findings are in agreement with the study conducted by Evans and Rogers,[6] who found that elderly people have decreased total sleep time (TST), decreased SE, and fragmented sleep. This can be explained based on alteration in circadian rhythm that occurs with aging. As people age, there is deterioration of the suprachiasmatic nucleus, resulting in less synchronized sleep-wake circadian rhythms due to decreased responsiveness to external cues which includes daylight processed through the retinohypothalamic pathway. Research suggests that the secretion of endogenous melatonin also decreases with age resulting in decreased SE and in increased incidence of circadian rhythm disturbance.[7] This results in less consistent periods of sleeping and waking across the 24 h day. In addition, the amplitude of the circadian rhythm may decrease with age. This can result in increased nighttime awakenings and subsequent excessive daytime sleepiness.[8] The inadequate exposure to daylight due modernization of lifestyle can be a synergistic factor in the disruption of circadian rhythm. Older adults also experience a shift, or advance, in circadian sleep rhythms. Circadian rhythm advancement may be a result of changes in core body temperature cycle, decreased light exposure, and may also be related to genetic factors.[8,9] Circadian rhythm disturbance is effectively treated with bright light therapy or adequate daylight exposure. This would help advance or delay sleep-wake rhythms and can also shift core body temperature and endogenous melatonin rhythms.[10]

These changes can also be attributed to one of the most common and preventable etiological factors, i.e., nutrition. Deficiency of Vitamin D, calcium, magnesium, and potassium can be responsible for the sleep disturbances in the elderly. Maintaining adequate serum levels of Vitamin D may be important for sleep duration and quality; however, these associations are not well understood. Vitamin D deficiency is common among the elderly because as skin ages it cannot synthesize Vitamin D from sunlight as efficiently as younger skin.[11] Further, lifestyle changes in the recent times have lead people to spend more time indoors; therefore, exposure to the sun is limited, leading to potentially even greater health consequences in an older population.[12-14] The mechanisms by which Vitamin D could affect sleep are not yet clear. In animal studies, nuclear concentrations of the Vitamin D hormone-target neurons have been found in specific areas of the brain and spinal cord, some of which are thought to play a role in sleep including anterior and posterior hypothalamus, substantia nigra, midbrain central gray, raphe nuclei, and the nucleus reticularis pontis oralis and caudalis.[15-18] Similar findings were reported in a study of immunohistochemical investigations with antibodies to Vitamin D receptor proteins, which found evidence for target neurons in the same regions of the brainstem and hypothalamus.[19] The presence of Vitamin D target neurons in these regions of the brainstem that affect sleep suggests Vitamin D may mediate an individual's sleep. Massa *et al*.[20] examined whether levels of serum Vitamin D are associated with objective measures of sleep in community-dwelling older men. Objective estimates of nightly TST, SE, and WASO were obtained using wrist actigraphy worn for an average of five consecutive 24-h periods. They concluded that among older men, low levels of total serum 25(OH)D are associated with poorer sleep including short sleep duration, lower SE, and increased WASO.

In magnesium deficiency, sleep is usually agitated with frequent nighttime awakenings. On the other hand, high magnesium, low aluminum diet has been found to be associated with deeper, less interrupted sleep. This was proven in a study conducted by James Penland at the Human Nutrition Research Centre in North Dakota.[21]

Although there is no definitive research to support the concept that low potassium causes sleep problems, the potential is there. Potassium channel dysfunctions, which can prevent potassium from getting into cells, and the muscle spasms, which are one of the symptoms of hypokalemia can result in sleep problems. Drennan *et al*.[22] in their study found that potassium supplementation in normal young males on low potassium diet showed increase in SE due to reduction in WASO.

Table 2 summarizes a reduction in stage REM%. This is in agreement with a study published in the European Neurology Journal in which researchers found that calcium levels in the body are related to REM phase. The study concluded that disturbances in sleep, especially the absence of REM sleep or disturbed REM sleep, are related to a calcium deficiency. Restoration to the normal course of sleep was achieved following the normalization of the blood calcium level. Calcium is directly related to our sleep cycles. Calcium helps the brain use the amino acid tryptophan to manufacture melatonin.[21]

The decline in stage N3 and REM sleep along with increased number of arousals or WASO [Tables 1 and 2] that we noted in our study is in agreement with the study conducted by Van Cauter *et al*.[23] who found that there is an age-related decline in slow-wave sleep (SWS) (N3%) and REM% and also there is increased sleep fragmentation which can be explained on the basis of dysregulation of hypothalamic-pituitary-adrenal (HPA) axis seen in the elderly. It is known that the HPA axis is acutely inhibited during early (SWS).[24-28] Furthermore, even

partial sleep deprivation results in an elevation of cortisol levels the following evening.[29] Thus, both decreased SWS and sleep loss resulting from increased sleep fragmentation could contribute to elevating cortisol levels in elderly. An elevation of evening cortisol levels is a hallmark of aging[30-32] that is thought to reflect an impairment of the negative feedback control of the HPA axis. Increased evening cortisol further promote sleep fragmentation and may promote awakenings forming a vicious cycle of events.[33,34]

Psychosocial factors also play a significant role in sleep derangements. Elderly people living with their families have multiple issues that worry them. Retirement, worry about family, loneliness, and grief bereavement can all be a cause of those sleepless nights. There is evidence that lonely individuals have lower SE and increased WASO.[35,36] Grief refers to feelings, thoughts, and behaviors following the loss of a loved one. Grief is closely connected to depression and sleep impairments.[37-41] In other studies, bereavement was found to be positively associated with the number of awakenings during the night.[42] According to Kales *et al*.,[43] the predisposition to internalize psychological conflicts leads to heightened levels of emotional arousal, which in turn provokes physiological hyperarousal and renders the individual unable to sleep. The majority of current etiological theories consider heightened levels of autonomic, cortical, cognitive, and emotional arousal to be a stable feature of people with sleep derangements.[44-46] Perlis *et al*.[47] postulated insomnia results from enhanced cortical hyperarousal, measured objectively, for example, as increased fast frequencies in the sleep electroencephalographic. This is experienced subjectively as cognitive hyperarousal (e.g., intrusive thoughts during the sleep-onset period, dysfunctional beliefs), which results in increased autonomic arousal. According to Espie,[46] affect dysregulation mediates the effect of cognitive and autonomic hyperarousal on sleep. From a neurobiological point of view, emotional stimuli interact with the basic homeostatic and circadian drives for sleep through the interaction between affect-related regions such as the infralimbic cortex or the central nucleus of the amygdala, and regions that control sleep and wake, in particular, the ventrolateral preoptic nucleus.[48] In addition, neuroimaging studies have shown significant elevations in activity in affect-related regions (e.g., amygdala, hippocampus, and anterior cingulate cortex) during REM sleep. Specifically, during REM sleep, due to the increased limbic activation, the emotional event would be first reactivated and then associated to previous events and processed.[49] Thus, emotions do have a role to play in the sleep derangement.

The gender differences in the sleep parameters that we noted in our study are shown in Table 3. It was noted that males have significantly prolonged RL compared to females. In a study conducted by Barrett-Connor *et al*.,[50] it was found that decreased SE, increased WASO, and decreased REM sleep were associated with low testosterone levels in the elderly. The exact mechanism underlying this association is unknown. Frequent arousals in male elderly can also be due to nocturia owing to high prevalence of benign prostatic hyperplasia seen in them. The significantly increased N1% and other non-significant yet noticeable changes in the sleep parameters like prolonged SL in female subjects compared to males can be because of hormonal changes seen postmenopause. Evidence suggests that low progesterone levels are associated with sleep difficulties and increased arousals.[39] Progesterone, injected intravenously, has direct sedative qualities resulting from stimulation of benzodiazepine receptors that stimulate the production of the non-rapid eye movement-associated gamma-aminobutyric acid receptors.[51] The effects of estrogen on sleep are somewhat more complex; however, evidence suggests that estrogen is associated with increased sleep time and decreased SL, nighttime awakenings, and arousals.[52] Considering that estrogen is also involved in temperature regulation of the body, decreased estrogen in menopause may also be associated with hot flushes, and thus increased arousals.[52,53] Further, estrogen is complexly related to melatonin and menopause-related changes in melatonin are also likely to affect sleep. A study by Toffol *et al*.[54] confirmed the decreased secretion of melatonin in postmenopausal women.

Our study has several important clinical implications. Keeping in mind the increasing geriatric population, it is important to address the sleep problems in elderly which is the most underreported yet common complaint among elderly. Not all the changes that we observed in the sleep architecture of elderly are physiological. The deviation of the sleep parameters from the normative data can be attributed to etiological factors such as daylight exposure, hormonal and nutritional deficiency, and psychosocial factors. These are modifiable and preventable risk factors which if taken care of can attenuate the deterioration of sleep parameters in the elderly. Improved sleep quality itself is a preventive and curative measure for several chronic illnesses.

To the best of our knowledge, this study is one of the few polysomnographic studies done on non-institutionalized, community-dwelling elderly who are apparently healthy. The polysomnographic method used is non-invasive and these objective measurements have added advantages over subjective parameters that were used in most of the previous studies. Since the sample is selected only from the residential areas of Bengaluru city, the results should be generalized with caution. Radiological investigations such as functional magnetic resonance imaging scans and hormonal assays could have added to the value of the study.

Further studies are required to assess the effect of interventions such as nutritional intervention, hormone replacement, or relaxation techniques on the sleep architecture of the senior citizens.

CONCLUSION

The sleep architecture in the elderly is deranged compared to the normative data. There are variations in sleep parameters among males and females as well. The cause for this deviation from the normative data may be due to altered circadian rhythm, nutritional factors, hormonal factors, or psychosocial factors. The derangements with respect to gender can be attributed to low testosterone levels and nocturia in males, and low estrogen and progesterone levels in females. Managing these factors may attenuate the sleep derangements and ensure a rejuvenating and restorative sleep in elderly.

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REFERENCES

1. United Nations Population Fund. Elderly to Constitute 20 Percent of India's Population by 2050: Report New Delhi; 2012.
2. Caruso L, Silliman R. Harrison's Principles of Internal Medicine. 17th ed., Vo. 1. USA: Mc Grawhill; 2008. p. 55.
3. Hirschowitz M. Normal human sleep: An overview. Med Clin North Am 2004;88:551-65.
4. Goldberg D, Williams P. A User's Guide to the General Health Questionnaire. Slough: NFER Nelson; 1988.
5. Rechtschaffen A, Kales A. A Manual of Standardized Terminology, Techniques, and Scoring System for Sleep Stages of Human Subjects. Washington, DC: US Department of Health Education, and Welfare Public Health Service-NIH/NIND; 1968.
6. Evans B, Rogers A. 24 hour sleep/wake patterns in healthy elderly persons. Appl Nurs Res 1994;7:75-83.
7. Richardson GS, Carskadon MA, Orav EJ, Dement WC. Circadian variation of sleep tendency in elderly and young adult subjects. Sleep 1982;5 Suppl 2:S82-94.
8. Duffy JF, Dijk DJ, Klerman EB, Czeisler CA. Later endogenous circadian temperature nadir relative to an earlier wake time in older people. Am J Physiol 1998;275:R1478-87.
9. Nakazawa Y, Nonaka K, Nishida N, Hayashida N, Miyahara Y, Kotorii T, *et al*. Comparison of body temperature rhythms between healthy elderly and young adults. Jpn J Psychiatry Neurol 1991;45:37-43.
10. Miles LE, Dement WC. Sleep and aging. Sleep 1980;3:1-220.
11. IOM (Institute of Medicine). Dietary Reference Intakes for Calcium and Vitamin D. Washington, DC: The National Academies Press; 2011.
12. Grant WB, Schwalfenberg GK, Genuis SJ, Whiting SJ. An estimate of the economic burden and premature deaths due to vitamin D deficiency in Canada. Mol Nutr Food Res 2010;54:1172-81.
13. Norman AW, Bouillon R. Vitamin D nutritional policy needs a vision for the future. Exp Biol Med (Maywood) 2010;235:1034-45.
14. Whiting SJ, Green TJ, Calvo MS. Vitamin D intakes in North America and Asia-pacific countries are not sufficient to prevent vitamin D insufficiency. J Steroid Biochem Mol Biol 2007;103:626-30.
15. Musiol IM, Stumpf WE, Bidmon HJ, Heiss C, Mayerhofer A, Bartke A, *et al.* Vitamin D nuclear binding to neurons of the septal, substriatal and amygdaloid area in the siberian hamster (*Phodopus sungorus*) brain. Neuroscience 1992;48:841-8.
16. Stumpf WE, Bidmon HJ, Li L, Pilgrim C, Bartke A, Mayerhofer A, *et al.* Nuclear receptor sites for vitamin D-soltriol in midbrain and hindbrain of siberian hamster (*Phodopus sungorus*) assessed by autoradiography. Histochemistry 1992;98:155-64.
17. Stumpf WE, O'Brien LP. 1,25 (OH)2 vitamin D3 sites of action in the brain. An auto radiographic study. Histochemistry 1987;87:393-406.
18. Stumpf WE, Sar M, Clark SA, DeLuca HF. Brain target sites for 1,25-dihydroxyvitamin D3. Science 1982;215:1403-5.
19. Eyles DW, Smith S, Kinobe R, Hewison M, McGrath JJ. Distribution of the vitamin D receptor and 1 alpha-hydroxylase in human brain. J Chem Neuroanat 2005;29:21-30.
20. Massa J, Stone KL, Wei EK, Harrison SL, Barrett-Connor E, Lane NE, *et al.* Vitamin D and actigraphic sleep outcomes in older community-dwelling men: The mrOS sleep study. Sleep 2015;38:251-7.
21. Breakthroughs, Nutrition. Insomnia: Studies Suggest Calcium and Magnesium Effective. Medical News Today. Medi Lexicon Intl 8 Sep 2009. Available from: http://www.medicalnewstoday.com/releases/163169.php. [Last accessed on 2015 Oct 26].
22. Drennan MD, Kripke DF, Klemfuss HA, Moore JD. Potassium affects actigraph-identified sleep. Sleep 1991;14:357-60.
23. Van Cauter E, Leproult R, Plat L. Age-related changes in slow wave sleep and REM sleep and relationship with growth hormone and cortisol levels in healthy men. JAMA 2000;284:861-8.
24. Weitzman ED, Zimmerman JC, Czeisler CA, Ronda JM. Cortisol secretion is inhibited during sleep in normal man. J Clin Endocrinol Metab 1983;56:352-8.
25. Spath-Schwalbe E, Uthgenannt D, Voget G, Kern W, Born J, Fehm HL. Corticotropin-releasing hormone-induced adrenocorticotropin and cortisol secretion depends on sleep and wakefulness. J Clin Endocrinol Metab 1993;77:1170-3.
26. Spath-Schwalbe E, Uthgenannt D, Korting N, Fehm HL, Born J. Sleep and wakefulness affect the responsiveness of the pituitary-adrenocortical axis to arginine vasopressin in humans. Neuroendocrinology 1994;60:544-8.
27. Gronfier C, Luthringer R, Follenius M, Schaltenbrand N, Macher JP, Muzet A, *et al*. Temporal relationships between pulsatile cortisol secretion and electroencephalographic activity during sleep in man. Electroencephalogr Clin Neurophysiol 1997;103:405-8.
28. Bierwolf C, Struve K, Marshall L, Born J, Fehm HL. Slow wave sleep drives inhibition of pituitary-adrenal secretion in humans. J Neuroendocrinol 1997;9:479-84.
29. Leproult R, Copinschi G, Buxton O, Van Cauter E. Sleep loss results in an elevation of cortisol levels the next evening. Sleep 1997;20:865-70.
30. van Coevorden A, Mockel J, Laurent E, Kerkhofs M, L'Hermite-Balériaux M, Decoster C, *et al.* Neuroendocrine rhythms and sleep in aging men. Am J Physiol 1991;260:E651-61.

31. Kern W, Dodt C, Born J, Fehm HL. Changes in cortisol and growth hormone secretion during nocturnal sleep in the course of aging. J Gerontol A Biol Sci Med Sci 1996;51:M3-9.
32. Van Cauter E, Leproult R, Kupfer DJ. Effects of gender and age on the levels and circadian rhythmicity of plasma cortisol. J Clin Endocrinol Metab 1996;81:2468-73.
33. Holsboer F, von Bardeleben U, Steiger A. Effects of intravenous corticotropin-releasing hormone upon sleep-related growth hormone surge and sleep EEG in man. Neuroendocrinology 1988;48:32-8.
34. Born J, Späth-Schwalbe E, Schwakenhofer H, Kern W, Fehm HL. Influences of corticotropin-releasing hormone, adrenocorticotropin, and cortisol on sleep in normal man. J Clin Endocrinol Metab 1989;68:904-11.
35. Cacioppo JT, Hawkley LC, Berntson GG, Ernst JM, Gibbs AC, Stickgold R, *et al*. Do lonely days invade the nights? Potential social modulation of sleep efficiency. Psychol Sci 2002;13:384-7.
36. Mahon NE. Loneliness and sleep during adolescence. Percept Mot Skill 1994;78:227-31.
37. Germain A, Caroff K, Buysse DJ, Shear MK. Sleep quality in complicated grief. J Trauma Stress 2005;18:343-6.
38. Reynolds CF 3rd, Hoch CC, Buysse DJ, Houck PR, Schlernitzauer M, Frank E, *et al.* Electroencephalographic sleep in spousal bereavement and bereavement-related depression of late life. Biol Psychiatry 1992;31:69-82.
39. Monk TH, Begley AE, Billy BD, Fletcher ME, Germain A, Mazumdar S, *et al.* Sleep and circadian rhythms in spousally bereaved seniors. Chronobiol Int 2008;25:83-98.
40. McDermott OD, Prigerson HG, Reynolds CF 3rd, Houck PR, Dew MA, Hall M, *et al.* Sleep in the wake of complicated grief symptoms: An exploratory study. Biol Psychiatry 1997;41:710-6.
41. Reynolds CF 3rd, Hoch CC, Buysse DJ, Houck PR, Schlernitzauer M, Pasternak RE, *et al.* Sleep after spousal bereavement: A study of recovery from stress. Biol Psychiatry 1993;34:791-7.
42. Hardison HG, Neimeyer RA, Lichstein KL. Insomnia and complicated grief symptoms in bereaved college students. Behav Sleep Med 2005;3:99-111.
43. Kales A, Caldwell AB, Preston TA, Healey S, Kales JD. Personality patterns in insomnia. Theoretical implications. Arch Gen Psychiatry 1976;33:1128-24.
44. Riemann D, Spiegelhalder K, Feige B, Voderholzer U, Berger M, Perlis M. The hyperarousal of insomnia: A review of the concept and its evidence. Sleep Med Rev 2010;14:19-31.
45. Harvey A. A cognitive model of insomnia. Behav Res Ther 2002;40:869-93.
46. Espie CA. Insomnia: Conceptual issues in the development, persistence, and treatment of sleep disorder in adults. Annu Rev Psychol 2002;53:215-43.
47. Perlis ML, Giles DE, Mendelson WB, Bootzin RR, Wyatt JK. Psychophysiological insomnia: The behavioural model and a neurocognitive perspective. J Sleep Res 1997;6:179-88.
48. Saper CB, Cano G, Scammell TE. Homeostatic, circadian, and emotional regulation of sleep. J Comp Neurol 2005;493:92-8.
49. Walker MP, Van der Helm E. Overnight theraphy? The role of sleep in emotional brain processing. Psychol Bull 2009;135:731-48.
50. Barrett-Connor E, Dam TT, Stone K, Harrison SL, Redline S, Orwoll E. The association of testosterone levels with overall sleep quality, sleep architecture, and sleep-disordered breathing. J Clin Endocrinol Metab 2008;93:2602-9.
51. Jehan S, Masters IA, Salifu I, Zizi F, Jean LG, Pandi PS, *et al*. Sleep disorders in postmenopausal women. J Sleep Disorders Ther 2015;4:pii: 1000212.
52. Polo-Kantola P, Erkkola R, Irjala K, Pullinen S, Virtanen I, Polo O, *et al.* Effect of short-term transdermal estrogen replacement therapy on sleep: A randomized, double-blind crossover trial in postmenopausal women. Fertil Steril 1999;71:873-80.
53. Guidozzi F, Alperstein A, Bagratee JS, Dalmeyer P, Davey M. South African menopause society revised consensus position statement on menopausal hormone therapy, 2014. S Afr Med J 2014;104:537-43.
54. Toffol E, Kalleinen N, Haukka J, Vakkuri O, Partonen T, Polo-Kantola P, *et al.* Melatonin in perimenopausal and postmenopausal women: Associations with mood, sleep, climacteric symptoms, and quality of life. Menopause 2014;21:493-500.

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