Psychiatric aspects of organic sleep disorders

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In recent years, a number of studies have attempted to characterize psychological disturbances related to various sleep disorders. The objective of this type of research is to investigate the possibility that psychopathology may represent an etiological factor, a complication, and/or a target for treatment. In addition, disordered sleep can present itself in a complex and atypical fashion in which the primary sleep-related component may not be immediately apparent. This article reviews the evidence for a relationship between organic sleep disorders and psychiatric morbidity. Generally, it can be concluded that organic sleep disorders have a profound negative impact on most domains of health-related quality of life. Results for the sleep disorders that have been studied (narcolepsy, idiopathic hypersomnia, sleep apnea/hypopnea syndrome, restless legs syndrome, periodic limb movement disorder, and circadian sleep disorders) show strong evidence for an association with mood disorders. After treatment, depression scores may or may not improve to the level of population norms, suggesting that this relationship is more complex than one of mere cause and effect.

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The discussion of the relationship between sleep and psychiatric states is not new. Sleep disorders medicine and psychiatry are related in numerous ways. Even though most psychiatric patients have a complaint about sleep, a primary sleep disorder may also result in neuropsychiatric complications. In fact, psychiatric morbidity is very high in patients seen in the sleep disorders clinic. In 1989, Mosko et al[1] showed that 67% of patients who presented to a sleep disorders center reported an episode of depression within the previous 5 years, and 26% described themselves as depressed at presentation. The high incidence of depressive feelings in patients with a sleep complaint was true not only of patients with insomnia, but also for those with organic sleep disorders (such as obstructive sleep apnea/hypopnea syndrome [OSAS], narcolepsy, or periodic leg movements during sleep [PLMS]). In a more recent survey, Vandeputte and de Weerd[2] also found that mood disorders are extremely common in patients who present at a sleep center. These authors analyzed data from 917 consecutive patients (excluding those with clinically overt depression) and found elevated scores of depression in patients diagnosed with psychophysiological insomnia (60.5%), but also in OSAS (41%), narcolepsy (37%), periodic limb movement disorder/restless legs syndrome (PLMD/RLS) (53%), inadequate sleep/wake hygiene (63%), delayed sleep phase syndrome (DSPS) (41%), snoring (31%), sleep state misperception (63%), parasomnia (29%), idiopathic hypersomnia (27.5%), and advanced sleep phase syndrome (83%). Although the prevalence of

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Selected abbreviations and acronyms

CPAP	*continuous positive airway pressure*
DSPS	*delayed sleep phase syndrome*
MMPI	*Minnesota Multiphasic Personality Inventory*
OSAS	*obstructive sleep apnea/hypopnea syndrome*
PLMD	*periodic leg movement disorder*
PLMS	*periodic leg movements during sleep*
REM	*rapid eye movement*
RLS	*restless legs syndrome*
SF-36	*Short Form 36 Health Survey*

depression in these patients is higher than in the general population, it can be argued that depression and a sleep disorder in the same patient may be a mere coincidence, given that psychiatric illness and sleep disorders are frequent in the general population. However, there is often evidence for a causal relationship between depression and the sleep disorder. For example, depression scores can be significantly improved following conventional treatment, suggesting that the primary sleep disorder was at the origin of the mood disturbance.[1] On the other hand, the assumption that psychiatric symptoms are always reactive to sleep disorders, secondary to sleepiness and fatigue, is probably too crude. For example, treatment of OSAS with continuous positive airway pressure (CPAP) can leave patients with residual sleepiness or fatigue, which may be a result of depression.[3]

Until now, studies on the prevalence of psychiatric comorbidity in the various sleep disorders have focused mainly on OSAS and narcolepsy. Studies in other common organic sleep disorders are scarce. The aim of this article is to review the evidence for a relationship between the various organic sleep disorders and psychiatric morbidity.

Narcolepsy

Narcolepsy is a chronic neurological disorder affecting sleep regulation. Narcolepsy is not a rare condition: its prevalence, about 0.05%, varies between countries because of genetic factors.[4] The classic clinical tetrad for narcolepsy include excessive daytime sleepiness, cataplexy, sleep paralysis, and hypnagogic hallucinations.[5] Patients experience a constant abnormal daytime sleepiness, which has been compared to the sleepiness one feels when trying to complete a boring task at 3 AM after 72 h of sleep deprivation.[6] Cataplexy refers to partial or generalized loss of skeletal muscle tone in response to emotion, especially joy or anger. Sleep paralysis refers to the inability to move at the beginning or the end of sleep. Finally, patients can present hypnagogic hallucinations, vivid dream-like experiences at the start of sleep, which can accompany sleep paralysis. People with narcolepsy enter rapid eye movement (REM) sleep more quickly than usual (sometimes immediately) when they fall asleep. Cataplexy, sleep paralysis, and hallucinations represent intrusion of REM sleep into wakefulness.

The impact of narcolepsy on psychosocial functioning has been long recognized. A detailed survey comparing life effects of narcolepsy in 180 subjects matched with local controls and drawn from centers in Canada, Japan, and Europe is a classic study in this area.[7] Occupational problems were prevalent in this study (over 75%) and included deleterious effects upon performance, promotion, earning capacity, fear of or actual job loss, and increased disability insurance. Work or home accidents attributed to sleepiness or sleep (49%) or related to smoking (49%) were much more common in these patients. There were also deleterious effects on education, recreation, and personality related to disease. A similar pattern of impairment of health status has been shown using the Short Form 36 Health Survey (SF-36) by Beusterien et al[8] in 481 narcoleptics who were not taking any stimulant medication. Compared with the general population, subjects with narcolepsy are most profoundly affected in vitality, social functioning, and difficulty when performing usual activities due to physical and emotional problems. Patients suffering from narcolepsy experience health-related quality of life effects as bad as or worse than patients with Parkinson's disease, epilepsy, or migraine. These extensive emotional and psychosocial correlates of narcolepsy have also been confirmed in other studies.[9,10]

Broughton et al[7] also outlined the difficulties in driving encountered by narcoleptics. Patients fell asleep at the wheel more frequently (66%) and had near or actual road accidents due to drowsiness or falling asleep (67%). The proportion of narcoleptics reporting sleep-related motor vehicle accidents is four times more than in controls.[11] These findings are confirmed by studies using a computer driving simulation task,[12-14] in which performance improves with methamphetamine treatment.[15]

Finally, approximately half of patients with narcolepsy suffer from subjective memory problems, mainly involving recent events.[7] In various studies, subjective memory complaints were not related to objective findings,[16-20]

although patients had more difficulties maintaining attention, suggesting that their deficits are not cognitive in nature, but represent an inability to maintain wakefulness and produce a sustained performance.

All these data illustrate the breadth of the impact of narcolepsy, and accumulation of all these effects can cause deterioration in emotional health and an increased vulnerability to psychiatric disorders. Narcolepsy has thus often been associated with psychiatric disturbance, but surveys have produced contradictory results. Schizophrenia has been found at rates ranging from 0% to 14% and depression at rates ranging from 5% to 30%.[9,21-23] Krishnan et al,[23] for example, showed that two thirds of narcoleptic patients had psychiatric disorders according to *Diagnostic and Statistical Manual of Mental Health, Third Edition (DSM-III)* criteria, including adjustment disorder, major depressive episode, alcohol dependence, and personality disorder. However, those studies were made in small samples,[22,23] with no control group,[21-23] often based on case notes and in patients taking amphetamines, which were, until recently, the mainstay of treatment for narcolepsy. The well-documented side effects of amphetamines (including increased feelings of anxiety, irritability and agitation, sexual dysfunction, and insomnia) may exacerbate existing or underlying psychiatric conditions.[24]

In a recent study, Vourdas et al[25] investigated the frequency of major and minor psychiatric disorders among patients with narcolepsy, as compared with a group of matched normal individuals, using a detailed structured psychiatric diagnostic interview. This study took advantage of the recent introduction of the wake-promoting drug modafinil, which improves vigilance via a non-dopaminergic/adrenergic mechanism and does not appear to induce psychosis. This study found little evidence for an increased frequency of psychotic disorders in narcolepsy. Although four patients (out of 45) had experienced episodes meeting criteria for probable psychotic disorder (in contrast to none of the controls), there were clear indications that the psychotic symptoms were related to amphetamine use in the past, since they disappeared when the dose was lowered or medication was changed to modafinil. None of the patients taking modafinil showed psychotic symptoms. They found that 24% of patients had criteria for simple depression, a rate similar of that found in other surveys in narcolepsy[1,2,7] and chronic medical disorders in general.[26]

Although some studies have shown a possible therapeutic role of stimulant medication for improving affect,[27] others have shown that depression in narcolepsy is independent of pharmacological treatment or did not improve after treatment.[1] Goswami[28] reported that, despite treatment for excessive daytime sleepiness, narcolepsy patients remain at significant risk for psychiatric and psychosocial limitations. Beusterien et al[8] have shown that treatment with modafinil produces higher scores than placebo for the physical role, energy/vitality, social function, and emotional role on the SF-36. This means that improvements with modafinil were seen not only in physical functioning and productivity, but also in aspects of psycological well-being. In an open-label study, Becker et al[29] also found that treatment with modafinil resulted in significantly decreased total mood disturbance. It should be pointed out that several preliminary reports show the utility of modafinil as an adjunctive treatment for depressed patients with complaints of significant fatigue and/or excessive sleepiness.[30-35] However, despite significant improvements compared with pretreatment, the majority of scores did not return to normal. Some authors have indeed suggested that depression may be endogenous to narcolepsy,[7,36] as abnormalities in REM sleep, such as reduced REM sleep latency, are common to depression[37] and narcolepsy.

Finally, it is worth mentioning that narcolepsy cases in which the hallucinatory component is unusually prominent may lead to the diagnosis of schizophrenia. Douglass et al[38] described five narcoleptic cases in which "psychotic symptoms" dominate the symptomatology. Conventional antipsychotic drugs were ineffective, and led the investigators to reconsider the diagnosis. The diagnosis of narcolepsy was ultimately confirmed and treatment with stimulants produced substantial improvement. It seems clear that the hypnagogic and other hallucinations of narcolepsy could cause difficulties with the differential diagnosis from schizophrenia and, vice versa, narcolepsy should be considered in the differential diagnosis of hallucinations of possible psychotic origin. The hallucinations in narcolepsy are in general visual; sleep paralysis can be associated; and these usually occur when the patient is half-asleep.

Idiopathic hypersomnia

Idiopathic hypersomnia is a rare condition. Its prevalence is about 10 times less than narcolepsy and it usually develops before the age of 30. In its polysymptomatic form, idiopathic hypersomnia is characterized by the fol-

lowing: excessive daytime sleepiness (not as irresistible as in narcolepsy, but usually lasting much longer); nocturnal sleep of abnormally long duration; signs of "sleep drunkenness" (difficulties in coming to complete wakefulness accompanied by confusion, disorientation, poor motor coordination, and slowness); and long and unrefreshing naps. The poorly defined monosymptomatic form manifests itself only by excessive daytime sleepiness.[39] Complications are mostly social and professional, including poor work performance, reduced earning capacity, poor results at school, impaired ability to enjoy recreational activities, frequent accidents, and deteriorated memory for recent events.[40] Due to these effects, it is possible to infer the possible psychological impact of the condition in the affected subjects, though no definitive conclusions can be made considering its frequency and the small series of published cases. In their survey, Bassetti and Aldrich[41] reported a lifetime prevalence of psychiatric symptoms (anxiety and depressive symptoms) in 57% of patients. It seems likely that most of the psychiatric symptoms in these patients are nonspecific responses to chronic illness, rather than essential elements of idiopathic hypersomnia. In certain cases, the clinical picture of idiopathic hypersomnia can be confused with "atypical depression."

Obstructive sleep apnea/hypopnea syndrome

OSAS is a frequent and probably insufficiently recognized condition, characterized by recurrent episodes of complete or partial obstruction of the upper airway, often resulting in oxygen desaturation and arousals from sleep. The classic daytime manifestation is excessive sleepiness, but other symptoms, such as unrefreshing sleep, fatigue, or impaired concentration, are commonly reported.[42] It is estimated that 4% of middle-aged men and 2% of middle-aged women in the general population meet minimal criteria for OSAS.[43] Several epidemiological and community-based studies have shown that OSAS is associated with cardiovascular and cerebrovascular morbidity.[44,45] Patients with OSAS also have increased risk of work-related and road accidents.[46-48]

OSAS is accompanied by significant cognitive and behavioral dysfunctions. Deficits have been observed especially in the area of attention and memory. Moreover, some studies have suggested executive dysfunction, assumed to be related to prefrontal lobe dysfunction caused by intermittent hypoxia.[49,50]

Although OSAS has been linked to anxiety,[51-53] nocturnal panic attacks,[54] and psychotic episodes,[55] it is with depression that it has been the most frequently associated. In fact, depressive symptoms are considered to be a typical clinical manifestation of OSAS,[56] though the nature of the relationship is poorly understood. Right from the initial studies in this field, mood disorders were described as significantly more frequent in OSAS than in the general population. In an early report, Guilleminault et al[57] showed that 28% of patients with sleep apnea had elevated depression scale scores on the Minnesota Multiphasic Personality Inventory (MMPI). Over the past few years, the burgeoning interest in psychopathological changes in patients with OSAS has resulted in a large increase in the number of published studies on this topic. Most of these studies have confirmed the elevated rates of depression, ranging from 20% to 63% in untreated patients.[51,58-62] However, some researchers have failed to find pathological levels of depression or only relatively mild depressive symptoms.[63-68] This discrepancy may be due, in part, to the types of approach used to assess depression and the inhomogeneity of the studied populations. Overall, studies using structured clinical interviews and the *DSM* criteria show rates of current depressive episode in around one-third of untreated patients.

When we consider the incidence of mood disorders in patients with OSAS, one important question is whether the incidence of these psychopathological changes is related to the disease itself or whether they are the result of other variables related to sleep fragmentation and apnea. It is clear that the sleep fragmentation related to respiratory events can lead to feelings of fatigue, lack of energy, and irritability, which are symptoms commonly reported in OSAS, and also somatic symptoms found in depressive states. In other words, many symptoms of clinical depression (sleep problems, fatigue, concentration difficulties, irritability, and social withdrawal) overlap with the symptoms of OSAS. In OSAS, general psychopathology and depression scores has been related to the arterial oxygen desaturation,[60,69] the severity of the disease (measured by the apnea/hypopnea index),[70] the degree of sleep perturbation,[62] the patient's age and body mass index,[71] the REM latency, and the use of antihypertensive drugs.[58] However, several studies agree that higher depression scores show a strong association with reduced daytime alertness; thus patients reporting higher daytime sleepiness are more likely to report higher

depression.[62,63,72,73] Sleepiness thus seems to have important effects on mood in apneic patients.

Patients with OSAS had impaired quality of life when assessed by the Functional Outcomes of Sleep Questionnaires,[74] the Calgary Quality of Life Index,[75,76] the Nottingham Health Profile,[77-80] or the SF-36.[81-84] In particular, the SF-36 domains of vitality, emotional role, mental health, and social functioning are consistently rated lower by sleep apnea patients, and are responsive to CPAP treatment.[42] The impaired quality of life derived from OSAS may be so severe that job performances and family and social life may be affected, leading in turn to emotional disturbances and personality changes. Thus, we can expect the lower perception of functional and emotional well-being to be a factor of vulnerability to depression. Although the determinants involved in the effect of OSAS on health status are not fully explored, Sforza et al[72] showed that, while objective assessment of OSAS severity (hypoxemia, apnea/hypopnea index, and sleep fragmentation) has a small impact on physical functioning, obesity and daytime sleepiness contributed more significantly to impairment on all domains of the SF-36 questionnaire. The results of this study suggest that the consequences of OSAS on health-related quality of life should be considered as a multifactorial phenomenon, but that at least some of the psychophysiological consequences of OSAS reflect the consequences of sleepiness. These data strongly suggest that the relationship between OSAS and depression should be regarded as a mood disorder secondary to a medical disorder, rather than being related to a distinct psychiatric entity.[58] Support for this hypothesis comes largely from studies showing reduced depression following CPAP therapy.[69,70,77,83,85-89] Mood improvements have been detected early after the beginning of treatment[83,87,88] and are maintained over the long term,[77,89] even when treatment adherence is poor[69] and even in patients with mild disease.[88]

Despite these findings, other researchers have not found a significant improvement in emotional status following CPAP treatment, despite significant improvement in cognitive function,[53] objective[90] and subjective sleepiness,[90,91] and vigilance,[91] perhaps due to discomfort related to CPAP treatment or other factors related to perceived health status.

Another possible explanation is that the relationship between OSAS and depression is indirect, mediated by a correlate of OSAS, such as obesity. Together with age, obesity is the strongest risk factor for the development of OSAS.[92-97] Obese individuals suffer body image dissatisfaction, discrimination, and psychosocial distress,[98] and several studies have shown an increased prevalence of depression among obese subjects.[99-101] The degree to which the severity of apnea and obesity contribute to the relationship between depressive symptoms and OSAS has recently been explored by Aloia et al.[73] They found that depressive symptoms that are predominantly associated with the somatic dimension of depression (such apathy, loss of energy, and irritability) were more strongly associated with apnea severity, whereas depressive symptoms associated with the cognitive dimension of depression (pessimism, feeling of failure, and self-dislike) were more strongly associated with obesity. In addition, gender appears to influence these relationships, since men and women with apnea manifest depressive symptoms differently. Men only showed a relationship between apnea severity and somatic complaints, and women only showed a relationship between obesity and the cognitive factor of depression. Pillar and Lavie[68] also found gender differences in the clinical manifestations of OSAS, with women scoring higher on depression and anxiety scales than men, independently of other factors. Those studies serve to stress the likely complex nature of the relationship between depression and OSAS, and highlight the multiple potential etiologies of mood disorders in these patients.

RLS and PLMS

RLS is a condition in which patients at rest, especially in the evening and during the night, report leg paresthesias accompanied by an urge to move their legs. According to the International Restless Legs Syndrome Study Group[102] obligatory features are: (i) a desire to move the extremities associated with discomfort; (ii) motor restlessness; (iii) worsening of symptoms at rest with relief with movement; and (iv) worsening of symptoms later in the day or at night. Up to 80% of patients with RLS present PLMS,[103] and this phenomenon is considered to be a supportive criteria for the diagnostic of RLS. PLMS appears as repetitive episodes of muscle contraction, 0.5 to 5 s in duration, separated by intervals of 5 to 90 s.[104] Isolated PLMS may also occur without complaints of RLS, leading to the diagnosis of PLMD. In PLMD, as an independent sleep disorder, the patient has no evidence of a medical or mental disorder that could account for the primary complaint of insomnia or excessive daytime tiredness,

and so it is assumed that the PLMS causes sleep disruption, nonrestorative sleep, and thus, the patient's sleep-related symptoms.[105] Therefore, RLS and PLMD are distinct by definition, but may coexist. A recent study found that several polysomnographic features in RLS differ from those of PLMD,[106] suggesting that different pathophysiological mechanisms may influence sleep in both conditions.

RLS and PLMD are highly prevalent. RLS is found in 9% to 15% of adults[107,108] and its prevalence increases with age. PLMS may occur in up to 6% of the general population[109] and in 20% of patients aged 60 years or older.[110]

The unpleasant sensations experienced by patients with RLS often lead to noticeable loss of sleep, with the more severely affected patients sleeping no more than 4 to 5 h and experiencing deficits in daily functioning. Patients also report problems with functioning in sedentary situations, particularly in physically constraining places, and also in the evening when the symptoms are usually exacerbated. As a result, patients may have problems accomplishing their jobs and participating in social and recreational activities.[111] Symptoms, along with the impairment of sleep, may cause distress and lead to psychiatric illness and decreased well-being. In the 19th century, Wittmaack described the cooccurrence of RLS with symptoms of depression and anxiety, and suggested the term "anxietas tibiarum."[112] Although the first modern study attracting attention to psychiatric comorbidity, showing higher scores on depression and psychoasthenia in RLS patients, was performed 40 years ago,[113] little progress has been made since then in attempts to explore this relationship.

Despite their high prevalence in the general population, little information is available on the impact of PLMS or RLS on quality of life. In a recent American Academy of Sleep Medicine review, reference is made to the "striking omission" of quality of life research and psychological impact with respect to this disorder.[114] In two drug trials utilizing a modified version of the Hamburg Visual Analog Scales, improvements after dopaminergic treatment (first-line therapy for RLS) were noted in activities of daily living, mental function, fatigue, and depressive feelings.[115,116] A more recent large survey suggested a substantial impact of RLS on quality of life equivalent to or worse than some other major chronic medical disorders.[117] This impact was apparent on all of the SF-36 items, but the more pronounced deficits occur for measures of vitality/energy and limitations of work and activities due to physical problems, suggesting a major decrease in the level of alertness and energetic engagement with daily function. The data also indicate that patients with RLS are likely to have problems with anxiety or depressed feelings. This is in accordance with other data suggesting that patients with RLS are likely to experience mental health problems. In a study to evaluate the prevalence of RLS in a population-based survey of the elderly, it was found that individuals with RLS had higher depression scores and lower quality of mental health compared with RLS-negative participants.[118] Among men, a high depression score was significantly associated with RLS severity. However, such a cross-sectional study cannot determine whether the depression is a consequence of the syndrome or if RLS existed before the RLS appears. In another study, around 45% of a sample of 218 RLS patients had been diagnosed as having a mood disorder (depression or affective psychosis) in the 5 years prior to the diagnosis of RLS.[119] As pointed out by these authors, and illustrated by some case reports,[120] it is possible that the sleep complaints of RLS could be incorrectly interpreted as a symptom of depression. However, it is also logical to consider that discomfort caused by RLS and the chronic sleep disturbances were triggers for depression, as it has been shown that persons complaining of insomnia have a high risk of developing depression.[121,122] In a study evaluating the prevalence and impact of RLS in the general male adult population, there was a tendency towards reported isolation related to RLS.[123] Subjects with RLS were more likely to report depressed mood (odds ratio [OR] =2.6) and complained more often of reduced libido (OR=2.2). In another recent study, RLS patients had significantly higher depression and anxiety scores measured by the Zung Self-Rating Scales than control subjects and had similar electroencephalographic (EEG) changes to patients with major depression.[124] In a population-based, cross-sectional study in adults, utilizing the Hamilton Rating Scales for Anxiety and Depression, the mean anxiety and depression scores of patients were 8.03 (±6.02) and 9.27 (±5.03), respectively, which were significantly higher than those of the control group.[125] Interestingly, these values correlated with the severity score of the RLS, with higher scores correlating with more severe RLS. No data on the temporal relationship of RLS and anxiety/depression symptoms were provided, and so the causality of this relationship could not be established. A more recent

study attempted to answer this question and added new insights to the relationship between RLS and psychiatric morbidity. In their survey, Winkelmann et al[126] revived the term "anxietas tibiarum" and examined rates of depression and anxiety according to *DSM-IV* criteria in patients with RLS, compared with a group of controls from a community sample with somatic illness. RLS patients reported higher 12-month rates of any depressive disorder (OR=2.6), panic attacks (OR=2.9), panic disorder (OR=5.2), or generalized anxiety disorder (OR=3.7). RLS patients with depression attributed their sleep disturbances, depressed mood, and reduced interest as being due to their RLS symptoms. Further analysis revealed that, in most patients, the psychiatric disorder appeared after the onset of RLS, suggesting that the RLS was the primary condition, in line with the causal interpretation of the patients that their mental disorder might be caused by their RLS symptoms. This association is of strong clinical relevance because antidepressants can aggravate RLS.[127]

As regards PLMD (independently of the presence of RLS symptoms), it has been shown that patients had a high rate of past treatment for depression prior to the diagnosis of their sleep disorder (30%), although a clear association has not be found between the PLMS index and the subjective complaints of disturbed sleep, daytime sleepiness, or a sense of awakening refreshed in the morning.[128] Previously, Mosko et al[1] have also shown that patients with sleep-related periodic leg movements had high rates of self-reported depressive symptomatology. Change scores on the Profile of Mood States were obtained in this study when patients were placed on clonazepam, suggesting that the depression could be secondary to the sleep disturbance induced by the PLMS. Recently, Saletu et al[129] found higher depression and anxiety scores on the Zung Self-Rating Scale than controls, while differences in quality of life did not reach the level of statistical significance, together with differences in electrophysiological brain function reminiscent of those of patients suffering from generalized anxiety disorder. Aikens et al[130] determined patterns and relative intensity of psychopathology, as measured by the MMPI among patients with OSAS, PLMD, and insomnia. Thirty-two percent of PLMD patients had current or prior history of depressive disorder. The occurrence of any MMPI elevation was more likely among patients with PLMD compared with OSAS or psychophysiological insomnia patients. Differences emerged on the specific scales of depression, psychoasthenia, and schizophrenia. Thus, PLMD patients seem more likely to show a wide range of depressive symptoms, such as guilt, tension, and worry, as well as social alienation and diminished mental concentration, and are more prone to dysthymia accompanied by generalized anxiety and interpersonal detachment. Although the results do not address the question of whether these psychological patterns represent a cause or a consequence of sleep disorder, the authors suggest that psychopathology could be due to sleep disturbance secondary to limb movements, daytime fatigue, and/or some other consequence of repetitive limb movement. This report conflicts with that of Zorick et al,[131] who reported relatively low psychopathology rates in these patients. In fact, patients with nocturnal myoclonus had the lowest number of MMPI elevations compared with patients with sleep complaints related to a psychophysiological or psychiatric disorder.

Circadian rhythm sleep disorders

There is a subset of sleep disorders in which the etiology is primarily due to circadian dysfunction. Circadian rhythm sleep disorders may be categorized into extrinsic and intrinsic disorders. In both types, there is a mismatch between the circadian timing of sleep propensity and the demands of the environment, resulting in symptoms of disturbed sleep and impaired daytime alertness.[132]

In extrinsic disorders, such as jet lag and shift work, the unnatural temporal demands of modern society impose on a completely normal circadian and sleep-wake physiology to produce such impairments. For some individuals, such as airline flight crew, the jet lag problem may be chronic and severe. Jet lag could produce dysphoria, anergia, apathy, sleep disturbances, increased irritability, anxiety, and psychosomatic disturbances, ie, symptoms overlapping depressive disorders. The possibility of a connection between jet lag and psychiatric disorders has been postulated.[133] Clinical and pathophysiological indications suggest that jet lag is a possible trigger in the exacerbation of existing affective disorders and in the appearance of de novo mood disturbances in predisposed persons. Depressive symptoms are more frequent subsequent to flights from east to west,[134] supporting the phase-advance hypothesis for depression.[135] In the other sense, eastbound flights, which can be see as a kind of sleep deprivation, can precipitate mania.[136] Psychotic symptoms occurring during long-distance trips (referred

to as "travel paranoia") have been also reported in the literature.[137] The most plausible explanation is that long-distance flights, which involve abrupt environmental changes, can represent a severe crisis situation for predisposed individuals. However, circadian rhythm abnormalities have not received much attention in studies of psychosis, and conclusions in this field are inconsistent.

Individuals engaged in shift work experience disturbed sleep and excessive sleepiness due to the fact that their behavioral sleep-wake schedules are out of phase and often in direct opposition to their endogenous circadian rhythms. In a recent large study that aimed to determine the prevalence and consequences of shift work sleep disorder in a sample of rotating and permanent night workers, Drake et al[138] showed greater rates of depression and somatic diseases (gastrointestinal ulcers and cardiovascular diseases), elevated work absenteeism, impaired social and domestic aspects of quality of life, and more accidents, mainly related to symptoms of insomnia or daytime sleepiness. These findings are in accordance with previous studies showing copious behavioral, health, and social morbidity associated with shift work.[139-141]

In intrinsic disorders, the pathology of the circadian system itself is responsible for the symptoms. DSPS is characterized by sleep onset and wake times, which are delayed in comparison to conventional sleep-wake times. Enforced "conventional" wake times may result in chronically insufficient sleep and excessive daytime sleepiness, and can be associated with irritability and poor performance.[142] Among other aspects, the psychological profile of patients with DSPS include higher rates of depression, nervousness, introversion, and hypochondriasis.[143] In particular, the DSPS appears to be associated with present or past depression in more than 75% of patients.[144] It could be hypothesized that the failure in their social life causes social withdrawal and consequently a loss of the social cues necessary to synchronize their circadian rhythm. This might lead to an even more delayed phase shift and an enforcement of the psychological characteristics of DSPS patients, such as introversion and depressive feelings. This situation becomes a vicious circle.

The advanced sleep phase syndrome is a much less prevalent entity, characterized by habitual and involuntary sleep and wake times that are at least several hours earlier than societal means. Patients complaint of early-morning awakening, and a diagnosis of depression may be made erroneously. The maladjustment of these patients to social life occurs less frequently than in DSPS, probably because societal constraints on sleep time are less rigid than on wake time.

Conclusion

Even though most psychiatric patients have a complaint about sleep, a primary sleep disorder may also result in neuropsychiatric complications. Assessment of psychiatric status in patients with organic sleep disorders is necessary to optimize treatment strategies. An adequate assessment of psychiatric manifestations should be part of their sleep evaluation. Psychiatrists need to be alert to the possibility that patients who present with cognitive and/or affective disorders may have an organic sleep disorder such as OSAS, narcolepsy, or RLS/PLMD. In particular, an organic sleep disorder should be considered in the differential diagnosis of atypical or resistant psychiatric disorders. ❑

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Aspectos psiquiátricos de los trastornos orgánicos del sueño

Recientemente algunos estudios han intentado caracterizar las alteraciones psicológicas relacionadas con varios trastornos del sueño. El objetivo de este tipo de investigación es investigar la posibilidad que la psicopatología pueda representar un factor etiológico, una complicación y/o un blanco para el tratamiento. Además, el sueño alterado puede presentarse de una manera compleja y atípica en la que el principal componente relacionado con el sueño puede no aparecer en primer plano. Este artículo revisa la evidencia de una relación entre los trastornos orgánicos del sueño y la morbilidad psiquiátrica. Generalmente se puede concluir que los trastornos orgánicos del sueño tienen un profundo impacto negativo en la mayoría de las áreas de la calidad de vida relacionadas con la salud. Los resultados de los trastornos del sueño que se han estudiado (narcolepsia, hipersomnia idiopática, síndrome de sueño con apnea/hipopnea, síndrome de las piernas inquietas, trastorno del movimiento periódico de las extremidades y trastornos del ritmo circadiano) demuestran una fuerte evidencia para una asociación con los trastornos del ánimo. Después del tratamiento los índices de depresión pueden mejorar o no, hasta alcanzar los valores de las normas de la población, lo que sugiere que esta relación es más compleja que sólo causa y efecto.

Aspects psychiatriques des troubles organiques du sommeil

Ces dernières années, certaines études ont tenté de caractériser les perturbations psychologiques liées aux différents troubles du sommeil. Ces recherches ont pour but de savoir si la psychopathologie peut représenter un facteur étiologique, une complication et/ou une cible pour le traitement. De plus, un sommeil perturbé peut se présenter lui-même sous une forme complexe et atypique dans laquelle la composante primaire liée au sommeil peut ne pas apparaître immédiatement. Cet article passe en revue les arguments en faveur d'une relation entre les troubles organiques du sommeil et la morbidité psychiatrique. Les conclusions montrent généralement que les troubles organiques du sommeil ont un impact négatif important sur la qualité de vie en général, liée à la santé. Les résultats pour les troubles du sommeil étudiés (narcolepsie, hypersomnie idiopathique, syndrome des apnées/hypopnées du sommeil, syndrome des jambes sans repos, syndrome des mouvements périodiques des jambes au cours du sommeil, troubles circadiens du sommeil) se sont montrés fortement en faveur d'une association avec les troubles de l'humeur. Après le traitement, les scores de dépression rejoignent ou non la norme de la population, ce qui suggère que la relation est plus complexe que celle d'une simple relation de cause à effet.

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