

National Consensus on the Diagnosis and Treatment of Sleep Disorders and Wakefulness

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National Consensus on Diagnosis and Treatment of Disorders of Sleep and Wakefulness

Today, November 9, 2019, we, the undersigned experts, have reached a consensus on the diagnosis and treatment of disorders of sleep and wakefulness:

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The consensus is in compliance with and supplements the standards of neurology, pulmonary diseases and otorhinolaryngology.

National Consensus on the Diagnosis and Treatment of Sleep Disorders and Wakefulness

Introduction. Sleep is a changed state of consciousness that occurs naturally and at certain intervals of time, which together with wakefulness forms the structure of the circadian rhythm. During sleep, the response to external stimuli is significantly reduced, volitional movements are suppressed. Changes in brain activity, body temperature and levels of some hormones are reported.

Sleep architecture. During sleep, several electrophysiologically distinct stages occur, repeating in cycles. The duration of sleep is 6-8 hours, with 4-6 cycles of 90-110 minutes duration being counted during this period. In each cycle, the two main types of sleep are represented in different ratio:

1. *Non-REM sleep* – it is divided into four stages: stage I is the most superficial, one can be easily awakened. It occupies 2-5% of the total sleep duration. Stage II is the longest (45-55%), sleep is deeper, characteristic K-complexes and sleep spindles appear in the EEG. Stages III and IV are the deepest, occupying 5-15% of sleep and covering mostly the first part of the night. The reactivity of the individual to exogenous stimuli is significantly reduced. In stage I eye movements are slow, while in the other stages (II, III and IV) they are missing partially or completely. Muscle tone decreases gradually, with stages III and IV being the lowest. EEG detects slow wave activity by theta waves (4-7 Hz) and in deep stages by high-amplitude delta waves (0.5–3 Hz).

2. *REM sleep* – it is associated with rapid eye movements (Rapid Eye Movements), occupies 20-25% of sleep and is quantitatively more present in the second part of sleep. Full muscle atony is reported, in EEG - low-amplitude mixed activity similar to that during wakefulness. Dreams are formed mainly during the REM phase of sleep.

The 'gold standard' in sleep research is polysomnography. It involves the registration of brain activity through EEG, the monitoring of eye movements (electro-oculography), muscle tone, respiratory effort, body position, heart rate, and oxygen saturation. Additionally, a snoring microphone as well as sensors for recording limb movements can be included. The inclusion of synchronized video surveillance in polysomnographic recording improves the diagnostic value of the study. Other studies are limited polygraphy and the multiple sleep latency test.

Classification of sleep disorders. The International Classification of Sleep Disorders, (ICSD) is the most widely used in the world, with its third edition covering seven major categories of diseases and more practical than previous editions (ICSD-3)⁽¹⁾:

- Insomnias
- Sleep-Related Breathing Disorders
- Central Disorders of Hypersomnolence
- Circadian Rhythm Sleep Disorders
- Parasomnias
- Sleep-Related Movement Disorders
- Other Sleep Disorders

ICSD-3 includes 60 specific diagnoses within the seven major categories, as well as a supplement for the classification of sleep disorders related to internal and neurological disorders. Pediatric diagnoses do not differ from those in adults, with the exception of breathing disorders during sleep⁽²⁾.

Insomnias. They are characterized by: difficulty falling asleep, including difficulty in finding a comfortable sleeping position, waking at night, not being able to fall back to sleep and maintain sleep, and the perception of poor quality, or nonrestorative, sleep at awakening. Secondly, daily drowsiness, anxiety and irritability are noted. Insomnia occurs in some period of life in about 60% of people, and as one is getting older, its frequency increases. In about 25% of people, complaints of insomnia are chronic. The time frame of symptoms is the most commonly used as the main criterion in the classification of insomnia.

It is diagnosed by means of a detailed case history, questionnaires and scales for assessing the quality of sleep and actigraphy (a device with the shape of a wristwatch that records the patient's activity within one or more days), sleep logs and polysomnography. Many sleep disorders can mimic insomnia, so knowing them is of the utmost importance (Table 1). Some internal and neurological disorders also cause insomnia and other sleep disorders (Table 2). The effects of the medications prescribed for other diseases should also be taken into account (Table 3). A polysomnographic study is not required in the existence of convincing insomnia data.

ICSD-3 identifies three main types of insomnia: short-term insomnia, chronic insomnia, and other insomnia (when the patient has symptoms of insomnia but does not meet the criteria for the first two types). The previous classifications divided the chronic insomnia into primary and comorbid, which is dropped out in the latest classification because it does not improve the accuracy of diagnosis^(1,2).

Chronic insomnia - ICSD-3 summarizes all diagnoses of chronic insomnia in one disease, chronic insomnia⁽¹⁾. In order to be diagnosed with chronic insomnia, it is necessary to record problems with falling asleep (initiation of sleep) and maintaining sleep, under optimal conditions

for sleep and daily consequences as a result of the symptoms of insomnia. Diagnostic criteria for chronic insomnia include sleep disturbance for a minimum of three days per week for a period of three months. Insomnia is part of many psychiatric and internal disorders, and therefore should be considered an independent disease unit only if it is very pronounced and requires additional testing, examination and treatment.

Short-term insomnia - The diagnosis of short-term insomnia is according to the criteria for the chronic one but with symptoms manifestation for less than three months. It often develops temporarily in connection with a significant stress factor. In order to be diagnosed, insomnia must be of extremely independent importance for the patient and require separate clinical attention. This type of insomnia often improves after the disappearance of stress or when the patient develops mechanisms for coping with stress⁽¹⁾. However, in some cases the disease can become chronic.

Treatment should be personalized and oriented towards the type of insomnia. Pharmacotherapy is indicated with short-term insomnias and cognitive-behavioural therapy should be predominantly used with the chronic type. The cognitive-behavioural therapy should not be underestimated and should be attempted even at the onset of the disease. When diseases that mimic insomnia are suspected, these should be carefully diagnosed. The goals of the therapy are to shorten the latency of falling asleep, to increase overall sleep time, to reduce awaking and to improve the efficiency and quality of sleep. This should also alleviate the daily symptoms of insomnia, and the effects should be constant over time. Diagnosis and treatment of insomnia is most often done on an outpatient basis. In some cases, however, insomnia improves in hospital settings and this is associated with changes in sleep conditions and habits.

Pharmacological therapy. Pharmacological therapy is recommended for short-term treatment. There is a wide range of drugs - benzodiazepines, benzodiazepine receptor agonists, sedative antidepressants, antihistamines, melatonin and its agonists, orexin receptor antagonists (Table 3). Benzodiazepines and benzodiazepine receptor agonists are divided into short-, intermediate- and long-acting agonists, one of which may be selected according to the individual needs of the patient. Generally, in cases of problems with falling asleep, a short-acting medication is appropriate, in cases of problems with sleep maintenance - intermediate-acting, and in the case of persons with early awakening - long-acting medications. However, the last two groups can have residual effects the next day. This group of medicines have few effects on the cardiovascular system and can be easily combined, but their negative effects are on memory, ataxia, tendency to fall, addiction and rebound phenomena.

Another group of drugs is melatonin and melatonin receptor agonists. The soporific effect of melatonin is lacking convincing evidence in literature. Melatonin receptor agonists Remelteon (8-24

mg dose) and Agomelatine (25-50 mg) have moderate effects on falling asleep with minimal side effects and carry-over effects the next day due to their short half-lives.

Sedative antidepressants - tricyclic, Trazodone and Mirtazapine, can be used especially in the presence of comorbid psychiatric symptoms and in the presence of side effects of benzodiazepines. Data on their efficacy in the treatment of insomnia are still scarce.

There are limited data on the use of antihistamines as hypnotics in the literature. In contrast, they are often sold and used for self-medication as OTC medications.

Antipsychotic drugs are also commonly used to treat insomnia - for example, Chlorpromazine, Clozapine, Olanzapine and Quetiapine. For this treatment, they are used off-label and their side effects such as metabolic syndrome and extrapyramidal disorders should be considered.

Pharmacological therapy for insomnia should be short-term, but its prolonged use is often required. There are reports of up to 6 months of use of benzodiazepine receptor agonists in the literature, with no side effects and no necessity of dose increase. This is especially appropriate when there is a lack of specialists with a focus on conducting cognitive-behavioral therapy. In general, the data on insomnia in literature are far from the strength of large randomized studies, and recommendations are often consensual⁽⁶⁾.

Pharmacological therapy is administered for as short term as possible and intermittently if possible. Upon return of the complaint, another course of pharmacotherapy may be attempted. Pharmacotherapy should be used with caution in elderly patients due to memory effects and increased risk of falls.

Non-pharmacological therapy. Cognitive Behavioural Therapy (CBT) is a leading treatment for chronic insomnia. It is multicomponent and includes training the patient on the physiology and hygiene of sleep, explaining wrong habits and beliefs, explaining the control of external stimuli during sleep, sleep restriction methods and relaxation techniques. The purpose of this type of therapy is to reduce the state of over-awakening in patients with insomnia, with specific types of therapies being appropriate on a case-by-case basis. It is laborious and more time-consuming to achieve an effect, but there is evidence from the literature that this type of CBT is comparable to the effects of drug therapy. It is usually necessary to combine more than one method, and explaining only the physiology of sleep and sleep hygiene are often insufficient. CBT is often not applicable in non-cooperative patients and those with memory and cognitive deficits.

Hypersomnia. The International Classification of Sleep Disorders pays particular attention to the concept of daytime sleepiness, defining it as 'the inability to stay awake and alert during the major episodes of wakefulness during the day leading to an overwhelming need for sleep or

involuntary naps or falling asleep.' Drowsiness is different in degree and is most pronounced in monotonous actions requiring less attention. Some patients feel drowsy, while others fall asleep suddenly (sleep attacks). Globally, daily somnolence occurs in 10-25% of the total population. In the modern world, the most common cause of daytime sleepiness is the combination of insufficient night-time sleep, poor sleep hygiene, and various work shifts. In addition, many sleep disorders can lead to daytime sleepiness - obstructive sleep apnea, circadian disturbances and periodic limb movements during sleep. Another reason for hypersomnia is in the group of central disorders with hypersomnolence - narcolepsy, idiopathic hypersomnia and others. All these causes lead to a significant risk to the patient and the public of road accidents and workplace incidents.

Sleep related breathing disorders are characterized by abnormalities in breathing during sleep in adults and children⁽¹⁾. There are four main groups of sleep related breathing disorders:

- Central sleep apnea syndromes
- Obstructive sleep apnea syndromes (OSA)
- Sleep related hypoventilation syndromes
- Sleep related hypoxemia

These syndromes are subdivided into subgroups according to their etiology.

Central sleep apnea syndromes are subdivided into the following nosological units:

- Central sleep apnea with Cheyne-Stokes breathing
- Central apnea due to a medical disorder without Cheyne-Stokes breathing
- Central sleep apnea due to high altitude periodic breathing
- Central sleep apnea due to a medication or substance
- Primary central sleep apnea
- Primary central sleep apnea of infancy
- Primary central sleep apnea of prematurity
- Treatment-emergent central sleep apnea

The latter is new to the ICSD-3 classification and has previously been classified as complex sleep apnea⁽¹⁾. The criteria for establishing this diagnosis are the presence of primary obstructive sleep apnea in the polysomnographic testing, which is mastered during positive pressure treatment, but new, persistent central apnea is recorded. These central events cannot be associated with other comorbidities such as Cheyne-Stokes breathing or substance use (e.g. opioids). Many patients show central apnea when starting positive pressure treatment most often during the wake-to-sleep transition, but with adaptation to therapy they most often disappear (usually within a month), so tracking data from CPAP devices is appropriate.

Obstructive sleep apnea (OSA) syndromes include OSA in children and adults. OSA is common (8% of men and 4% of women) in the population with a peak around the fifth decade of life, with an increase in recent population surveys. It is characterized by recurrent (5-100 times/hour) episodes (lasting more than 10 s and desaturations of at least 3-4%) of complete upper airway obstruction (apnea) or incomplete obstruction with ineffective respiratory effort (hypopnea). Central apnea, unlike obstructive, lacks respiratory effort during apnea/hypopnea. There are also mixed apneas, which most often begin as central but continue in obstructive ones. The presence of apneas and/or hypopneas leads to disruption of the structure of sleep by quantitatively increasing the superficial stages of sleep at the expense of deep stages. As a result, sleep becomes fragmented and excessive daytime sleepiness occurs with frequent short-term (seconds or minutes) sleeps, especially in a monotonous environment - watching TV, driving long distances, etc. The disease is also manifested by frequent (2-7 times) waking up to pee at night (increased diuresis due to increased secretion of the natriuretic peptide during apnea pauses), severe snoring - often preceding the occurrence of sleep apnea with years, observed from the side the bed partner pauses in breathing during sleep and waking up with a feeling of dry throat and deposits - due to pronounced snoring and breathing mainly through the mouth and feeling of unrest. Night apnea and hypopneas occurring every night lead to chronic hypoxia with severe secondary effects: increased risk of vascular disease (heart attack, stroke), therapeutically resistant arterial hypertension, arrhythmia, obesity, hypercholesterolemia, diabetes mellitus, etc. The risk of road accidents for no apparent reason is repeatedly increased (3-4 times) due to falling asleep behind the wheel on a straight road. A major risk factor for OSA is obesity and some anatomical features of the pharyngeal airways. Untreated OSA leads to the development of pulmonary hypertension, arrhythmias, diabetes mellitus, memory and cognitive dysfunction, and sexual problems. OSA is often associated with depressive symptoms, and overweight psychiatric illness can in turn lead to sleep apnea. Diagnosis is important because untreated OSA leads to increased overall and cardiovascular mortality.

The **diagnosis** of OSA in adults is established in the presence of 15 or more obstructive events per hour, even in the absence of subjective symptoms or comorbid diseases⁽¹⁻³⁾. In adults with accompanying internal or psychiatric illnesses (eg., hypertension, coronary heart disease, atrial fibrillation, congestive heart failure, stroke, diabetes, cognitive problems or mood disorders) or signs of excessive daytime sleepiness, fatigue and/or insomnia, a diagnosis of OSA can be given in the presence of five or more mainly obstructive respiratory events per hour. OSA is considered severe in the presence of more than 30 obstructive events per hour.

The diagnosis is established on the basis of medical history and physical examination (overweight, large neck circumference, male, middle age, large tonsils and tongue, other nasopharynx abnormalities, short lower jaw, high blood pressure, etc.). Other important anamnestic

data for patients with suspected OSA are - presence of depression, use of sedatives, stimulants and alcohol, presence of hypothyroidism and other sleep disorders.

Some questionnaires for assessing daytime sleepiness (ESS, Figure 1) and the risk of sleep apnea (STOP-Bang test, Figure 2) may also be helpful for diagnosis.

The gold standard for OSA research is laboratory polysomnography with CCTV in laboratory conditions, but more recently, limited polygraph testing (LPT) has even more often been admitted even in outpatient settings in selected patients (also known as ambulatory sleep testing). LPT measures respiratory events over the entire recording period, not sleep time, which may result in a lower apnea/hypopnea index. It is indicated for more severe cases of OSA⁽⁵⁾.

Pediatric OSA criteria require the presence of at least one clinical factor (snoring, difficulty in breathing, apnea, daytime drowsiness, hyperactivity, or others). With polysomnography in children it is sufficient to have one or more obstructive events per hour of sleep or the presence of obstructive hypoventilation ($\text{PaCO}_2 > 50$ mmHg for $> 25\%$ of sleep time). In addition to obstructive events and/or obstructive hypoventilation, there should also be associated snoring, fluctuations in nasal pressure waves, or paradoxical thoraco-abdominal movements.

Treatment for sleep apnea includes the following therapeutic options: patient training, behavioural interventions, non-surgical and surgical treatment, treatment of comorbidities.

Patients and their relatives should be informed about the pathophysiology, risk factors, consequences and possible treatment of OSA. This information should also cover behavioural interventions - lifestyle changes that would lead to improved symptoms. These include: losing weight, improving exercise regime, reducing or stopping smoking and drinking, not using sedatives and sleeping pills, not sleeping on the back.

Non-surgical interventions include treatment with positive airway pressure and administration of oral devices.

The gold standard and the highest level of evidence from clinical trials of OSA with moderate and severe disease is treatment with continuous positive airway pressure through the nasal or facial mask (CPAP). The optimum individual pressure is titrated manually or automatically and results in a complete reduction of apnea/hypopneas and associated wakes. There are also modifications to positive-pressure treatment such as variable-pressure automatic CPAP according to the characteristics of apnea and double pressure administration during both inspiratory and expiratory (BiPAP). In some non-responding cases, as well as in patients with predominant central apnea, it is appropriate to use more sophisticated methods such as adapted servo ventilation⁽⁷⁾. Positive-pressure treatment should be monitored as up to 50% of patients discontinue treatment or use the apparatus for less than 4 hours per night. Some side effects - dry mouth and nose, redness of eyes etc., can be overcome by better choice of mask and placement of moisturizer.

Oral devices (splints) are indicated in milder forms of OSA and in patients who do not tolerate CPAP treatment. They act by moving the mandible along with the tongue forward, thus increasing the size of the pharyngeal airways.

Surgical treatment is indicated for patients with moderate to severe apnea who cannot adapt to CPAP treatment. In selected cases, they may undergo uvulopaloplasty or surgical correction of the jaw and nose, which will improve the habituation to CPAP.

Central sleep apnea is the presence of breathing pauses during sleep (according to the general criteria for apnea/hypopnea), which lack respiratory effort (open airways, with no breathing trigger). It is found in some stem lesions - stem strokes, encephalitis and polio, post-hypoxic encephalopathy, olivopontocerebellar degeneration, tumors, etc. Central sleep apnea also occurs with congestive heart failure. Pathophysiologically refers to impaired central chemoreceptor sensitivity and/or delayed circulatory time leading to decreased or increased ventilatory response to CO₂ change during sleep. Clinical manifestation of central sleep apnea may be of the insomnia or hypersomnia type. Treatment of central sleep apnea in chronic congestive heart failure begins with a correction for the treatment of the underlying disease, but oxygen therapy and positive airway pressure may also be attempted.

The treatment of comorbidities is also of great importance. For example, in hypothyroidism it is essential as it can lead to the deposition of mucoproteins in the nasopharynx, obesity and impaired ventilatory control. The treatment of depression in the presence of sleep apnea also leads to an improvement in the outcome for both diseases.

Sleep related hypoventilation disorders:

- Obesity hypoventilation syndrome
- Congenital central alveolar hypoventilation syndrome
- Late-onset central hypoventilation with hypothalamic dysfunction
- Idiopathic central alveolar hypoventilation
- Sleep related hypoventilation due to a medication or substance
- Sleep related hypoventilation due to a medical disorder

Hypoventilation is defined by increased PaCO₂, so the criteria for the diagnosis of sleep-related hypoventilation require presence of either elevated PaCO₂ or directly (measurement of arterial gases) or indirectly (CO₂ at the end of exhalation or transcutaneous CO₂).

ICSD-3 includes criteria for the diagnosis of obesity hypoventilation. These include increases in PaCO₂ (> 45 mmHg) during the day in a patient with a body mass index (BMI) > 30 kg/m². Other forms of hypoventilation during sleep do not require the presence of hypoventilation during the day.

Pulmonary diseases (COPD, asthma) are not accepted a priori for breathing disorders during sleep. However, they can worsen normal breathing during sleep (hypoventilation, hypoxemia). In these cases, sleep-related hypoventilation and hypoxemia are monitored. Sleep-related laryngospasm and choking are not considered respiratory disorders during sleep, but rather somatic disorders occurring during sleep.

There are overlapping syndromes. Treatment with long-term non-invasive home ventilation is based on the accepted ERS recommendations.

Central disorders of hypersomnolence include diseases in which the primary complaint of hypersomnia is not due to another sleep disorder, including disturbed nighttime sleep or circadian rhythm. Excessive daytime sleepiness is defined as episodes of overwhelming need for daytime sleep or falling asleep during the day⁽¹⁾.

Central disorders of hypersomnolence are:

- Narcolepsy type 1 (Narcolepsy with cataplexy ICD 10)
- Narcolepsy type 2 (Narcolepsy without cataplexy ICD 10)
- Idiopathic hypersomnia
- Kleine-Levin syndrome (Recurrent hypersomnia ICD 10)
- Hypersomnia due to a medical disorder
- Hypersomnia due to a medication or substance
- Hypersomnia associated with a psychiatric disorder
- Insufficient sleep syndrome

In the diagnosis of hypersomnia, attention should be paid to the presence of sleep deprivation, especially in patients with greater need for sleep. Completion of sleep diaries and actigraphic examination for at least one week prior to the objective examination with Multiple Sleep Latency Test (MSLT) may help to verify the availability of sufficient sleep, sleep patterns and wakefulness. The diagnostic criteria for narcolepsy and idiopathic hypersomnia include criteria for MSLT.

Narcolepsy. Narcolepsy type 1 includes patients with cataplexy and/or hypocretin-1 deficiency. It is diagnosed with hypocretin 1 deficiency in CSF (<110 pg/mL or less than one-third of standardized reference values) or an average sleep latency of < 8 minutes and two REM periods at the onset of sleep (Sleep onset rapid eye movement periods - SOREMP) within 15 minutes of the start of MSLT recording and/or polysomnography, together with a history of clear episodes of cataplexy. Notably, not all patients with hypocretin 1 deficiency have cataplexy, but they are still thought to have narcolepsy type 1. Some of them develop cataplexy later in the course of the

disease. At present, the study of hypocretin 1 is not widely introduced in clinical practice. Patients have hypersomnia during the day with episodes of sudden falling asleep or an overwhelming need for sleep. After short periods of sleep, they wake up refreshed until the next nap. Some patients may have a change in vigilance during wakefulness combined with automatic behavior. In addition, 30-80% of narcolepsy patients have hypnagogic hallucinations and/or sleep paralysis. Hypnagogic hallucinations are described as living dream-like experiences during the transition from wakefulness to sleep. They are often multimodal - a combination of auditory, visual and tactile experiences. Hypno-pumping hallucinations have similar content, but appear in the transition from sleep to wakefulness. Sleep paralysis is expressed by the inability to move volitional muscles, including the opening of the eyes, the transition from sleep to wakefulness. The patient feels awake but cannot move and the experience is very stressful. Other symptoms of narcolepsy may be blurred vision, ptosis and diplopia, most often as an element of drowsiness. Obesity is also more common in narcoleptics⁽⁸⁾.

Narcolepsy type 1 is also secondary to central nervous system diseases - autoimmune and paraneoplastic diseases associated with anti-Ma2 and aquaporin 4 antibodies, tumors and other hypothalamic lesions or severe cranial trauma.

Narcolepsy type 2 is diagnosed with the same parameters of MSLT for SOREMP described for narcolepsy type 1. However, cataplexy and hypocretin 1 deficiency in CSF are not present. Despite the absence of cataplexy, there may be some unusual changes in the background of intense emotions or stress. Episodes of daytime sleepiness are the main manifestation. There may be sleep paralysis, hypnagogic hallucinations, automatic behavior, memory gaps, ptosis, diplopia, and blurred vision.

Narcolepsy type 2 has been described for the same neurological diseases as type 1, but can also be found in multiple sclerosis, CNS sarcoidosis, myotonic dystrophy, Prader-Willi syndrome and Parkinson's disease.

Idiopathic hypersomnia is diagnosed when the patient has subjective somnolence and a median sleep latency of < 8 minutes with MSLT with less than two SOREMPs in this test and nocturnal examination, no cataplexy and no hypocretin 1 deficiency (if tested). It is important to rule out other causes of hypersomnolence, including sleep deprivation. Characteristic of the disease is sleep inebriation with difficulty waking up, re-falling asleep, automatic behavior and a state of confusion. Patients usually cannot wake up with the usual alarms. The afternoon nap is also more than an hour long and is non-resting. Often there are symptoms that suggest autonomic dysfunction - orthostatism, headache, feelings of temperature dysregulation, or peripheral vascular complaints.

Based on the existing evidence, it is not clear whether idiopathic hypersomnia is a single disease or a group of diseases with different pathological mechanisms and a similar clinical phenotype.

Recurrent hypersomnia. Kleine-Levin syndrome is characterized by recurrent episodes of excessive sleepiness, often associated with cognitive-behavioural abnormalities. A typical episode lasts an average of 10 days (3-80), rarely reaching several months. An episode trigger is usually an infection or abuse of alcohol, and the periods during which the episode occurs are from 1 to 12 months, sometimes even years. During the episode, patients spend 16-20 hours sleeping, waking up only for the natural needs of eating and emptying pelvic vessels. They may remain awake, but they appear exhausted, indifferent, psychomotorically delayed, and if forced to stay awake, become irritable. Typical for them is anterograde amnesia and conditions of derealization. About 66% of patients have hyperphagia, and about half have hypersexuality (mostly men). Some patients have childhood behaviours and about half have depression (mostly women). Patients often have fearful experiences, delusions, and hallucinations, and in the intervening periods, they are completely normal and without complaints.

Other hypersomnia. In case of hypersomnia due to internal and psychiatric diseases, as well as the use of medicines and substances, the diagnosis can be made without MSLT. Determining for the diagnosis of all central hypersomnia is clinical assessment. In addition, many other conditions (OSA, circadian rhythm disorders) can lead to excessive daytime sleepiness, and diagnosis and treatment should precede the diagnosis of central hypersomnia. Patients have prolonged night-time sleep, daytime sleepiness and prolonged afternoon sleep associated with other internal and neurological disorders. Some elements of the disease may be similar to narcolepsy and idiopathic hypersomnia. Hypersomnia has been described in a number of diseases such as metabolic encephalopathy, stroke, CNS trauma, encephalitis, systemic inflammation (chronic infection, rheumatology and cancer), genetic and neurodegenerative diseases.

Hypersomnia due to a medication or substance occurs with the use of sedative drugs, alcohol and other addictive substances, as well as with the withdrawal of stimulant drugs.

Hypersomnia associated with a psychiatric disorder has the general characteristics of hypersomnia, together with a poor-quality sleep complaint. Patients are fixated on their hypersomnolence and psychiatric symptoms often remain in the background. Hypersomnia is more common in mood disorders, conversion and undifferentiated somatoform disorders, and less commonly in schizoaffective and personality disorders.

Insufficient sleep syndrome is due to the separation of longer periods from night-time sleep for social and professional activities. Sleep deprivation has a cumulative effect and people with this syndrome remain in a state of chronic partial sleep deprivation, unable to achieve optimal levels of

alertness and attention. Somatic status provides no explanation for hypersomnia. A detailed history of sleep habits guide to this diagnosis. Sleeping during the holidays or at the end of the week are also typical. Patients with this syndrome may have cognitive and behavioural abnormalities - irritability, attention deficit, energy, dysphoria, fatigue, impatience, discoordination, etc.

Post-diagnosis **treatment** of hypersomnia includes pharmacological and behavioural therapy. The medications used to treat daytime sleepiness and cataplexy are listed in Table 5. Behavioural therapy consists of the following main components:

- Clarification the type of hypersomnia, the course and prognosis of the disease, and the principles of treatment
- Recommending regular periods of 10-20 minutes of sleep every 2-4 hours during the day
- Observance of regular bedtime, getting up and length of the night sleep
- If possible, achieve 9 hours of night sleep.

Circadian rhythm sleep-wake disorders. Circadian rhythm disorders are characterized by chronic or recurrent sleep disturbance due to interference with the circadian system or mismatch between the biological clock of the individual and the environment⁽¹⁾. There are several types of circadian disorders:

- The most common disorders are *shift work and jet lag*, but they often do not reach the physicians' attention. The latter is characterized by varying degrees of difficulty falling asleep, daytime sleepiness, reduced attention and coping with daily tasks, and somatic, most commonly gastrointestinal, travel symptoms and frequent change of time zones. Shift work requires sleep and wakefulness at non-physiological, changing intervals, leading to insomnia and/or hypersomnia.
- *Delayed sleep-wake phase disorder* is characterized by sleep and wakefulness occurring later than usual hours. Once started, sleep is usually normal. The typical patient has difficulty falling asleep and wakes up late. The disease is characteristic of a younger age.
- *Advanced sleep-wake phase disorder* is characterized by sleep and wake-ups that occur earlier than normal hours. The typical patient falls asleep spontaneously early and wakes up early. This disease is less common and is more common in the elderly.
- There is an *Irregular sleep-wake rhythm disorder* in the absence of a clearly defined sleep rhythm and wakefulness. It is common in children with developmental problems and in adults with neurodegenerative disorders such as Alzheimer, Huntington and Parkinson's.
- *Non-24-hour sleep-wake rhythm disorder* is characterized by fluctuating periods of insomnia and/or excess daytime sleepiness that result from a lack of synchronization of the internal

circadian pacemaker with the 24-hour light and dark cycle. Most patients with this disease have total blindness and do not receive a light signal to the circadian pacemaker. In 2014, the US Food and Drug Administration approved the melatonin agonist Tasimelteon to treat circadian disorders in blind patients. It is also the only approved treatment for circadian disorders in America.

- *Circadian sleep-wake disorders not otherwise specified (NOS).*
- *Circadian rhythm sleep-wake disorders due to medical, psychiatric or neurological disorders.*

The general criteria for all circadian rhythm sleep-wake disorders are:

- Chronic or recurrent sleep-wake rhythm disorder that is due to damage to the endogenous circadian system and does not conform to the desired or required sleep and wake pattern.
- The presence of sleep and wakefulness disorders including insomnia and/or excessive daytime sleepiness.
- Disturbance and discomfort associated with the disorder.

A main feature of all diseases in this group is the inability to stay awake or to sleep when necessary. Patients therefore complain of insomnia or hypersomnia. Once a night's sleep has occurred, it is usually normal. In case of suspected presence of other sleep disorders as a cause of insomnia or hypersomnia, the diagnosis of circadian rhythm disturbance should not be made. For example, patients with insufficient sleep who have a change in the sleep and wake phase should not be diagnosed as circadian. All illnesses except jet lag must have a minimum duration of three months. Circadian disorders are best objectified by actigraphy and/or study of biomarkers - melatonin. Validated chronotype questionnaires are also available, which may help with diagnosis but are optional. Keeping a sleep journal can also be helpful. No polysomnographic examination is required.

Treatment. Short planned sleeping periods are recommended when working shifts. It is also appropriate for them to be treated with light therapy at the respective shift periods. Hypnotics, but not stimulants, may also be appropriate in shift working patients. Light therapy is also indicated in patients with an advanced or delayed phase of sleep. Melatonin may be administered at a regimen in cases of jet-lag.

Parasomnias are undesirable physical phenomena (movements, behaviours) or experiences (emotions, sensations, dreams) that occur when falling asleep, during sleep, or when awakening from sleep⁽¹⁾. Observed movements are more complex and appear to be more focused than stereotypical movement disorders during sleep. Parasomnias are divided into parasomnias during

slow wave non-REM sleep, parasomnias during REM sleep and other parasomnias. Most often, parasomnias are benign and often associated with childhood.

NREM-related parasomnias are diseases of waking. They are confusional arousals, sleepwalking (somnambulism) and sleep related eating disorder. The general criteria for the diagnosis of non-REM parasomnia are:

- Recurring episodes of incomplete awakening
- Missing or inadequate response
- Limited or missing cognitive memory or dream memory
- Partial or total amnesia for the event

There are also additional diagnostic criteria for each disease, and patients may also have symptoms for more than one disease - for example, "night fear" (pavor nocturnus) and somnambulism.

REM-related parasomnias include the penetration of REM sleep elements in the waking state (eg. sleep paralysis), exaggregation of REM sleep elements (eg., nightmares), or the absence of essential REM sleep elements (lack of atony during REM with behavioural changes in REM sleep).

The diagnosis of REM sleep behavior disorder (RBD) is made in the presence of recurrent episodes of motor behavioural response and vocalization during REM sleep. Movements can range from shaking and waving your hands to falling out of bed and hitting your partner. Additionally, there must be a record of REM sleep without atony during polysomnography (most often with more than one EMG electrode). Preserved muscle activity or increased phase muscle activity is recorded. Most cases start over the age of 50, most often over 65. These are more commonly men whose first complaint is self-harming or injuring a partner on bedtime. The patient often reports of very vivid and threatening dreams - persecution, self-defence, etc. Very important for diagnosis is the history of the disease given by the bed partner.

The frequency of RBD in the general population is 0.5%, but in the population over 70 and in psychiatric populations it may exceed 5%. The onset of the disease below the age of 50 is more common in women and has a mild clinical course. Clinical manifestations of behavioural changes in REM sleep may also occur with the administration of certain drugs (serotonin and noradrenaline reuptake inhibitors, beta blockers, alcohol and barbiturates withdrawal). In these patients, the diagnosis should be RBD, not parasomnia due to the use of drugs and substances if the criteria for RBD are met. The disease is often combined as a syndrome of overlap with narcolepsy, depression and other parasomnias⁽⁹⁾. Very often, RBD is associated with a number of neurological disorders: multisystemic atrophy, dementia with Lewi bodies, Parkinson's disease, progressive supranuclear palsy, Alzheimer's disease, multiple sclerosis, brain tumors, etc. Therefore, in the presence of

polysomnographic and clinical data for this type of parasomnia, a detailed neurological and imaging study should be performed. It is estimated that more than half of RBD cases can develop Parkinson's disease after 5 to 15 years. Patients with RBD also have a high risk of self-harm and injury to the partner, estimated at 30-95%. Patients with OSA have shown symptoms similar to RBD, which is referred to as a pseudo behavioural change in REM (OSA-related pseudo-RBD).

A variety of drugs are available for the **treatment** of RBD - benzodiazepines, non-benzodiazepine sleeping pills, melatonin and melatonin receptor agonists, dopaminergic, antipsychotic and anti-epileptic drugs. Prior to initiating drug treatment, as with any other parasomnia, the nature of the disease should be clarified and self-injury and family injury measures taken. Such measures are: sleeping low to the floor, mattress on the floor, moving away from furniture, windows or securing windows and doors, securing dangerous and sharp objects, partners sleeping in separate rooms, etc. It is also important to avoid sleep deprivation, avoid antidepressants, alcohol and exciting substance. The only antidepressant that has not been linked to the onset of RBD is Bupropion.

The only parasomnia that has clear indications for drug treatment is RBD. The first choice, as with most parasomnias, is Clonazepam. Initial response was observed in more than 90% of patients at doses of 0.5 to 2 mg, but at the later stage of treatment habituation and attenuation have been observed. The risk of accumulation, daytime drowsiness and increased risk of falls should also be considered.

Recent data suggest that melatonin intake is comparable to the effect of Clonazepam in the treatment of RBD, with a better profile of side effects. Most patients respond to a 3 mg oral dose. The mechanism of the effect of melatonin in these patients remains unclear, but it is suggested that it may be calmodulin antagonism. In the absence of possibility for administration or lack of efficacy of Clonazepam or melatonin, it is not clear what the next choice is. Even for them, the evidence is from small controlled series or from retrospective data. Treatment with acetylcholinesterase inhibitors is recommended in patients with dementia or synucleinopathies⁽⁹⁾.

Nightmares are characterized by recurrent, dreaded dysphoric dreams, which patients remember well after waking and which lead to significant distress and sleep disturbance. Nightmares are often an element of other ailments, such as post-traumatic stress. This diagnosis is made only when the nightmares are the focus of clinical attention and/or require separate treatment.

Other parasomnias. These parasomnias do not occur at a particular stage of sleep. These include: exploding head syndrome, sleep related hallucinations, sleep enuresis, parasomnia due to a medical disorder, parasomnia due to a medication or substance or unspecified parasomnia.

Sleep related movement disorders are characterized by simple, stereotyped movements that disturb sleep⁽¹⁾. Patients may or may not be aware of them.

The diagnosis of sleep related movement disorders requires the presence of sleep disorders such as daytime drowsiness, fatigue, insomnia. Movements that occur during sleep but do not adversely affect the day-to-day functioning of the individual are not considered sleep related movement disorders. Restless legs syndrome (RLS, Willis-Ekbom disease) is also included in this category of disease, but the primary symptom of it is unpleasant, dysesthetic sensations when awake.

Sleep related movement disorders are:

- Restless legs syndrome/ Willis-Ekbom disease (RLS)
- Periodic limb movement disorder (PLMS)
- Sleep related leg cramps
- Sleep related bruxism
- Sleep related rhythmic movement disorder
- Benign sleep myoclonus of infancy
- Propriospinal myoclonus at sleep onset
- Sleep related movement disorder due to a medical disorder
- Sleep related movement disorder due to a medication or substance
- Sleep related movement disorder, unspecified

Restless Legs Syndrome (RLS) is categorized as a sleep related movement disorder because of its association with periodic movements during sleep and its temporal relationship with the onset of sleep, although its clinical manifestation is not with simple, stereotyped movements.

The diagnostic criteria for RLS according to ICSD-3 include the need for foot movement and/or the presence of uncomfortable sensation that occurs during rest in the absence of activity and at least partially alleviates movement (as long as the movement continues). The disorder is circadian in nature and occurs mainly in the evening and at night⁽¹⁾. Another necessary criterion for diagnosis is the presence of complaints of distress or sleep disturbance. Diagnosis is entirely clinical. A number of questionnaires have been proposed to determine the severity of the disease in the literature. The presence of periodic movements during sleep is an additional criterion for diagnosis and requires their registration by polysomnographic examination. Some other diseases such as night cramps, varicose veins, arthralgic complaints, edema, etc. should also be rejected for diagnosis. The mechanism of the disease suggests involvement of the dopaminergic and opioid systems in the CNS, which is why it is appropriate to test serum iron (such as the cofactor of the tyrosine hydroxylase enzyme, which is important for dopamine synthesis) and ferritin. The

frequency of RLS in different populations varies between 2-11%, increasing with age. The frequency is also higher in pregnant women, in patients with Parkinson's disease and in cases of terminal renal failure⁽¹⁰⁾.

Treatment of the disease depends on a number of factors such as the severity of the complaints, the age of the patient, the presence of concomitant illnesses and complaints (pain, depression, anxiety, obsessive-compulsive behavior), the side effects of the medication, and the patient's preferences. The disease is most often chronic, and clinical studies provide us with follow-up data within up to 12 weeks. Treatment is generally non-pharmacological and pharmacological.

Non-pharmacological treatment includes, in particular, the avoidance of factors that enhance RLS: sleep deprivation, use of psychotropic drugs (antidepressants of all groups, antipsychotics and other drugs blocking the dopamine system), sedating antihistamines, caffeine, etc. In many patients, antidepressants cannot be stopped, requiring RLS to be treated in the same way as the primary one. Other methods of relief are moderate exercise, massages, pneumatic socks, cycling, etc.

Serum ferritin concentrations below 50 mcg/L have been associated with increased RLS severity. Oral or parenteral iron replacement therapy at ferritin levels below 75 mcg/L is recommended.

The pharmacological treatment of RLS requires that the disease is moderately and severely chronic and the symptoms should be at least two evenings a week. Dopaminergic agents and alpha-2-delta calcium channel ligands are the drugs of choice. The choice of medication depends on the comorbidities:

- For patients with severe level of the disease, comorbid depression and/or metabolic syndrome, dopamine agonist treatment should be preferred.
- For patients with comorbid pain, insomnia, anxiety, history of habituation, and impulse control difficulties, alpha-2-delta calcium channel ligand is preferred.
- In most patients, starting with the alpha-2-delta calcium channel ligand is preferred because of the potentiation in case of administration of dopamine agonists.
- Elderly patients are more susceptible to the side effects of alpha-2-delta calcium channel ligands.
- Of the dopamine agonists, Pramipexole (0.125-0.25 mg) and Ropinirole (0.25-3 mg) are preferred, the latter showing a higher class of evidence from clinical trials. The medicines are administered once a night, about 2 hours before the onset of symptoms. The administration of Rotigotine in the form of a 24-hour patch is less effective and starts with the lowest possible dose.
- Of the alpha-2-delta calcium channel ligands, the most effective are Gabapentin enacarbil and Pregabalin at a dose of 75-300 mg daily. Gabapentin has limited efficacy data for RLS.

- Non-pharmacological agents should be tried primarily in pregnant women and, in the absence of effect, the less effective low dose Levodopa and Clonazepam may be administered.

Periodic limb movement disorder (PLMS) are often recorded during polysomnography. They are diagnosed when more than 15 movements per hour are recorded during polysomnography and more than 5 movements in children. In addition, the patient should complain of a sleep disturbance or functional impairment associated with the periodic movements.

The diagnosis should not be established in the presence of untreated OSA, RLS, narcolepsy or RBD, as periodic limb movements are common in these diseases.

Bruxism (tooth grinding) is more common at a young age (up to 20% of the total population). Often, it is observed by the partner in bed and does not constitute a genuine complaint by the patient. Not infrequently bruxism is combined with periodic limb movements. In case of tooth enamel disturbance, silicone bars are used to treat bruxism.

Night cramps in the calves and soles occur mainly during sleep and represent painful contractions in the affected area.

Sleep related rhythmic movements are benign and require treatment only if they cause insomnia or are secondary to another disease.

Other sleep disorders. This group includes diseases that cannot be classified precisely in another section of the ICSD-3 classification, either because the disease overlaps with more than one category or because there is insufficient data to make any of the above diagnoses⁽¹⁾. Diseases that are mainly related to environmental impacts could also be classified here.

Isolated symptoms and normal variants include symptoms occurring during sleep that do not meet the criteria for sleep disorders⁽¹⁾.

- Some happen during normal sleep, such as somniloquy (sleep talking).
- Some are on the border between normal and pathological. For example, banal snoring, without affecting respiratory function, the presence of sleep disturbance or other consequences, is considered normal, while severe snoring is more often an element of obstructive sleep apnea.

Specification of the activity for the diagnosis and treatment of sleep disorders and wakefulness (SDW)

A. GPs' actions to diagnose SDW - outpatient care

The diagnosis of SDW by GPs involves the following activities:

- Making a detailed medical history, including a family history of sleep disorders and other illnesses
- Interview with the relatives, parents and partner in the room or bed
- A general examination for finding comorbidities
- In case of symptoms referring to SDW, the patient should be referred to specialist neurologists/pulmonologists and subsequently to specialized laboratories for SDW testing to be diagnosed by a physician trained in SDW.

B. Actions of the specialist (neurologist) for the diagnosis of SDW - outpatient care

- Makes a detailed medical history, including a family history of sleep disorders and other illnesses, including in conversation with the partner or parents
- Takes the neurological status to identify concomitant diseases
- Refers the patient to specialized laboratories for SDW testing to be diagnosed by a physician trained in SDW.

C. Actions of GPs with patients with confirmed diagnosis of SDW

- Depending on the type of illness, the GP conducts regular monitoring of the patient in the form of an interview and examination.
- Prescribes monthly the treatment prescribed by the specialist, without altering neither the medication nor the non-medication treatment.
- Ensures compliance with the hygienic and general sleep measures prescribed by the specialist and with patients with OSA treated with CPAP, monitors the use of the device on a regular basis and the occurrence of complications.

D. Actions of the physician in the specialized laboratories for SDW - hospital care

- In order to confirm or reject the diagnosis, the SDW specialist physician takes a thorough general, neurological and psychiatric status. In addition, he interviews the patient and his or her relatives, completing appropriate scales for presence of SDW symptoms.

- For definitive clarification of the type of nocturnal disease it is necessary to appoint and carry out specialized studies: polysomnography, limited polygraphy, night videography, Multiple Sleep Latency Test (MSLT), genetic tests, etc.
- **Polysomnography** is the registration of breathing (respiratory flow through the mouth and nose, thoracoabdominal respiratory effort, transcutaneous oxygen saturation), sleep (electroencephalogram, -oculogram, -myogram), ECG and motor activity (recorded videographically and electromyographically).
- **Limited polygraphy** involves the recording of respiratory parameters only (respiratory flow through the mouth and nose, thoracoabdominal respiratory effort, transcutaneous oxygen saturation), ECG and body posture. The method also enables the individualization and control of the effectiveness of CPAP treatment.
- The **Multiple Sleep Latency Test** is a study of electroencephalogram, -oculogram, and -myogram to determine the time of falling asleep and pathological events in five sleep attempts of 20 minutes during the day.
- The performed appliance tests receive automatic and manual evaluation, and after clinical discussion, most often by a group of specialists, the patient is diagnosed.
- Due to the necessity of spending 2-3 nights in laboratory (hospital) conditions for the diagnosis of SDW, most of the diagnostic activities are a priority for hospital care.
- Positive pressure titration, therapeutic attempts for positive pressure treatment and follow-up should be performed by a SDW specialist (neurologist, pulmonologist).

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Table 1. Sleep disorders that can mimic an insomnia

Sleep related breathing disorders

Obstructive sleep apnea

Central sleep apnea

Sleep related movement disorders

Restless legs syndrome

Sleep related leg cramps

Bruxism

Nocturnal myoclonus (at sleep onset)

Sleep related rhythmic movement

Central disorders with hypersomnolence

Narcolepsy

Parasomnias

Sleepwalking

Sleep terrors

Nightmares

Circadian rhythm sleep-wake disorders

Advanced sleep-wake phase

Delayed sleep-wake phase

Shift work

Irregular sleep-wake rhythm

Others

Sleep related epilepsy

Sleep related laryngospasm

Sleep related asystole

Table 2. Other diseases that can lead to sleep disorders

Cardiovascular diseases

Arrhythmias

Congestive heart failure

Myocardial infarction

Limb varices

Pulmonary diseases

COPD

Scoliosis

Interstitial diseases

Gastrointestinal diseases

Ulcer

Gastroesophageal reflux disease (GERD)

Chronic constipation

Diseases of the Central Nervous System

Space-occupying processes

Dementia

Epilepsy

Stroke

Parkinson's disease

Neuromuscular diseases

Urological diseases

Benign prostatic hypertrophy

Chronic kidney disease

Musculoskeletal disorders

Rheumatoid arthritis

Diseases of the connective tissue

Table 3. Medications that lead to insomnia

ANTIDEPRESSANTS

Serotonergic (fluoxetine, fluvoxamine)

HYPNOTICS AND ANXIOLYTICS

Benzodiazepines can induce insomnia, such as phenomena of tolerance and effect depletion, or as a phenomenon of withdrawal in benzodiazepines with ultra-short and short half-lives.

PSYCHOSTIMULANTS

Amphetamines

Modafinil

ANTIEPILEPTICS

Carbamazepine

Phenobarbital and benzodiazepines in children

ANTIPARKINSONIAN DRUGS

Levodopa

Bromocriptine

CARDIO VASCULAR DRUGS

Methyldopa

Clonidine

Betablockers and calcium antagonists

PULMOLOGICAL DRUGS

Theophylline

Salbutamol and specific agonists of β_2 receptors

ANTI-INFLAMMATORY DRUGS

Corticosteroids

Chloroquine and gold salts

HORMONAL MEDICINES - thyroid

Table 4. Sleep medicines

/time to reach maximum plasma concentration and elimination half-life/

Medicines	Tmax /h/	Half-life /h/
With slow elimination /24 h/		
Clozapate	1	40
Diazepam	from 0,5 to 1,5	32
Nitrazepam	2	25

With medium elimination /6 – 24 h/		
Bromazepam	from 1 to 4	20
Flunitrazepam	1	19
Lorazepam	1	from 10 to 20
Alprazolam	0,9 to 2,4	from 12 to 15

Medicines	Tmax /h/	Half-life /h/
With fast elimination /6 – 3 h/		
Zopiclone	from 1, to 2	5
With very fast elimination /< 3 h/		
Midazolam	10 min.	2
Triazolam	1,5	2,7
Zolpidem	from 0,5 to 3	2,4

Table 5. Medicines for excessive daytime sleepiness and cataplexy

I. FOR EXCESSIVE DAYTIME SLEEPINESS	
Dextroamphetamine	5-30 mg per day to 60 mg per day
Methamphetamine	5-30 mg per day to 60 mg per day
Methylphenidate	10-30 mg per day to 60 g per day
Other non-analeptics	

Mazindol	2- mg
Anorexics	
Amfepramone	75 mg
Fenethylline	50-100 mg
Alpha1 adrenergic agonists	
Modafinil	100-400 mg
Dopaminergic agonists	
L-tyrosine	60-120 mg/kg
Antidepressants	
Venlafaxine	75-150 mg
Fluoxetine	20 mg
Selegiline	5-20 mg
II. FOR CATAPLEXY	
1. Tricyclic antidepressants	
Clomipramine	10-75 mg
Imipramine	10-75 mg
Desipramine	25-50 mg
2. Antidepressants inhibiting serotonin reuptake	
Fluoxetine	20-60 mg
Fluvoxamine	50-100 mg
3. Noradrenergic antidepressants	
Viloxazine	100-300 mg
Gamma – OH Butyrate	
4. Monoamine oxidase B inhibitors	
Selegiline	5-20 mg
5. Alpha1 adrenergic agonist	
Modafinil	100-400 mg
6. Substances releasing monoamines	
Amphetamines and methylphenidate	

Figure 1. Epworth Sleepiness Scale – *published with permission on Johns MW, A new method for measuring daytime sleepiness: the Epworth sleepiness scale. Sleep, 1991, 14 (6),540-5.*

EPWORTH SLEEPINESS SCALE

How likely are you to doze off or fall asleep in the following situations? This refers to your usual way of life in recent times. If you haven't been in a similar situation recently, try to work out how you would have reacted in such situations.

Choose the most appropriate answer from 0 to 3 for each situation, where:

0 – never; 1 – unlikely; 2 – likely; 3 – with a high probability.

1. Sitting and reading				
2. Watching TV				
3. Sitting, inactive in a public place (e.g. a theatre)				
4. As a passenger in a car for an hour without a break				
5. Lying down to rest in the afternoon				
6. Sitting and talking to someone				
7. Sitting after a lunch/dinner without alcohol				
8. In a car if you are the driver				

Total:

Figure 2. STOP-BANG Questionnaire – *modified on Chung F, et al. Anesthesiology 2008, 108, 812-21.*

STOP-BANG QUESTIONNAIRE

Height _____ cm Weight _____ kg	
Age _____	
BMI _____	
Shirt collar circumference: S, M, L, XL, or _____ cm	
Neck circumference/size _____ cm	
1. Snoring	
Do you snore loudly (louder than talking or loud enough to be heard through closed doors)?	
Yes	No
2. Tiredness	
Do you often feel tired, fatigued or sleepy during the daytime?	
Yes	No
3. Observations	
Has anyone observed you stop breathing during your sleep?	
Yes	No
4. Do you have or are being treated for high blood pressure?	
Yes	No
5. BMI	
Is your BMI more than 35 kg/m ² ?	
Yes	No
6. Age	
Are you older than 50?	
Yes	No
7. Neck circumference/size	
Is your neck circumference/size more than 40 cm?	
Yes	No
8. Gender?	
Are you a male?	
Yes	No
Neck circumference is measured by the staff.	