Hypersomnia

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Hypersomnia, a complaint of excessive daytime sleep or sleepiness, affects 4% to 6% of the population, with an impact on the everyday life of the patient. Methodological tools to explore sleep and wakefulness (interview, questionnaires, sleep diary, polysomnography, Multiple Sleep Latency Test, Maintenance of Wakefulness Test) and psychomotor tests (for example, psychomotor vigilance task and Oxford Sleep Resistance or Osler Test) help distinguish between the causes of hypersomnia. In this article, the causes of hypersomnia are detailed following the conventional classification of hypersomnic syndromes: narcolepsy, idiopathic hypersomnia, recurrent hypersomnia, insufficient sleep syndrome, medication- and toxin-dependent sleepiness, hypersomnia associated with psychiatric disorders, hypersomnia associated with neurological disorders, posttraumatic hypersomnia, infection (with a special emphasis on the differences between bacterial and viral diseases compared with parasitic diseases, such as sleeping sickness) and hypersomnia, hypersomnia associated with metabolic or endocrine diseases, breathing-related sleep disorders and sleep apnea syndromes, and periodic limb movements in sleep.

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The obvious main symptom of hypersomnia is a constant complaint of excessive daytime sleep or sleepiness, which affects the everyday life of the patient. In our mechanized societies, it may impair work performance and even be involved in accidents at work or while driving. Although hypersomnia syndromes have been described for more than a century, starting with narcolepsy,[1] it is only in the last 30 years that modern sleep medicine has stressed the health and economic impacts of falling asleep at any time. Hypersomnia syndromes affect a growing proportion of the 15% to 30% of people suffering from sleep problems. Hypersomnia is present in 4% to 6% of the general population,[2] with a higher prevalence in men because of sleep apnea syndromes, the main purveyor of excessive daytime sleepiness.[3] In recent years, sleep medicine has benefited from the impact of hypersomnia and is now in full development in industrialized countries.

Although lack of nocturnal sleep is evidently the first etiology to be suspected, excessive sleepiness may result from a number of different causes, imposing an arbitrary classification. The approach we propose in this review is to describe hypersomnia syndromes under several headings. First, we shall discuss the methodological tools available to explore sleep and wakefulness and then we will examine the clinical classification of hypersomnia syn-

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dromes: narcolepsy, idiopathic hypersomnia, recurrent hypersomnia, insufficient sleep syndrome, medication- and toxin-dependent sleepiness, hypersomnia associated with psychiatric disorders, hypersomnia associated with neurological disorders, posttraumatic hypersomnia, infection and hypersomnia, hypersomnia associated with metabolic or endocrine diseases, breathing-related sleep disorders and sleep apnea syndromes, and periodic limb movements in sleep.

Methodological and diagnostic tools

The physician's first step is to thoroughly interview the patient and his or her partner to determine the patient's sleep habits and hygiene. If hypersomnia is suspected, the number and duration of diurnal sleep or sleepiness episodes should be specified. The patient should be asked whether daytime sleep bouts are refreshing and recuperative. A report on sleep quality of the preceding night should also be obtained.

A sleep diary made up of monthly forms gives an estimate of the number, duration, and chronology of daily episodes of sleep and sleepiness. On the monthly form, days are represented on the vertical axis and hours horizontally. Areas corresponding to the intersection can be filled in to represent sleep or somnolence, or even yawning. Eating times can also be indicated. The diary is completed by subjective sleep quality questionnaires, such as the Stanford Sleepiness Scale[4] and the Epworth Sleepiness Scale.[5] For example, an Epworth score >10 indicates a complaint of hypersomnia.

Objective measures of daytime somnolence involve polysomnographic techniques (recording of electroencephalogram [EEG], electro-oculogram [EOG], electromyogram [EMG], electrocardiogram [ECG], leg movements, and/or respiratory parameters) during both nocturnal sleep and the daytime. Polysomnography allows the distinction between wakefulness, rapid eye movement (REM) sleep and non-REM sleep (stages 1 to 4, stages 3 and 4 constituting slow-wave sleep [SWS]).[6]

Daytime measures of sleepiness use the Multiple Sleep Latency Test (MSLT).[7] The patient lies down in the dark for five 20-min sessions spaced 2 h apart. For example, in the case of a patient with nocturnal sleep between 11 PM and 7 AM, MSLT sessions should occur at 9 AM, 11 AM, 1 PM, 3 PM, and 5 PM. The variables of interest are sleep latency and REM sleep latency (or REM latency). Sleep latency less than 8 min indicates excessive daytime sleepiness.[8] The patient is considered normal if sleep occurs after 10 min. Between these two values, the interpretation should take into account the clinical status of the patient. REM latencies less than 15 min may also be pathological, indicating the presence of sleep-onset REM periods (SOREMP). The presence of at least two SOREMP on the MSLT, although not highly specific and sensitive, is generally considered as abnormal and indicative of narcolepsy as the cause of hypersomnia.

A variant of the MSLT is the Maintenance of Wakefulness Test (MWT).[9] The patient is not lying down, but is seated comfortably in bed, with low lighting placed behind. During two daytime 20-min sessions, the patient is asked to look ahead and stay awake as long as possible. The test ends either after 15 s of microsleep, or after the occurrence of the first sleep stage, or at the end of the session if no sleep occurs. The normal limit is 11 min.

Continuous polysomnography can also be performed for 24 h or more to assess the degree of sleepiness. This also serves to evaluate circadian variations in sleepiness.

Psychomotor tests are used by physicians to evaluate eventual performance decrements due to excessive daytime sleepiness.[9] Most tests are based on reaction time, and may be simple tests in which the patient has to respond to identical stimuli as fast and efficiently as possible, or choice reaction time tests, which use stimuli with clear and specific differences. Among simple reaction time tests, the psychomotor vigilance task, which lasts only 10 min, has proven highly sensitive to performance decrement in hypersomnia. The Oxford Sleep Resistance Test is an indirect measure of sleep occurrence. The patient sits in darkness and presses on a button when a diode lights up (1 s every 3 s). The patient is considered to have fallen asleep when he or she has missed seven light cycles.

Many other tests have been proposed, but our purpose is not to be exhaustive in this review. The application of such tools and tests helps evaluate fatigue, somnolence, and vigilance and diagnose hypersomnia syndromes and distinguish between them.

Narcolepsy

Narcolepsy is a rare, chronic neurological disorder (0.026% of the general population) caused by the inability to regulate sleep-wake cycles normally. It is characterized by excessive diurnal sleepiness and cataplexy.[10] At various times throughout the day, a narcoleptic person

experiences fleeting urges to sleep that are often overwhelming. Individuals fall asleep for a few seconds to several minutes. The essential characteristic of such sleep episodes is that they are short and refreshing, and are followed by a normal wakeful state. Cataplexy is a sudden loss of voluntary muscular tone, without any alteration of consciousness, in relation to strong emotive reactions (laughter, noise, fright, etc). In addition to these symptoms, either at sleep onset or upon awakening, the patients may suffer from vivid hallucinations and brief episodes of sleep paralysis, and report poor sleep quality. Narcolepsy generally begins in adolescence, but the age of the first occurrence varies enormously. The cause of narcolepsy remains unknown, but probably involves an interaction between genetic and environmental factors, which trigger the alteration of the hypocretin system leading to sleep disturbances. Narcolepsy is highly related to HLA subtypes.[11] The familial form of narcolepsy cataplexy is only observed in 10% of cases.

The diagnosis of narcolepsy is essentially clinical, but also involves a nocturnal polysomnographic recording followed by an MSLT, during which sleep latency should be below 8 min with at least two SOREMPs. The diagnosis is reinforced by the finding of a serological DR2-DQ1 HLA typing (more precisely DRB1*1501-DQB1*0602). Such an oligonucleotidic typing is found in 92% of Caucasian narcoleptics, compared with only 20% in the general population. More recently, narcolepsy has been related to impaired function of hypocretin-secreting neurones located in the laterodorsal hypothalamus. In the cerebrospinal fluid (CSF) of patients, hypocretin-1 concentration drops[12] and the postmortem pathological examination of the brain reveals the disappearance of hypocretinergic neurons.[13,14] An autoimmune origin is one hypothesis. However, like in the canine narcolepsy model developed at Stanford, in which a mutation of the gene coding the receptor 2 of hypocretin is present,[15] a mutation of the gene coding for preprohypocretin has been reported in one atypical and severe case of human narcolepsy.[13]

Narcolepsy without cataplexy has been described as a phenotypic variant. The clinical diagnostic criteria are similar to that of narcolepsy with cataplexy, except that the cataplexy is not present. However, the association with the HLA DQB1*0602 is weaker and the decrease in CSF hypocretin is less frequently encountered. A common pathophysiology is still a matter of debate.

There is no cure for narcolepsy. None of the currently available medications enables patients to maintain a permanent normal state of alertness. However, the most disabling symptoms, excessive daytime sleepiness and cataplexy, can be controlled in most patients. In more recent years, amphetamine derivatives have frequently been replaced by modafinil for the treatment of excessive daytime sleepiness.[16] In cases of persistent excessive daytime sleepiness, methylphenidate, amphetamine, and mazindol (a derivate of amphetamine) may be of value. The control of cataplexy is still obtained with antidepressants: tricyclics (including imipramine, desipramine, clomipramine, and protriptyline) and also selective serotonin reuptake inhibitors (including fluoxetine and sertraline) and serotonin and noradrenaline reuptake inhibitors (venlafaxine), which do not have the side effects of tricyclics. If the symptoms persist, mazindol may be used since it is active on both diurnal sleepiness and cataplexy.[17] Gamma-hydroxybutyrate or sodium oxybate in its most recent form represents an alternative treatment for hypersomnia, cataplexy, and poor sleep at night.[18] In patients at sickness onset, the intravenous administration of immunoglobulins, at a high dose of 1 g/kg/day for 2 days, repeated three times at 4-week intervals, has been shown to decrease the frequency and severity of cataplexy for several months.[19] The authors suggest that such a treatment may change the course of the sickness at a time when the autoimmune process targeting the cataplexy networks can still be reversed.

Common-sense behavior counseling may prove to be useful as a complement to pharmacotherapy. Maintenance of regular sleep schedules to enhance nighttime sleep quality and short daytime naps are indicated. Patients are advised to avoid drinking alcohol and caffeine-containing beverages before bedtime. The treatment regimen can be modified as symptoms change. Symptoms may get worse, especially over the two to three decades following their first occurrence, but they may improve in older patients.

Idiopathic hypersomnia

Idiopathic hypersomnia is a rare condition and is 10 times less frequent than narcolepsy, although no prevalence study has ever been conducted.[20] For a long time, it was confused with narcolepsy, but it was clearly individualized through polysomnographic studies.[21,22] The International Classification of Sleep Disorders (ICSD)[8] defines idiopathic hypersomnia as a normal or prolonged nocturnal sleep episode that is associated with excessive

daytime sleepiness consisting of prolonged (1- to 2-h) sleep episodes of non-REM sleep. Roth et al[21] described two clinical forms: a monosymptomatic excessive daytime sleepiness and a polysymptomatic excessive daytime sleepiness associated with abnormally long nocturnal sleep and "sleep drunkenness" upon awakening. These forms are now described as idiopathic hypersomnia without a long sleep time and idiopathic hypersomnia with a long sleep time, respectively. Contrary to narcolepsy, diurnal sleep bouts are not irrepressible and do not restore normal vigilance, and nocturnal sleep remains undisturbed, except for a delayed morning awakening.

The condition often starts before 30 years of age. Familial occurrences are frequent. It is a life-long disorder that has severe social and professional impact.[23]

The diagnosis of idiopathic hypersomnia is often overestimated and requires a thorough examination. It is appropriate in patients with excessive daytime sleepiness and/or sleep drunkenness, without any narcolepsy, periodic limb movements, or sleep apnea syndrome. Nocturnal polysomnography, to eliminate the latter conditions, is followed by MSLT sessions, which reveal short sleep latencies below 8 to 10 min, with less than two SOREMPs. However, the MSLT procedure requires that the patient be woken up early. To obtain evidence of abnormally long sleeping hours, 24-h to 48-h polysomnographic recordings may be necessary. They will reveal that the patient sleeps for more than 12 h per 24 h, with both long nocturnal sleep and long daytime naps.[20] If numerous microarousals are detected, the upper airway resistance syndrome should be considered. A new sleep recording with esophageal pressure measures will be made to evaluate transpleural pressure. Personality tests are undertaken to exclude psychiatric hypersomnia, and cerebral imaging may be performed according to the clinical context.

As no animal model of idiopathic hypersomnia is available, pathophysiology of the sickness is speculative. Treatment of idiopathic hypersomnia uses a number of stimulant drugs, such as modafinil, which is often effective on excessive daytime sleepiness. However, sleep inertia persists in most cases.

Recurrent hypersomnia

Recurrent hypersomnia is characterized by episodes of excessive sleep lasting from a few days to several weeks. Patients may sleep for at least 18 h a day, and rise only to eat and void. The episodes are typically separated by weeks or months, during which normal sleep patterns are resumed. Excessive sleep may accompany behavior abnormalities, such as overeating, sexual disinhibition, and other mental disturbances. This polysymptomatic form is represented by the Kleine-Levin syndrome which occurs in adolescent boys bearing the HLA DQB1*0201* type.[24] The syndrome may also be idiopathic in relation to menstruation.[25] It may even be secondary to neurological or psychiatric conditions, or a viral infection that occurred weeks before. The diagnosis of Kleine-Levin syndrome is mainly clinical. Differential diagnosis must distinguish recurrent hypersomnia from obstructive sleep apnea syndrome, narcolepsy, or periodic limb movement disorder. In order to confirm hypersomnia and exclude epilepsy and organic pathology, EEG and polysomnographic recordings, and cerebral imaging may prove useful. The etiology of idiopathic recurrent hypersomnia remains elusive, though most symptoms can be regarded as a hypothalamic dysfunction.

Recurrent hypersomnia results in major disturbances in social and family life, and its prognosis is unknown. The evolution throughout life is favorable in most cases, with a progressive disappearance of symptoms. Treatment of recurrent hypersomnia episodes includes stimulants, despite their frequent lack of efficacy. Prophylactic measures may be successful, such as prevention with valproate, carbamazepine, or lithium carbonate,[26] or estroprogestative ovulatory inhibitors in case of menstruation-related disturbances.[25]

Insufficient sleep syndrome

The insufficient sleep syndrome is a disorder that occurs in an individual who fails to obtain sufficient nocturnal sleep to support normally alert wakefulness.[8] The individual is in fact chronically sleep-deprived at his own will, without being aware of it. Such a situation is increasing in our modern technologically inclined societies. The individual is pressured by socioprofessional imperatives and feels that he or she does not have the time to do everything. The worker may also be doing shift work. As the nights pass by, sleep debt increases.

The insufficient sleep syndrome causes 5% to 10% of consultations for excessive daytime sleepiness.[27] The subject is generally an active or overactive 40-year-old man, with responsibilities and a high social status. The syndrome can also be related to shift work or frequent transmeridian airplane trips.

As the sleep debt develops, the individual starts suffering from excessive somnolence in the afternoon, in the evening, or after meals. Patients report that they sleep 5 to 6 h nightly on weekdays, and 9 h during the weekends. They have difficulty rising in the morning and sometimes experience sleep drunkenness–like episodes. Work and cognitive performance, as well as decision-making, may be impaired. The patient may also complain of increasing levels of subjective fatigue, mood deterioration, muscular pain, gastrointestinal upset, and visual disturbances. Symptoms disappear on weekends and during the holidays.
The diagnosis is mainly performed during interview. However, in cases of a suspected associated pathology, such as respiratory disturbances during sleep, a polysomnographic recording may be indicated. In the case of insufficient sleep syndrome, this recording will show a good sleep efficiency (>90%) and short sleep latency, indicative of a sleep rebound.

Medication- and toxin-dependent sleepiness

Numerous medications are potentially responsible for excessive daytime sleepiness, such as hypnotic, anxiolytic, antidepressant, neuroleptic, antihistaminic, antiepileptic (except for lamotrigine), and antiparkinsonian drugs. Alcohol presents sedative or stimulant effects, which depend on dosage and individual susceptibility.[28] Acute alcohol consumption may delay or, in contrast, advance sleep induction. REM sleep is postponed to the second half of the night and SWS increases in the first half. Discontinuation of alcohol consumption is often reported to be followed by an REM sleep rebound. Chronic alcoholics sleep poorly, with a reduced amount of REM sleep, sleep being composed almost exclusively of non-REM sleep. Acute withdrawal is accompanied by more frequent REM sleep episodes determining a shortening of REM–non-REM sleep cycles with decreased SWS. Hallucinations occurring in this condition have been hypothesized to be related to REM sleep fragmentation and/or rebound. In chronic alcoholics who become sober, shortened REM latency, elevated REM percentages, and high REM density,[29] as well as lowered SWS percentages,[30] have been reported as predictors of relapse.
Interview should eliminate such drug–sleep interactions. In the case of drug-induced hypersomnia, careful and progressive withdrawal and/or substitution must be undertaken. Treatment withdrawal from stimulants could in fact be expected to lead to a rebound of sleep, as is the well-known case in sleep deprivation.[31] Such a rebound is not observed. For example, amphetamine withdrawal after its administration during a 64-h sleep deprivation leads to an alleviated recovery sleep for at least two nights.[32] Tobacco consumption withdrawal in a heavy cigarette smoker was reported to provoke excessive daytime sleepiness with an impairment of work performance, which was successfully treated with modafinil.[33]

Hypersomnia associated with psychiatric disorders

In contrast to insomnia, excessive daytime sleepiness is rarely associated with psychiatric disorders such as major depression or major mood disorders.[34] However, no specific sleep disturbance can be evidenced and no substance can be blamed for it. In addition, MSLT is mostly normal; therefore, the diagnosis of hypersomnia in these conditions is still subject to controversy with a more probable diagnosis of fatigue. In the northern countries of the northern hemisphere, seasonal affective disorders associate hypersomnia with anxiety, irritability, sadness, sugar bulimia, and increase in body weight.[35]

Hypersomnia associated with neurological disorders

A number of neurological affections may be accompanied by excessive daytime sleepiness. Brain tumors[36] or stroke[37] that provoke lesions or a dysfunction of the thalamus, hypothalamus, and brain stem can cause hypersomnia. For example, cases of symptomatic narcolepsy have been described as being associated with such lesions. Neurodegenerative conditions, Alzheimer's disease, Parkinson's disease, or multisystem atrophies will often provoke hypersomnia.[36] In such associations, the main causes of hypersomnia, such as sleep apnea syndromes, medications, and periodic leg movements, should be explored.
Neuromuscular diseases may provoke breathing disorders and predispose to abnormal sleepiness. Myotonic dystrophy is of particular interest, and is often associated with hypersomnia with SOREMP.[38]

Posttraumatic hypersomnia

Abnormal sleepiness may be observed within 6 to 18 months of head trauma. Clinically related to idiopathic hypersomnia, it may be associated with headaches, mem-

ory loss, and lack of concentration.[39] Its course depends on the location and the extent of the initial lesions.

Infection and hypersomnia

Although the initial description of von Economo's lethargic encephalitis in patients who suffered from pharyngitis dates back to 1917,[40] the influence of bacterial agents on sleep was revealed 20 to 30 years ago, when the pyrogenic and hypnogenic actions of muramyl peptides and endotoxin (bacterial lipopolysaccharides) were described.[41,42] The hypnogenic effect was recently extended to the viral envelope glycoproteins. This action may be mediated by host immune reactions. Several cytokines are pyrogenic and somnogenic, such as tumor necrosis factor-α, interferon-β, and interleukin-1. However, sleep has rarely been analyzed in infectious patients, due to the general emergency aspects of diseases such as meningitis or severe viral infection. Research on polysomnographic examinations in infectious diseases is therefore scarce, though animal models of bacterial and viral infection have been extensively used. In parasitic diseases, human sleep was studied by our team in the Gambian form of human African trypanosomiasis, sleeping sickness, which is due to the injection of trypanosomes by tsetse flies.

In both viral and bacterial infections, the initial host's immune reaction and hypersomnia, especially centered on SWS, develop concomitantly. In some diseases, such as Whipple's disease[43] or human immunodeficiency virus (HIV) infection, autoantibodies are produced in the second phase and a dramatic decrease in, or disappearance of, sleep may occur.[44] On the contrary, despite an extensive immune reaction during the initial hemolymphatic stage of human African trypanosomiasis, sleep remains undisturbed.[45] However, in the second meningoencephalitic stage, autoantibodies against nervous structures are widely produced, and both sleep and wakefulness are impaired due to the penetration of the trypanosome into the central nervous system. At this stage of sleeping sickness, a "complex polysomnographic syndrome" is observed with disappearance of circadian rhythmicity of the alternation of sleep and wake episodes, which occur at any moment of the day or night, and appearance of anomalies in sleep structure, and the occurrence of SOREMP.[45,46] In any case, sleeping sickness is not a hypersomnia per se, but rather a major disorder of sleep structure and its circadian regulation.

Therefore, in bacterial and viral diseases sleep modifications are coupled with immune reactions. The uncoupling between the acute phase of the immune reaction in sleeping sickness and sleep-wake patterns is not yet understood.

Hypersomnia associated with metabolic or endocrine diseases

Rarely, hypersomnia may complicate diabetes, hepatic encephalopathy, hypothyroidism, and acromegaly.[47] Breathing disorders and periodic leg movements that often accompany metabolic disorders should be explored in the genesis of such hypersomnia.

Breathing-related sleep disorder and periodic limb movements in sleep and sleep apnea syndromes

Sleep apnea syndromes[3] and periodic limb movements in sleep[48] are the most frequent causes of excessive daytime sleepiness.

Limb movement disorders

Limb movement disorders related to rest and sleep have been divided into two syndromes, restless legs syndrome and periodic limb movements during sleep. The latter are commonly associated with the former, as well as with various sleep disorders, such as insomnia, narcolepsy, and sleep apnea syndrome, and also physiological aging. The limb movements arouse the patients who is unaware of his or her highly fragmented sleep.[48] Polysomnography shows lengthened sleep latency, increased waking after sleep onset, and numerous sleep stage changes; stage 1 is elevated, while SWS is low. A chronic sleep-wake disorder may develop, causing excessive daytime sleepiness.

Restless legs syndrome is a sensorimotor disorder characterized by an urge or a need to move the limbs. It is usually associated with uncomfortable or unpleasant sensations in the legs, which worsen in the evening or at night.[49] The symptoms begin at rest when the person is lying or sitting and are relieved by movement such as walking or stretching. The condition is present in 5% to 10% of the general population. It has been said to be the "most common and least diagnosed" sleep disorder.[50] Diagnosis relies essentially on the interview with the physician.[51] Restless legs may be caused by polyneu-

ropathy, arthritis, positional discomfort and ischemia, neuroleptic exposure, or cramps. Objective measures include polysomnography, with EMG electrodes placed at the level of the anterior tibialis muscles on both legs, and the suggested immobilization test (SIT) to provoke the restless legs symptoms. In the SIT, the patient is required not to move for 1 h while measures of leg movement and movement intensity are monitored. In 80% of patients, polysomnography will show periodic limb movements while awake and sensory discomfort may rise during the SIT.

Periodic limb movements in sleep are repetitive movements that primarily involve the legs, especially during non-REM sleep. When patients report sleep-wake complaints, they are said to suffer from periodic limb movement disorder. Movements are counted if they last 0.5 to 5 s and occur in a series of four or more at intervals of 5 to 90 s. The EMG amplitude of the nocturnal limb movements must be 25% or more of the baseline EMG amplitude while awake. The severity of the condition is determined by the periodic limb movement index (number of periodic movements per hour of sleep). The periodic limb movement arousal index is the number of periodic limb movements associated with EEG arousals per hour of sleep. Periodic limb movement disorder is defined as mild (5 to 25 periodic limb movements occur per hour of sleep), moderate (25 to 50 movements per hour of sleep), or severe (more than 50 periodic limb movements per hour of sleep or more than 25 associated with arousals per hour of sleep).

The pathophysiology of periodic movements has been related to iron deficiency, which itself is related to dopaminergic dysfunction, which has led to the recent publication of dopaminergic-centered therapeutic standards,[52] dopamine agonists giving positive results. With the exception of anticholinergics, Parkinsonian medications benefit the condition.

Sleep apnea-hypopnea syndromes and the upper airway resistance syndrome

Two clinical entities constitute the essential part of breathing disorders during sleep: sleep apnea-hypopnea syndromes and the upper airway resistance syndrome.[53] Obstructive sleep apnea affects up to 4% of middle-aged male adults, but may also affect women. The generally obese heavily snoring male patient stops or reduces (by more than 50%) breathing between 10 s to 1 min. Efforts to breathe arouse the patient, leading to an extremely fragmented sleep and excessive daytime sleepiness. The latter varies to a large extent, from simply falling asleep in nonstimulating situations to a major symptom with mental performance decrements, which can be responsible for car or work accidents. Headaches and fatigue in the morning, nocturnal polyuria and sexual problems (from reduced libido to impotence) are commonly encountered. Hypertension, angina pectoris, cardiorespiratory failure, and stroke may develop as the disorder progresses.

The diagnosis is confirmed by polysomnography with the recording of respiratory parameters. Oronasal sensors made up of thermocouples or thermistors detect the passage of airflow by measuring temperature variations at the nose and mouth openings. However, they may be uncomfortable, and so unobtrusive nasal cannulae with separated left- and right-sided tubings connected to two pressure transducers have recently been developed. The efficacy of the measurement of nasal and oral airflow is limited in case of hypopnea and upper airway resistance. Thoracic and abdominal straps, made of mercury strain gauges or graphite rubber, indicate the presence or absence of central respiratory drive. Pathological indices are set at five apneas per hour in the adult (10 per hour in the aged). These measures allow the distinction between obstructive (respiratory effort) and central (no respiratory effort) sleep apnea syndromes. Respiratory effort is measured by recording pleural pressure through esophageal pressure. Inspiratory effort is also quantified by pulse transit time, the time taken for the arterial pulse pressure wave to travel from the aortic valve to a peripheral site. Practically, it is measured from the R wave on the ECG to the appearance of the pulse wave at the finger. Pulse transit time is inversely proportional to blood pressure, and so the drop in blood pressure with inspiration determines rises in pulse transit time. Pneumotachography is the standard method to evaluate the airflow volume. Oxymetry by infrared wavelength absorption is necessary to calculate the ratio between oxyhemoglobin and reduced hemoglobin. The sensors are placed on the finger, or on the ear or nose.

The clinical symptoms of the upper airway resistance syndrome overlap widely those of the sleep apnea syndrome.[53] Patients affected are nonobese men or women, with a complaint of excessive daytime sleepiness, snoring (especially in men), with frequent fatigue upon awakening. Clinical examination often reports a triangular face, a small chin, an arched palate, a class II mal-

occlusion, and a retroposition of the mandible. The diagnosis is ascertained during polysomnography associated with esophageal pressure monitoring, by the presence of repetitive increase in esophageal pressure that leads to transient arousals without any changes in respiratory disturbance index (index of apnea/hypopnea <5 per hour) and in oxygen saturation.

Differential diagnosis with idiopathic hypersomnia requires the recording of esophageal pressure. Here also, the recording of esophageal pressure will show the gradual increase in respiratory effort preceding the microarousal.

Sleep is entirely disorganized, especially in apnea syndromes. Microarousals due to respiratory efforts may add up to hundreds of episodes during the night. Non-REM sleep is highly fragmented and some patients do not reach SWS. REM sleep is also fragmented. The patient is sleep-deprived and daytime sleepiness may become extreme. The apneic index, ie, the number of apneas per hour of sleep, has long been used to quantify the breathing disorder.

Treatment of apneic patients has been transformed by the use of continuous positive airway pressure (CPAP) of 5 to 15 cm H_2O.[54] The only disadvantage of such therapy is the constraint to the patient and his or her partner. Apneas are rapidly eradicated and the symptoms of the illness disappear, especially daytime sleepiness. CPAP is also efficient in the upper airway resistance syndrome, though no other means have been tested extensively to date. Mandibular advancement devices may be of value in nonsevere cases of obstructive sleep apnea syndrome. Postural alarm can be used in position-dependent apneics. No specific medications, drugs (eg, ventilation stimulants), or oxygen therapy have been shown to be effective on sleep apneas. In all cases, the patient should be asked to lose weight.

Surgical treatments[55] are based on the identification of the site of airway obstruction (nose, soft palate, and/or tongue). The protocols include various forms of reconstruction depending on the needs and the anatomy of the patient. They range from tracheotomy, glossectomy (surgical removal or laser evaporation of a portion of the tongue), radiofrequency shrinkage of the obstructive tissues of the upper airway (nose, palate, or tongue), or suspension techniques with sutures of the tongue base, nasal reconstruction, uvulopalatopharyngoplasty, mandibular osteotomy with genioglossus advancement, hyoid myotomy and suspension, and maxillomandibular advancement.

Conclusion

Excessive daytime sleepiness is a frequent complaint in industrialized societies, and has a significant impact on work and family life. Physicians have several methods to affirm the existence of daytime somnolence and diagnose between the numerous causes of hypersomnia. They also have access to large array of medications and therapeutic strategies, which are constantly increasing in quantity and quality. ❑

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Hipersomnia

La hipersomnia es la cantidad excesiva de sueño durante el día o somnolencia excesivo y afecta al 4% a 6% de la población con un impacto en la vida diaria del paciente. Hay herramientas metodológicas para explorar el sueño y la vigilia (entrevista, cuestionarios, diario de sueño, polisomnografía, prueba de latencia múltiple del sueño, prueba de mantención de la vigilia) y pruebas psicomotoras (por ejemplo, la prueba de vigilancia psicomotora, la prueba de resistencia al sueño de Oxford y la prueba de Osler) que ayudan a distinguir las causas de la hipersomnia. En este artículo las causas de hipersomnia se detallan siguiendo la clasificación convencional de síndromes hipersómnicos: narcolepsia, hipersomnia idiopática, hipersomnia recurrente, síndrome de sueño insuficiente, somnolencia por medicación o toxinas, hipersomnia asociada con trastornos psiquiátricos, hipersomnia asociada con trastornos neurológicos, hipersomnia postraumática, infección e hipersomnia (con especial énfasis en las diferencias entre enfermedades bacterianas y virales comparadas con enfermedades parasitarias, como la enfermedad del sueño), hipersomnia asociada con enfermedades metabólicas o endocrinas, trastornos del sueño relacionados con la respiración, síndromes de apnea del sueño, y síndrome de movimientos periódicos de extremidades durante el sueño.

Les hypersomnies

L'hypersomnie est une plainte de somnolence diurne excessive qui affecte entre 4 et 6 % de la population générale et influe sur la vie quotidienne du patient. Le diagnostic étiologique de l'hypersomnie bénéficie d'une panoplie de questionnaires et de tests qui explorent la qualité de l'éveil et du sommeil du patient (entretien dirigé, questionnaires, agenda de sommeil, polysomnographie, test de latence multiple du sommeil, test de maintien de l'éveil) et de tests psychomoteurs capables d'établir le retentissement de la somnolence excessive sur les performances mentales et l'équilibre psychique du patient (par exemple, test de vigilance psychomotrice, test de résistance au sommeil d'Oxford, test d'Osler, etc.). Les différentes catégories d'hypersomnie sont détaillées selon la classification conventionnelle actuelle : narcolepsie, hypersomnie idiopathique, hypersomnie récurrente, syndrome d'insuffisance de sommeil, somnolence liée aux médicaments et aux toxiques, hypersomnie associée à des troubles psychiatriques, hypersomnie associée à des troubles neurologiques, hypersomnie posttraumatique, hypersomnie liée à un syndrome infectieux (en insistant sur les différences entre les infections virales et bactériennes, par rapport aux infections parasitaires telles que la maladie du sommeil), hypersomnie associée à des maladies métaboliques ou endocriniennes, hypersomnie associée aux mouvements périodiques des membres et au syndrome des jambes sans repos, et hypersomnie liée aux syndromes d'apnées et d'hypopnées au cours du sommeil.

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