ABSTRACT
One-fourth to half of all postmenopausal women will suffer from sleep disturbances. The incidence of sleep disorders increases from 16–42% to 39–47% at perimenopause and 35 to 60% at postmenopause. The prevalence of sleep disturbance is higher in perimenopausal, postmenopausal, and surgical menopausal women than in premenopausal women. There is an independent relationship between menopausal stages and sleep disturbance beyond the effects of aging and other confounders, although the magnitude of the relationship is small. Culture, ethnicity, or both might affect the levels of sleep disturbance at various menopausal stages.

Keywords: Insomnia, Menopause, Sleep.

INSOMNIA
There is no one clear definition of insomnia in medical literature. However, there is a general consensus that insomnia consists of difficulties in either initiation of sleep, maintenance of the sleep early morning wakening and it significantly affects day time functioning. Depending on the course of the sleep disturbance, insomnia can be classified as transient (lasting only a few days before or during stressful experience), short term (lasting a few weeks during an extended period of stress or adjustment), or chronic (enduring several months or years after a precipitation event).

INSOMNIA AND MENOPAUSE
It would be reasonable to assume that if women have insomnia prior to menopause it is likely to get worse; however there are no specific reliable studies to support this. Comorbid presence of any of the independent risk make insomnia worse. In one large study there reported early morning wakening and 49% reported difficulty falling asleep. Menopausal sleep disturbance is a disorder that does not meet criteria for a specific clinical disorder and occurs in women without a baseline factor or know cause for sleep disorder. Despite adequate opportunity for sleep, there is difficulty in initiating sleep, maintaining sleep resulting in daytime functional impairments sleepiness and fatigue. Insomnia during menopausal transition generally may be due to vasomotor symptoms, such as hot flushes and sweating or due to psychopathological factors, such as mood or anxiety symptoms. There is also an opinion that the above are general change mediated through loss of estrogenic effects on neuronal modulation of energy metabolism. One recent study found that insomnia is more close related to psychological than somatic symptoms.
There are conflicting results from studies about the correlation between objective measurement of menopause, such as skin conductance and temperature measurements and subjective quality of sleep reported by women. Some studied have found positive correlation between these factors, whereas others have found mixed results.2

Although in general, women have a lower incidence of sleep syndrome sleep-disordered breathing (SDB). Obstructive sleep apnea syndrome can be more frequent in postmenopausal women as compared to premenopausal women. This can be possibly due to the protection effects of female sex steroids even when controlled for neck circumference and body mass index (BMI).

Mood and anxiety symptoms are also more common in women and can increase with age. Teasing out depression related insomnia can be different from hormonal. Early morning wakening can sometime be a clue as it is more specific for depressive illness. Up to 90% of climacteric women seeking treatment report depressive symptoms and association between depressive and vasomotor symptoms and sleep difficulties. Restless leg syndrome (RLS) and periodic limb movement (PLM) syndrome are well-known causes of insomnia and are known to increase in incidence with age this can further complicate the assessment and treatment of insomnia in menopausal age group.

Other adverse lifestyle factors sometime coaggregate during the menopausal year leading to sleep difficulties.3 There are social factors, such as changes in roles and responsibilities of women. Empty nest syndrome and adverse relationship dynamics between family members can contribute to stress and insomnia. Relationship and marital issues can affect the quality of sleep as well. Basic environmental factors, such as housing and lighting can play a part; so can occupational and financial problems. In addition to medical illnesses, general physical health, obesity, smoking and drinking were found to be risk factors for worsening of sleep according to a recent study. In fact short sleep duration has recently been found to be a significant risk factor for the development of metabolic syndrome. Side effects of medication affection quality of life can often be missed and need to be ruled put.

There is hardly any evidence about menopause and sleep problems in Indian women. However, one study indicated 40.1% of menopausal patients showing sleep-related problems. The same study shows financial constraints and family problems were the main reasons for not taking treatment for these menopausal symptoms.4

**Screening, Investigation and Diagnosis**

Despite numerous advances made in polysomnography (which includes electroencephalogram, electrooculogram and electromyogram) and other investigation modalities, insomnia still mostly remains a clinical diagnosis based on subjective complaints of patients. However, Hamilton depression rating scale (HDRS) and Hamilton anxiety rating scale (HAM-A) can be used as one of the diagnostic tools.5 If insomnia is diagnosed during menopause, causes (depression, anxiety, etc.). Validated sleep questionnaires and sleep diaries can be used to clarify the nature and pattern of sleep difficulties.

**General and Psychological Therapy**

A recent study showed that inadequate sleep hygiene was responsible for sleep disorders in menopause.7 The first lines of management instilling these sleep hygiene measures and lifestyle modification. Psychological treatments, such as cognitive behavioral therapy (CBT) have been found to result in sustained improvement in sleep (Table 1).5

**Sleep Hygiene Measures**

- **Proper sleep scheduling:** Keep regular bedtime and waking time, avoid spending excessive time in bed
- **Avoid use of sleep disrupting products:** Avoid caffeine, alcohol, nicotine before bedtime
- **Avoid stimulating activities close to bedtime:** Avoid exercise 2 hours before bedtime

**Table 1: Cognitive behavioral therapy for insomnia**

<table>
<thead>
<tr>
<th>Element</th>
<th>Description</th>
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<tbody>
<tr>
<td>Cognitive therapy</td>
<td>Aimed at changing patient’s belief and attitudes about insomnia</td>
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<tr>
<td>CBTI</td>
<td>Combined cognitive therapy and behavioral techniques; the behavioral component may include stimulus control and/or sleep restriction therapy with or without of relaxation therapy</td>
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<tr>
<td>Sleep hygiene education</td>
<td>Making patient aware of health practices and environment, but not a standard recommendation for using it as single therapy</td>
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<tr>
<td>Relaxation training</td>
<td>Progressive muscle relaxation and guided imagery rehearsal</td>
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<tr>
<td>Stimulus-control therapy</td>
<td>Training patient to reassociate bed and bedroom with sleep and re-establish a consistent sleep-wake schedule</td>
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<tr>
<td>Sleep-restriction therapy</td>
<td>Limiting time bed, creation mild sleep deprivation, and then lengthening sleep time as sleep efficiency improves</td>
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<tr>
<td>Paradoxical intention</td>
<td>Instructing patient to passively remain awake and avoid any effort to fall asleep; the goal is eliminate performance anxiety</td>
</tr>
<tr>
<td>Biofeedback</td>
<td>Visual or auditory feed to help control physiological variable to reduce arousal</td>
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<tr>
<td>Multicomponent therapy</td>
<td>Multiple behavioral techniques without cognitive therapy</td>
</tr>
<tr>
<td>CBTI: Cognitive-behavioral therapy for insomnia</td>
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</table>
• **Avoid use of bed for activities other than sleep**: Avoid watching television, reading, snacking
• Get out of bed if unable to fall asleep
• Restrict naps to 30 minutes in the early afternoon
• Exercise regularly
• Limit liquids in the evening
• **Bedroom environment**: Dim light, no television or cellphone or computer.

**Hormonal Therapy**

There is lack of consistency in studies using objectively measured sleep following hormone therapy (HT) at least partly due to differences in preparations of HT, age, symptomatology and type of menopause. Some recent studies have concluded that HT offered no significant advantage.

In a World Health Initiative (WHI) linked trial when continuous combined estrogen and progesterone therapy, 0.625 mg of conjugated equine estrogen (CEE) and 2.5 mg of medroxy progesterone acetate (MPA) were given to women aged 50 to 79 years, for 1 year. The results showed only a small benefit. In the same study, no benefit was observed in 3 years in 1511 women. The same study also revealed that HT improved moderate to severe vasomotor symptoms initially and there was a small improvement in insomnia. Another study found that combined therapy (CEE+micronized progesterone) was superior to CEE+MPA in improving objective sleep quality. One study found improvement in sleep disorders with only micronized progesterone 300 mg. Some reported improved sleep quality and mental functioning with 2 mg estradiol valerate alone or in combination with 3 mg diergost.8

One study that combined HT alleviated climacteric symptoms and improved psychological well-being, but had no effect on polysomnographically determined sleep quality.

Although the evidence of efficacy of tibolone in menopausal insomnia has been conflicting. One recent study showed definite improvement.

**OBSTRUCTIVE SLEEP APNEA**

This is characterized by repetitive upper airway collapse during sleep. The diagnosis is made by nocturnal polysomnography, it measures the total number apneic and hypopneic episodes per hour of sleep [the apnea-hypopnea index (AHI)]. The American academy of sleep medicine has defined obstructive sleep apnea (OSA) as mild (AHI 5–15), moderate (AHI > 30) based on the frequency of these events.

Repetitive intermittent hypoxia from OSA also produces oxidative stress and inflammation resulting in pathologic vasoconstriction and an increased incidents of atherosclerosis. Diabetics have a high prevalence of OSA, and results in worsening of diabetics control, and continuous positive airway pressure (CPAP) treatment improves glucose control in these patients. Insulin-resistant women with polycystic ovary syndrome are at high-risk for OSA, early-onset. Diabetics and cardiovascular disease; CPAP treatments improves insulin sensitivity, decreases sympathetic nervous system outflow, and decreases diastolic blood pressure in these patients. Cognitive dysfunction, including dementia, memory loss depression and anxiety, also can result from untreated OSA. The classic symptoms are loud snoring, sleep apnea during sleep and excessive daytime sleepiness but may present the symptoms, such as insomnia, morning headache, fatigue, tiredness, depression and anxiety. The atypical presentation is more common in women.

**Physical Examination**

It would include measurement of neck size (>16”) a BMI above 30 kg/m² and presence of structural abnormalities (oropharyngeal narrowing, retrognathia, macroglossia, uvula elongation, high arched hard palate, nasal-septa deviation.

The risk factors are obesity, ethnicity, genetics for frontomaxillary bone structure precipitates OSA. Obesity may be most important factor for OSA. In a study of women BMI above 30 kg/m² one-third of asymptomatic women were found to have OSA by polysomnographic criteria, and a significant correlation was identified between AHI and BMI in this cohort.

Hormonal therapy is not a recommend treatment for premenopausal OSA at this time. Further long-term outcomes studies are needed to evaluate the net effect of HT on postmenopausal women with OSA.10,11

**MANAGEMENT**

Obstructive sleep apnea is a chronic disease requiring long-term, multidisciplinary management. Medical, behavioral and surgical treatments are the options and need patient cooperation. Positive airway pressure (PAP) therapy is the treatment of choice for all degree of OSA. provides pneumatic splinting of the upper airway and is effective in reducing AHI. It may be delivered in CPAP, bilevel PAP, or autotitrating PAP modes.

Behavioral therapy including sleep hygiene, weight reduction, exercise, alcohol avoidance are a vital part of the treatment oral appliances (mandibular repositioning or tongue relating devices) may improve upper airway patency.

Surgical option is site-directed and patient-based. Nasal (septoplasty, turbinate reduction, nasal polypectomy), oropharyngeal, and global (maxilla-mandibular advancement) airway procedures can be considered.
based upon patient anatomy in individuals who fail less-invasive treatment.

RESTLESS LEG SYNDROME

The prevalence ranges from 4 to 29% in the general population and increases with age. The overall prevalence is higher among women (13.9%) than men (6.1%). The cause of this disease is unknown: dopaminergic dysfunction is thought to be responsible. Restless leg syndrome reduces the quality of life may predispose to significant morbidity and excess mortality.

REFERENCES