Sleep in Patients with Restrictive Lung Disease

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KEYWORDS

• Restrictive lung disease • Interstitial lung disease • Sleep • Sleep-disordered breathing
• Hypoxemia

KEY POINTS

- Restrictive lung disease is associated with nocturnal pathophysiology, including sleep disturbances and breathing and oxygenation impairments during sleep.
- Sleep is disrupted because of changes in sleep architecture and comorbid sleep disorders.
- Sleep changes in restrictive lung diseases affect sleep quality and contribute to daytime fatigue in this population.
- Little is known about the impact of treatment of sleep disorders and sleep disruption on sleep quality and daytime complaints in restrictive lung disease.

Restrictive ventilatory defects occur from several pathologic mechanisms. Intrinsic lung disease such as pulmonary fibrosis and other interstitial lung disease (ILD) may cause reduced lung volumes as well as diffusion impairment. Restrictive ventilatory defects may also occur due to musculoskeletal abnormalities in the thoracic cage or due to respiratory muscle weakness from neuromuscular disease. In addition, obesity may cause significant restrictive lung physiology and lead to obesity hypoventilation syndrome (OHS). Restrictive lung disease is associated with nocturnal pathophysiology, including sleep disturbances and breathing and oxygenation impairments during sleep.

INTERSTITIAL LUNG DISEASE

Fatigue is a common complaint among patients with ILD. Sleep is disrupted due to respiratory pathophysiology such as nocturnal hypoxemia, changes in sleep architecture, and comorbid sleep disorders. These sleep changes in ILD affect sleep quality and contribute to daytime fatigue in this population.

Respiratory Physiology During Sleep

Sleep onset is normally characterized by reduced responsiveness to hypercapnia and hypoxemia, as well as reduced cortical and lung mechanic responsiveness. As a result, minute ventilation decreases from a reduction in tidal volume, whereas respiratory rate generally remains unchanged.[1,2] It is unclear whether patients with restrictive pathophysiology due to intrinsic lung disease experience a similar effect. During wakefulness, patients with ILD often have rapid, shallow breathing as a result of decreased lung compliance and a sensation of dyspnea due to afferent stimulation from vagal receptors.[3] Few studies suggest subjects with ILD may experience persistently elevated respiratory rates with shorter inspiratory and expiratory durations during sleep. The lack of change from wake to sleep state may be attributed to the maintenance of

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vagal-mediated reflexes causing hyperventilation during wakefulness.[4,5]

Others have found a significant reduction in respiratory rate with sleep onset in patients with ILD.[6] To eliminate the potential confounding of nocturnal hypoxemia on differences in ventilatory pattern in patients with ILD, Shea and colleagues[7] studied breathing patterns in patients with ILD whose oxygen saturations were maintained greater than 95% by breathing supplemental oxygen. During the wake state, respiratory frequency was higher and inspiratory and expiratory times were shorter among patients with ILD despite adequate oxygenation compared with normal subjects, supporting a respiratory stimulatory effect independent of hypoxemia during the daytime. During sleep, and specifically in slow wave sleep, however, there was no difference in the respiratory rate between normal controls and subjects with ILD, suggesting hyperventilatory responses during wakefulness are not maintained during sleep in subjects with ILD. Midgren and colleagues[8] similarly found greater hyperventilation reflected by reduced transcutaneous partial pressure of carbon dioxide ($Ptcco_2$) in patients with ILD compared with controls. However, during sleep, both controls and patients with ILD had comparable $Ptcco_2$, suggesting hyperventilation is ameliorated in patients with ILD during sleep.

The pattern of breathing may also be more inefficient and labored during sleep in patients with ILD. Hira and Sharma[9] showed through respiratory inductance plethysmography that patients with ILD produce similar tidal volume per rib cage and abdomen excursion during wakefulness. However, during sleep, patients with ILD required more excursion or effort per tidal volume compared with healthy controls.

Physiologically, respiratory muscles with the exception of the diaphragm become atonic during rapid eye movement (REM) sleep. Moreover, during phasic REM sleep, inspiratory drive is reduced. These normal physiologic changes are associated with only minor gas exchange abnormalities in healthy individuals; however, these changes may lead to profound hypoventilation and hypoxemia in patients with restrictive lung disease. It is not uncommon to observe sustained desaturations during REM sleep and pulse oximeter oxygen saturation (Spo_2) reaches a nadir of less than 70%.[5] Hypoxemia during REM sleep tends to be worse in those with more severe daytime hypoxemia.[4,5] The degree of desaturation during REM sleep is often more severe than that occurring during exercise.[5]

Nocturnal desaturation has been shown to be common in ILD and associated with worse clinical outcomes. Hira and Sharma[9] reported an average decline in Spo_2 of nearly 9% during sleep compared with approximately 4% among normal controls. Spo_2 reached a nadir by 10% to 16% in patients with ILD compared with 3% to 6% in healthy controls. Patients with ILD spent 17% of total sleep time with Spo_2 less than 85%, whereas none of the controls had such desaturations during sleep. Corte and colleagues[10] found that 37% of their subjects with ILD spent more than 10% of sleep with Spo_2 less than 90%. In their study, the desaturation index, defined as the number of desaturation events greater than 4%, was shown to be independently associated with mortality. Similarly, Medeiros and colleagues[11] found in a series of women with lymphangioleiomyomatosis that nocturnal hypoxemia (again defined as 10% total sleep time with Spo_2 <90%) occurred in 56% of patients and sleep time spent with Spo_2 less than 90% was associated with worse diffusion capacity and forced expiratory volume more than 1 second, as well as increased residual volume to total lung capacity ratio.

In contrast, McNicholas and colleagues[6] studied 7 patients with severe ILD and found none had significant complaints of sleep-disordered breathing (SDB), and, although 2 patients were found to have apneas, neither had greater than 3% desaturations associated with apneic episodes. The investigators concluded that nocturnal hypoxemia in this population is not severe and not of clinical relevance. However, Midgren[12] found nocturnal hypoxemia was in fact severe in patients with ILD, although to a lesser degree than that observed in patients with chronic obstructive lung disease. Similar to other studies, mean Spo_2 was reduced to a greater extent during REM sleep than during non-REM sleep in all 3 groups, although the difference was the least in the ILD group.

Sleep Architecture/Disturbances

Patients with ILD often have disrupted sleep and frequent arousals. Sleep in these patients is characterized by an increase in arousals, increased stage N1 and stage N2 sleep, and a decrease in stage N3 and REM sleep. Perez-Padilla and colleagues[4] showed stage N1 sleep comprised 33% of total sleep time in patients with ILD compared with 14% in controls. Sixty-five percent of patients with ILD did not have slow wave sleep, and not only was REM sleep delayed in onset but REM time was also only 12% of the total sleep time compared with 20% in healthy controls. There were also more frequent sleep stage shifts in the ILD group. Sleep architecture changes were

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