Dementia and Parkinsonism

Dementia refers a loss of higher cortical functions due to a chronic, progressive degenerative disease process of the brain. Dementing illnesses include a wide variety of nonreversible conditions, including Alzheimer's disease (AD), Pick's disease, and certain forms of hydrocephalus. AD is the most common cause of dementia, accounting for as high as 70% of all cases (Bliwise, 1992). Sleep disturbances in demented patients are very common. Their severity appear to parallel the progression of the underlying illness and their pathophysiology is probably related to the dementia process itself. In addition, demented patients are often treated with psychotropic medicines, and these drugs may worsen sleep disturbances and daytime symptoms. Nighttime behavior of demented patients is characterized by delirium, agitation, and sundown syndrome (ASDA, 1997). *Sundowning* refers to the phenomenon of agitation strongly associated with darkness and is characterized by nocturnal wandering and confusion. Polysomnographic studies of AD patients have documented altered sleep architecture including decreased sleep efficiency, slow-wave and REM sleep, increased Stage 1 sleep, and an increase in the number of and duration of nocturnal awakenings (Prinz, 1995). Some patients also exhibit a fragmented 24-hour sleep-wake pattern in the form of partial or complete reversal of the sleep-wake schedule, with increased wakefulness at night and somnolence during the daytime (Chokroverty, 1996).

Parkinsonism refers to a group of neurological disorders characterized by resting tremor, akinesia (reduced spontaneous movement), postural rigidity, and bradykinesia (slowness of movement). Sleep disturbance is reported by 60% to 90% of patients with Parkinson's disease (PD) and it tends to gradually worsen with disease progression. Insomnia is the most common complaint in these patients. The inability to get out of or turn over in bed, nocturia, painful leg cramps, back pain, limb jerks, central or obstructive sleep apnea, and medication sleep effects can all cause insomnia, sometimes with concomitant daytime sleepiness (Aldrich, 1994, ASDA, 1997; Partinen, 1997).

Characteristic polysomnographic abnormalities in PD patients include increased sleep latency, Stage 1 sleep, and the number and duration of nocturnal awakenings as well as reduced

amounts of REM and slow-wave sleep. Sleep disordered breathing (various forms of sleep apnea), REM sleep behavior disorder (RBD), and periodic limb movement disorder (PLMS) are prevalent in PD patients and can further disrupt their sleep or produce daytime sleepiness. While levodopa treatment of Parkinsonism can improve sleep disturbance due to such symptoms of the disease as rigidity or bradykinesia, it may at the same time cause new symptoms (e.g., insomnia, vivid dreaming, nocturnal vocalizations, myocolonic movements, dyskinesias). Depression, which is a common comorbid illness in PD patients, can also contributes to sleep disturbance (Chokroverty, 1996).

Sleep-Related Epilepsy

Sleep-related epilepsy is associated with either abrupt awakenings, unexplained urinary incontinence, or abnormal muscle activity during sleep. To be considered sleep-related epilepsy, 75 percent or more of the seizures must occur during sleep and at least two other features must be present (i.e., automatisms, specific limb movements, or tongue biting) (ASDA, 1997). Individuals suspected of this disorder usually undergo daytime EEG monitoring, nocturnal polysomnography with video monitoring, and possibly CT or MRI scanning.

Sleep-Related Headaches

Cluster or migraine headaches occurring during sleep seem to be related to REM sleep. Breathing disturbances during sleep or alcohol ingestion may bring on the cluster headaches. High levels of daytime stress, weather change, or other events may precipitate migraine headaches during sleep. Individuals typically awaken from sleep, usually late in the sleeping period and out of a REM period, aware of the presence of the headache.

Sleep Disorders Associated With Medical Disorders

There is a broad range of physical illnesses that affect sleep. Our discussion is selective and focuses on respiratory disorders, nocturnal cardiac ischemia, chronic pain, fibromyalgia, and AIDS.

Respiratory Disorders

Respiratory disorders that affect sleep include disorders of respiratory control, disorders of the respiratory musculature, and disease of the airways and lung parenchyma. Respiratory disorders often worsen during sleep causing sleep disturbances. The severity of sleep disturbances appears to correlate with the progression of the underlying pulmonary disease. Chronic obstructive pulmonary disease (COPD) and asthma are the respiratory disorders which frequently lead to sleep complaints.

COPD is characterized by a chronic reduction in airflow, with symptoms of dyspnea and cough being most common. Hypoxemia, hypoventilation, and impaired lung function are the physiologic consequences. A high percentage of patients with COPD experience difficulty in initiating and maintaining sleep (Douglas, 1994a). Shortness of breath in the recumbent position and frequent episodes of coughing keep COPD patients from falling asleep. Severe sleep fragmentation, again due to respiratory symptoms, often leads to a unrefreshed feeling upon awakening and excessive daytime sleepiness. Polysomnographic features include decreased total sleep time, amount of slow-wave and REM sleep, frequent sleep-stage changes, and increased number of arousals and awakenings. COPD patients become more hypoxemic during sleep, especially in REM sleep, than during resting wakefulness, because of hypoventilation, worsening ventilation perfusion mismatch, the loss of accessory muscles, and the medicines used to treat COPD, particularly the xanthine derivatives such as theophylline, can also contribute to sleep disturbance (ASDA, 1997; Douglas, 1998).

Patients with asthma have reversible reduction in airflow. The reduction can be abrupt and dramatic, triggering acute attacks of wheezing, shortness of breath, coughing, and chest tightness. In general, asthmatic individuals have hyperreactive airways. Exposure to allergens or irritating stimulus produces airway obstruction, secondary to spasm of the bronchial muscles as well as increased secretion and, if exposures are recurrent, bronchial mucus hyperplasia.

Bronchodialators, such as beta against drugs (albuteral, solmeterol) or theophylline compounds ameliorate asthma symptoms but may cause increased wake time during sleep. Many

asthmatics patients have symptoms at night, as in studies of these patients, sixty-one to 74 report nighttime awakenings due to asthma attack. Unrestorative sleep and daytime fatigue are also common complaints of asthmatics. Polysomnographic studies reveal that nocturnal asthma attacks rarely occur in slow-wave sleep and tend to cluster in the later part of the sleep period, perhaps as the bronchodialecting effects of medicine taken before bed declines. Hypoxemia with attacks is rarely severe (ASDA, 1997; D'Ambrosio & Mohsenin, 1998). Compared to sleep of controls, asthmatics patients have less total sleep time, lower sleep efficiency, and more number of awakenings (Montplaisir et al. 1982).

Nocturnal Cardiac Ischemia

The relationship between sleep and cardiac ischemia is controversial because the factors which precipitate nocturnal cardiac ischemia are not clear. It may occur as the result of increased myocardial oxygen demand, decreased cardiac perfusion, or increased filling of the left ventricle as a result of lying in a recumbent position (Motta & Guilleminault, 1985).

Symptoms of nocturnal cardiac ischemia include the feeling of pressure during sleep that is often described as a "clenched fist." Pain often radiates to the left arm and upwards into the neck or jaw (ASDA, 1997). These symptoms may be present during waking hours but result from activity.

The prevalence of nocturnal cardiac ischemia is unknown. However, patients with obstructive sleep apnea may experience severe oxygen desaturation and have a higher incidence of cardiac ischemia than the general population (Burack, 1984). Ischemia can also cause severe sleep fragmentation in the absence of sleep apnea (Schafer, Koehler, Ploch, & Peter, 1997). Electrocardiographic changes that occur in the early morning hours are typically associated with REM sleep while changes that occur at the beginning of sleep are typically associated with a fall in blood pressure and heart rate.

The presence of ischemia on the electrocardiogram is indicative of coronary artery disease. Other possible precipitating factors to nocturnal ischemia include hypertension, elevated blood

cholesterol and low-density lipoprotein, cigarette smoking, obesity, and sleep-induced hypoxemia (ASDA, 1997).

Chronic Pain

Chronic pain is a common cause of sleep complaints, especially in the elderly. Rheumatological disorders such as osteoarthritis, a degenerative joint disease, tend to be frequent causes of insomnia. During the day the joints are kept mobile which helps to maintain flexibility and reduce pain. These joints become stiff at night and sleep may be disturbed by sudden pain when the individual moves or attempts to turn over in bed (Morgan, 1987). Adequate dosage of anti-arthritic medication before bedtime can greatly alleviate the problem.

Other chronic pain conditions that may precipitate complaints of insomnia include burning foot pain from diabetes and chest and epigastric pain due to angina, reflux, esophagitis, or peptic ulcer disease. Cancer patients often exhibit more severely disturbed sleep patterns than either normals or other patients with medical conditions. Complaints of insomnia are often reported with feelings of anxiety or depression rather than pain which has lead to the suggestion that antianxiety drugs may be a consideration to help improve sleep (Anch, et al., 1988).

Fibromyalgia

Fibromyalgia (FMS) is a central nervous system disorder of unknown etiology. Its main symptom is widespread musculoskeletal pain with tenderness in at least 11 of 18 specific anatomic sites. In addition, symptoms of fibromyalgia include fatigue and dyssomnia, with unrefreshing sleep, and higher than average prevalence of alpha sleep (Ware, Russell, & Campos, 1986; Moldofsky, et al., 1975) in which an alpha EEG frequency wave occurs simultaneously with slower EEG waves. Compared with controls, fibromyalgia patients have less delta (.5 - 3.5) and theta (3.5, 8 Hz ) and more alpha 8-12 Hz and sigma (12-14.5 Hz) in all stages of sleep but especially so during NREM sleep (Drewes, et al., 1995).

It has been proposed that alpha sleep may cause the musculoskeletal symptoms of fibromyalgia (Moldofsky & Scarisbrick, 1976) and that it may be involved more generally in disorders that affect the immune system. Not surprisingly, there is a strong association between

fibromyalgia disease severity, based on clinical examination as well as on average daily self-rating of pain, and disturbed sleep as indicated by lower sleep efficiencies and greater morning difficulties (Manber, et al., 1998).

Acquired Immune Deficiency Disorder (AIDS)

Approximately one-third of asymptomatic HIV-seropositive individuals report sleep onset or maintenance difficulties (Rothenberg, et al., 1990). These differences are not attributable to those taking zidovudine (AZT) (Moeller, Wiegand, Oechsner, Krieg, Holsboer, & Emminger, 1992). Polysomnographic studies of asymptomatic individuals have often, but not always, found increased sleep fragmentation, increased amounts of slow wave sleep, slow wave sleep distributed throughout the night, and an alpha sleep pattern (Norman, et al., 1992; Wiegand, et al., 1991). Abnormal sleep architecture was evident at a 1 year follow-up (Norman, Chediak, Kiel, Gazeroglu, & Mendez, 1990).

## CONCLUSION

As indicated throughout this chapter, an understanding of sleep and sleepiness is crucial to an understanding of all behavior, including psychopathology. Sleep is a sensitive barometer of the quality of our lives. It is affected by, and in turn affects, developmental, physiological, psychological, and sociocultural processes. To understand sleep disorders, research must continue to be pursued as a multidisciplinary enterprise. This chapter indicates that there have been substantial advances in our understanding of the etiology and treatment of sleep disorders. This has included advances in the neurophysiology and pharmacology of sleep, our understanding of the sleep-wake circadian rhythm, and behavioral treatments for sleep disorders. But, this is a field in its infancy and we eagerly anticipate the advances still to come.

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