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Insomnia and Sleep Disruption: Relevance for Athletic Performance

Damien Leger, MD, Biol D[a,*], Arnaud Metlaine, MD[a], Dominique Choudat, MD[b]

[a]*Centre du Sommeil, Hotel Dieu de Paris, 1, Place du Parvis Notre Dame, 75181 Paris Cedex 04, France*
[b]*Department of Occupational Medicine, Hopital Cochin, University of Paris V, School of Medicine, 45, Rue de s Saintes-Peres, 75270 Paris Cedex 06, France*

Insomnia is a very common complaint among adults around the world [1,2]. Despite its high occurrence, insomnia is still largely unrecognized by health professionals. One reason is that insomnia is frequently considered a symptom rather than a true disease; practitioners are not clear if it should be considered a symptom and a disease. Another challenge is that it is often difficult for patients and health professionals to understand when insomnia is severe enough to require treatment. Furthermore, there is still insufficient knowledge about the management of insomnia. Several consensus meetings have convened in the last decade to address the recognition, diagnosis, and treatment of insomnia [3–7]. This paper's aim is to resume the major aspects of these discussions to help sports doctors and scientists manage insomnia in athletes.

Recognition and diagnosis of insomnia

Definition of insomnia: criteria

In terms of clinical practice and epidemiology, chronic insomnia is usually defined based on the criteria of the *Diagnostic and Statistical Manual of Mental*

* Corresponding author.
E-mail address: damien.leger@htd.aphp.fr (D. Leger).

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Disorders (DSM-IV) [8] or the *International Classification of Sleep Disorders* (ICSD) [9]. These may include

- Difficulty falling asleep (sleep-initiating insomnia), the occurrence of nocturnal awakenings with difficulties getting back to sleep (sleep-maintenance insomnia), an early morning awakening (sleep-offset insomnia), or a nonrefreshing or nonrestorative sleep, and often a combination of all of these factors
- Symptoms occurring at least three times a week for a minimum of 1 month
- Clinically significant distress or impairment in social, occupational, or other important areas of daytime functioning

Insomnia may be primary or secondary to a variety of disorders, environmental factors, or comorbidities. Identifying and treating potential underlying conditions are priorities in the management of insomnia. Otherwise, insomnia will remain unresolved.

Primary insomnia

Primary insomnia is an intrinsic sleep disorder that is characterized by the presence of insomnia that

- Does not occur exclusively during the course of another sleep disorder (eg, sleep-disordered breathing, periodic limb movements, restless leg syndrome, or circadian rhythm disorder)
- Does not occur exclusively in the course of another mental disorder
- Is not caused by the direct physiologic effect of a general medical condition, substance or treatment, or physical environmental factor

A common mechanism of persistent insomnia is conditioned (ie, learned or psychophysiologic) insomnia. It begins usually by an episode of acute situational insomnia, secondary to insomnia-precipitating factors such as a stress, jet lag, pain, illness, or medication. The patient then begins to associate bed with nonsleeping, becomes hyperaroused at night, and develops strategies for coping that perpetuate insomnia (insomnia-perpetuating factors) [10]. Individual differences in vulnerability to sleep disturbances may constitute a continuum from transient or episodic insomnia through overt chronic primary insomnia [11]. Ruminating about not being able to sleep plays a major role in perpetuating insomnia.

Secondary insomnia

Insomnia is frequently associated with numerous other conditions, such as sleep disorders, mental or physical disorders, or toxicologic or environmental factors. The role of the practitioner is to carefully check all of these conditions before considering insomnia as a primary disorder.

Almost all other sleep disorders disturb sleep seriously enough to induce a complaint of insomnia or poor sleep, as Emsellem et al describe elsewhere in this issue. Sleep apnea affects 5% to 10% of the general population, increases with age and body mass index, and causes arousals and awakenings during all stages of sleep. Patients usually complain of nonrestorative sleep rather than real insomnia. Restless leg syndrome may also affect sleep initiation and sleep maintenance. Restless leg syndrome affects between 5% and 10% of the general population and may be associated with apnea. Circadian rhythm disorders are linked to a dysfunction of the biological clock, caused by an internal condition (delayed or advanced phase syndromes) that is secondary to the underexposure of the retina to light (eg, as with blind people) or a misalignment between external and endogenous rhythms (eg, caused by shift work or jet lag). Circadian rhythm disorders may induce sleep-onset insomnia, early morning awakening, frequent arousals, and daytime sleepiness.

Mental disorders or comorbidities are commonly associated with insomnia. One survey of the general population found that 30.1% of the subjects who suffered from insomnia reported prior consultations for anxiety symptoms, and 23% reported prior consultations for depression [12]. Similarly, in primary care patients who experienced severe insomnia, there was a high prevalence of psychiatric diagnoses: 21.7% of severe insomniacs suffered from depression, 7.2% had neurosis/personality disorders, 10.2% suffered acute psychologic distress, 4.6% reported alcohol or drug abuse, 5.6% had psychosomatic disorders, and1% demonstrated psychosis [13].

A variety of medical disorders may impact on sleep and awaken patients, such as central nervous system disorders, cardiorespiratory troubles, musculoskeletal disorders, and pain. Studies of specific populations reveal a strong correlation between pain and complaints of sleep disturbances [14]. Several endocrine and gastrointestinal disorders are also associated with sleep disruption. For example, nocturnal gastrointestinal reflux episodes may arise during sleep and induce abrupt arousals [15]. A large number of medications and toxins (eg, alcohol, drugs) also impact sleep continuity.

Environmental factors may also induce sleep disruption and fragmentation, even in good sleepers. Noise is one of the most common factors. Recently, the World Health Organization European Centre for Environment and Health considered insomnia one of the major health effects of noise exposure [16]. Low or high temperature, altitude, and light also influence sleep continuity.

Transient or chronic insomnia

The duration of a patient's complaint has important implications. The ICSD defines acute or transient insomnia as persisting for no more than 1 week and subacute or short-term insomnia as lasting from 1 week to 3 months [9]. Both types of insomnia are considered adjustment sleep disorders that are associated with a reaction to an identifiable stressor. Transient insomnia usually disappears with the reduction of or adaptation to the stressor; however, it may also be the

foundation of a long-term condition. The individual's emotional and behavioral responses to the first episodes of transient insomnia seem to play an important role in the course of the disease [10,11,17]. Therefore, early identification and management of insomnia may play a role in the prevention of long-term insomnia. Insomnia is considered chronic if it lasts more than 1 [8] to 3 months [9]. This article's investigators believe 1 month is a reasonable period to begin to talk of chronic insomnia. Retrospective studies indicate that about 80% of severe insomniacs have had the problem for more than 1 year, with approximately 40% reporting a duration of more than 5 years [12,18]. Longitudinal studies suggest that 30% to 80% of moderate to severe insomniacs show no significant remission over time [5,18,19].

Severity

There is no clear consensus on the definition of severe insomnia; it seems insufficient to base it on patient reports. Many studies have observed that a large number of so-called "severe insomniacs" did not consult any practitioner for years about their sleep problem. Chronic duration or nightly frequency may be criteria for severity. The magnitude of the impaired daytime functioning may also be used to assess severe insomnia. In several studies focused on the daytime consequences of insomnia, severe insomniacs were considered to be subjects reporting at least two symptoms of poor sleep according to the DSM-IV definition of insomnia [20,21].

Epidemiology

Many studies in the past decade have used the clinical criteria for insomnia to assess its prevalence in the general population and some subgroups of adults [1,2,5,12]. These studies found a median prevalence for all insomnia of about 15% with a range of 10% to 25%. The studies also revealed that insomnia is more common in women than in men and that prevalence increases with age. With few exceptions, these studies do not attempt to identify etiologies of insomnia.

Sleep disruption and insomnia

The terms *insomnia* and *sleep disruption* are sometimes confused. Sleep-maintenance insomnia is defined by the occurrence of sleep disruptions (eg, arousals, awakenings) during sleep. Sleep may be disrupted by other sleep disorders or organic diseases, such as sleep apnea, restless leg syndrome, pain, and cardiac arrhythmia, and also by environmental disturbances, such as noise and temperature. Sleep disruption is always associated with an increased wake after sleep onset (WASO) and a decreased sleep efficiency. It may also affect the percentage of slow-wave and rapid eye movement (REM) sleep.

Subjective and objective assessments

Several subjective and objective tools are used by sleep specialists to assess insomnia, attempt to find a cause, and encompass the severity of the syndrome.

Subjective

Sleep logs are widely used in practice. These logs include data about time in bed, rising time, sleep latency, number and duration of awakenings, and napping. They are also frequently completed by visual analogic scales that inquire about sleep quality, whether there is a refreshed feeling in the morning, and about daytime functioning. Studies evaluating sleep logs versus objective recordings have shown that patients have a tendency to underestimate their total sleep time and overestimate their sleep latency on sleep logs [22,23]. However, the sleep log is a very easy and convenient tool to assess sleep symptoms on a medium–long-term basis.

Sleep questionnaires are also frequently used to address characteristics of sleep complaints and behaviors and to find the causes of secondary insomnia. The most widely used questionnaire in research and clinical practice is the Pittsburgh Sleep Quality Index [24], which has been applied in many settings to evaluate sleep in the elderly and in patients with psychiatric disorders, and in evaluating treatment response [25–27].

Recognizing psychiatric diseases that cause insomnia or that coexist with insomnia is one major aspect of the diagnosis. Psychiatric conditions are highly prevalent in insomniacs. Therefore, patients should be carefully interviewed to determine their psychiatric history and their current psychiatric complaints and treatments. In research, and in the case of a chronic and unresolved complaint of insomnia, the self-administrated Primary Care Evaluation of Mental Disorders [28], the Profile of Mood States (POMS), and the Minnesota Multiphasic Personality Inventory may help physicians recognize mental disorders, and have therefore been used extensively in insomnia [29–31].

Objective

Actigraphy is a very simple device that looks like a watch and is worn on the wrist to record the number and amplitude of movements in a medium–long-term period (at least 1 week). When used conjointly with a sleep log, it provides an estimate of total sleep time (TST), sleep latency, sleep efficiency, and the number of awakenings. It may also give an estimate of napping and bouts of sleepiness during the daytime. Sleep habits are also well assessed by actigraphy, especially when the lag time between workdays and weekends is observed. Actigraphy has been used extensively in studies on insomnia. It is not indicated as a primary endpoint tool, although it may be useful for patients who have chronic and severe insomnia, or if there is a suspicion of a circadian disorder caused by shift work, jet lag, phase delay, or free-running rhythms [32].

Actigraphy has also been used with success in combination with respiratory polygraphy to detect sleep apnea [33]. In a recent study comparing polysomnography, actigraphy, and use of a sleep diary in 17 insomniacs, Vallières and Morin [34] concluded that, compared with polysomnography, actigraphy and sleep-diary instruments underestimated TST and sleep efficiency and overestimated total wake time. Also, actigraphy underestimated sleep-onset latency, whereas the sleep diary overestimated it compared with polysomnography (PSG). Actigraphy data were more accurate than sleep-diary data when compared with polysomnography. Finally, actigraphy was sensitive in detecting the effects of treatment on several sleep parameters. The investigators concluded that "actigraphy is a useful device for measuring treatment response and that it should be used as a complement to sleep-diary evaluation" [34].

PSG consists of one all-night simultaneous recording of the electroencephalogram (EEG), electromyogram, and electro-oculogram, considered conjointly with other secondary variables (eg, cardiopulmonary measurements, temperature, body position, leg movements). PSG may be used for insomnia when a primary sleep disorder is suspected, such as sleep apnea syndrome or restless leg syndrome. PSG is usually performed in a sleep laboratory; however, ambulatory recordings are also easily performed on insomniacs. Edinger et al [35] have shown that 34% of one sample of 100 chronic insomniacs who conducted ambulatory home recordings had PSG-dependant diagnoses and that the home assessment provided useful information for 65% of the patients. Other studies have shown a high level of night-to-night variability in home PSG [36], and an important first-night effect that made the use of ambulatory PSG very controversial in the clinical assessment of insomnia by itself [37]. WASO, sleep latency, and TST are the commonly accepted criteria to describe the presence and severity of insomnia.

A very simple device, the Nightcap, which is based on head-movement actigraphy and the detection of eyelid movements using a piezoelectric film sensor attached to the upper lid, has been shown to be a highly reliable tool to assess sleep latency, REM sleep, and TST, comparable to PSG [38]. Compared with conventional EEG techniques, the Nightcap is unable to separate slow-wave sleep from stage 1 and 2, but more acceptable in real-life settings such as sport. Q6

Consequences of insomnia Q7

Insomnia and daytime functioning

Even if there is a large consensus that the daytime impact is a major criteria in the definition of insomnia [8,9], the nature of this impact is still controversial. Recent studies have underlined objective impairments in subjects with primary insomnia, such as a high level of cortisol [39] and higher heart rate variability [40]. However, these objective data do not implicate a demonstrated link between insomnia and fatigue, irritability, and impairment of daytime functioning. In their review on insomnia and daytime functioning, Riedel and Lichstein [41] proposed

that a paucity of objective findings in the literature may have occurred either because attempts at objective verification have focused on variables that are not impaired rather than areas of actual impairment, or because methodological problems, such as between-subjects variability, have hidden actual differences between insomniacs and persons with no insomnia. Daytime sleepiness has received the most attention, but it is becoming clear that a large number of insomniacs are not sleepy during the day [42,43]. Using MSLT, Bonnet and Arand [43] demonstrated that insomniacs were even more alert than good sleepers during the daytime. Q8

Relevance for athletic performance

The impact of insomnia on athletic performance is a crucial issue. Many athletes consider competition stressful enough to impact their sleep the night before the event and may redoubt the influence of poor sleep on performance. Q9 There are few studies, if any, devoted to the impact of insomnia on athletic performance. Optimum performance requires a combination of physical and cognitive excellence, so it is important to understand how insomnia may affect both aspects. Travel and jet lag are also sleep disruptive and frequently experienced by athletes before competition, as discussed by Reilly et al elsewhere in this issue.

On the physical aspect, some experimental work has been made recently on sleep deprivation and physical activity in healthy volunteers. Mougin et al [44] tested the effect of a voluntary partial sleep deprivation (3 hours) in seven athletes. Their findings showed a significant increase in heart rate and ventilation at submaximal exercise on the day after sleep deprivation, compared with the results obtained after a baseline night. Variables were also significantly enhanced at maximal exercise, although the peak of VO_2 dropped even though the maximum sustained exercise intensity was not different [44]. Meney et al [45] also observed the effect of one night's sleep deprivation on temperature, mood (assessed by POMS), and physical performance of 11 healthy men. The physical activity indicators included muscle strength, self-chosen work rate, perceived exertion, and heart rate. There was a significant negative effect of sleep deprivation on mood state the day after sleep deprivation, but no significant effect on other variables. The investigators commented that there were considerable interindividual variations in the responses to sleep loss among subjects and therefore it was difficult to give general advice about sport training capability after sleep loss.

Another study involved 13 healthy men who were sleep deprived for 1 night and tested the following day at 6 AM and 6 PM and the results compared with tests performed the day before [46]. The physical performance was assessed by the maximal power [P(max)], the peak power [P(peak)], and the mean power [P(mean)]. Blood lactate concentrations were measured at rest, at the end of the force-velocity test (F-V), and just after the Wingate test, and again 5 minutes later. Oral temperature was also measured every 2 hours. Analysis of variance

revealed a significant (sleep × time of day of test) interaction effect on P(peak), P(mean), and P(max). These variables improved significantly from morning to afternoon during the baseline condition and after sleep deprivation. The reference night was followed by a greater improvement than the SDN. Anaerobic power variables were not affected up to 24 hours after waking; however, they were impaired after 36 hours without sleep. Analysis of variance revealed that blood lactate concentrations were unaffected by sleep loss, by time of day of testing, or by the interaction of the two. The investigators therefore concluded that sleep deprivation reduced the difference between morning and afternoon anaerobic power variables. Anaerobic performances were unaffected after 24 hours of wakefulness but were impaired after 36 hours without sleep [47]. A more recent study by these investigators on 19 young athletes showed the impact of circadian rhythms on performance during anaerobic cycle leg exercise, which was more easily assessed than the effect of sleep deprivation by itself.

In addition to physical performance, attention, concentration, and memory have major impacts in some sport competitions, and therefore insomnia may also impact these cognitive factors of performance excellence. However, it is not clear how insomnia affects next-day cognitive performance. In a review of 56 studies exploring the impact of poor sleep on cognitive dysfunction, Fulda and Schulz [48] found that neuropsychologic functions, such as attention, verbal immediate memory, and vigilance, were affected in only 22.8% of the studies. Impairment of memory performance was present in 20% of the studies on insomniacs compared with control subjects. Bonnet and Arand [49] also demonstrated that experimentally induced sleep disturbance, matched to the degree of disturbance in objective insomniacs, failed to produce significant performance decrements. Sateia et al [5] hypothesize that

> The absence of consistent deficits in this area suggests that either the current assessment methodology is not sensitive enough to detect impairments or that insomniacs amplify the adverse effects of disturbed sleep. Alternatively, it may also be the case that under mild sleep deprivation most individuals are able to sustain performance in a time-limited testing situation.

This discussion and the contradiction between the patient's feeling and the absence of objective performance deficits are illustrated in Fig. 1 and Fig. 2, where actigraphy was performed on athletes who were complaining of insomnia in our laboratory.

Fig. 1 illustrates the 21-day actigraphy of a 28-year-old woman, who was at the time of the recording the European champion in athletics and an Olympic gold medalist. She complained of insomnia the nights before important selections, especially when the race was early in the morning. The actigraphy showed regular and evening-oriented sleep schedules with an average bedtime of midnight and an average awakening time of 9 AM. Night awakenings were rare, the average sleep latency was 18 minutes, and the average TST was 7 to 8 hours. The peak of diurnal activity was around 11 AM, coinciding with the time of her

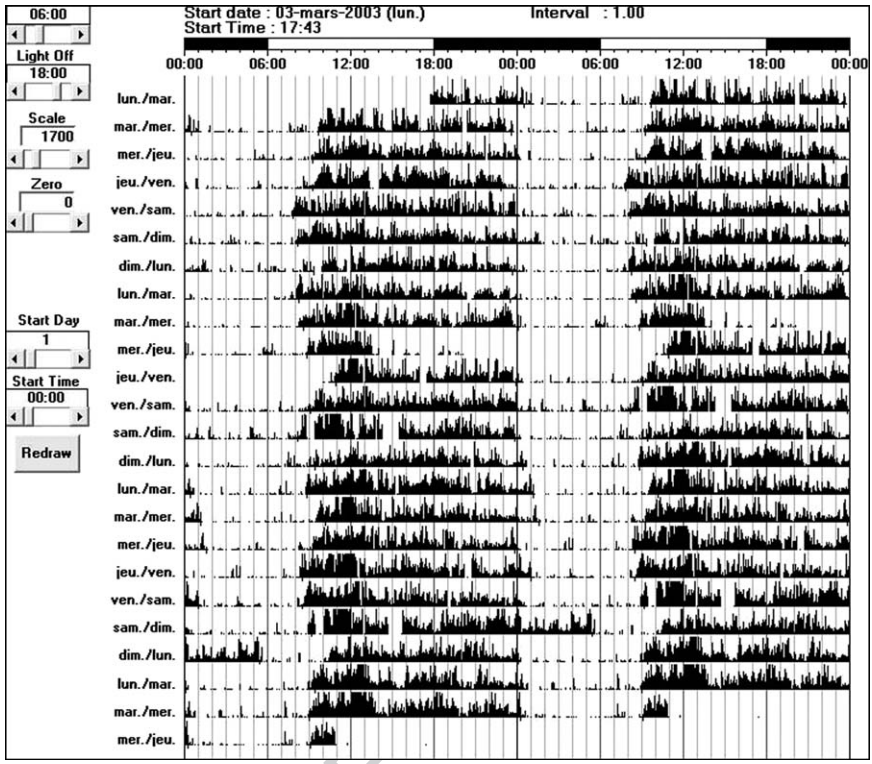

Fig. 1. 21-day actigraphy of a 28-year-old female athletic champion who complained of insomnia on the nights before important selections. Every line shows 48 hours of sleep/wake. The black parts of the lines represent daytime activity. The irregularity reflects the different levels of activity during the day, and the white part reflects the nights. Using the midnight line (00:00) as a reference, it is clear that the subject is rarely in bed before midnight. Referring to the end of the nights, the subject demonstrates irregular morning awakenings. However, there is almost no lag between workdays and weekends and no napping in the afternoon. Finally, the actigraphy shows regular and evening-oriented sleep schedules, with an average bedtime of midnight and an average awakening time of 9 AM. There were rare night awakenings, the average sleep latency was 18 minutes, and the average total sleep time was 7 to 8 hours. The peak of diurnal activity was around 11 AM (coincidental to the time of her regular training exercise) demonstrated by the black density, indicating that the daily pattern is higher at this time of day. Q14

regular training exercise. This subject's complaint of insomnia did not fit with the objective assessment: she did not take any medication and had a rather good sleep at the actimetry. She received only cognitive behavioral therapy and won the European championship during the summer.

Fig. 2 is the 41-day actigraphy of a 38-year-old healthy man who was at the time the winner of a cross-oceanic sailing race. He complained of sleep-initiating and sleep-maintaining insomnia, exacerbated in the days preceding each race. The investigators performed an actigraphy during a selection race including 3 nights of sailing and four following days of 24-hour regattas, and then regis-

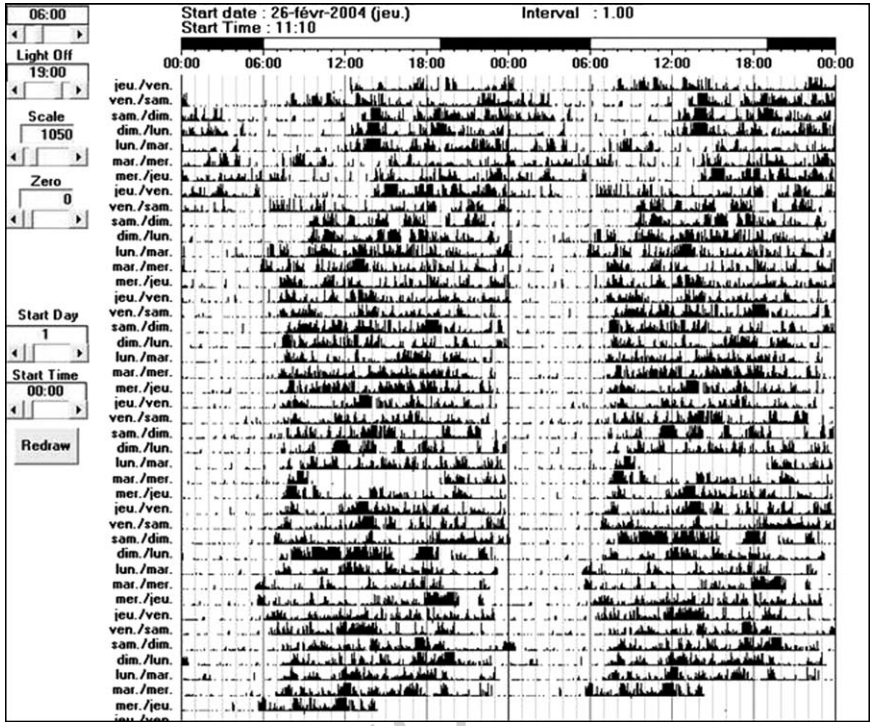

Fig. 2. 41-day actigraphy of a 38-year-old male sailor who was at this time the winner of a cross-oceanic sailing race. The subject complained of sleep-initiating insomnia and sleep-maintaining insomnia, exacerbated in the days preceding each race. An actigraphy was performed during a selection race including 3 nights of sailing and four following days of 24-hour regattas. Differences can be observed in the two parts of the figure. There are not regular patterns of sleep on the first days,. The subject is sleeping normally on the first line, but on the three following lines, it may be observed that he went to sleep around 6 AM after the afternoon and night regattas and his sleep is disturbed, with the thin lines representing night awakenings. The four following lines show that the subject was managing to stay awake as much as possible to stay alert during the race. He slept around 3 to 4 hours in 24 hours on a multiphasic pattern. Then, he went back to more regular patterns, going to bed around 11:30 PM and waking up around 7 AM during the 34 days after competition. However, the hour of awakening is irregular.

tered him during the next 34 days without competition. The investigators found that he phase shifted pretty well on night shift during the first part of the competition, with an average TST of 6.4 hours between 6 AM and noon, and that he managed around 4 hours a day of polyphasic sleep (several phases of short sleep among the 24-hour cycle) during the 3 days after. He then returned to his regular schedule of going to sleep at midnight and getting up at 7 AM. He received advice on sleep hygiene, relaxation training, avoiding intense exercise and bright light before sleeping, and protection against noise. The use of hypnotics was discussed, but the athlete thought it unnecessary at the time.

Management of insomnia

There are several consensus publications about sleep management in insomnia [3–7], which involves educational, behavioral, and often pharmacologic intervention. There is ample evidence from this consensus that a multidisciplinary approach is required for the management of insomnia. Based on a review of 123 controlled medication studies (N = 9114 patients) and 33 controlled behavioral intervention studies (N = 1234 patients), Kupfer and Reynolds [6] concluded that

> Subjective symptoms and objective signs of chronic insomnia respond to short term behavioral and pharmacologic intervention. Although pharmacologic appear to act more reliably in the short term and behavioral interventions appear to produce more sustained effects, no direct comparisons with respect to long term efficiency are available. On the basis of these data, benzodiazepines, zolpidem, antidepressants and melatonin (one study) are effective pharmacologic agents. Stimulus control, sleep restriction, relaxation strategies, and cognitive behavioral therapy are effective behavioral interventions for short term management.

Treating the cause

When a secondary insomnia has been identified, it is first necessary to treat the cause and find an appropriate treatment for the medical, behavioral, or psychiatric diagnosis. However, multiple causes are the rule and not the exception. Additionally, although it is usually easier to find a precipitating factor and treat it in acute insomnia, it is more difficult to identify precipitating and perpetuating factors in chronic insomnia.

Educational and behavioral management

Educating patients is essential to reduce insomnia

Sleep hygiene consists of avoiding caffeine and stimulants and adopting regular sleep schedules. People should get out of bed at the same time regardless of how much they slept during the night, and those who have insomnia should avoid daytime napping. However, there might be circumstances when a well-timed short nap can restore daytime alertness and be beneficial for performance. Exercise during the day is recommended, but not too late in the evening. Particular attention should be given to stress reduction.

Stimulus control consists of limiting the time in bed and using the bedroom only for sleep and sexual relations. Patients should avoid reading or watching television in bed. When patients are unable to sleep for 15 or 20 minutes, they should get out of bed and go to another room. They can read, but should avoid bright light and watching television and should return to bed when sleepy. The goal of this method is to reestablish a link between the bed and sleeping [50].

Relaxation therapy has been used extensively in insomnia. It consists of using deep breathing and muscular relaxation. Several methods of approach have been used, although biofeedback is the most common.

Sleep restriction consists of curtailing the amount of time in bed to the evaluated time of sleeping assessed by the patient. This method produces a slight sleep debt that increases the patient's ability to fall asleep and sleep efficiency [51]. Then, the patient adds approximately 15 minutes in bed per day every week, as long as at least 85% of the time in bed is spent sleeping. This method should not be applied before important tests or competitions, or on a long-term basis.

Cognitive behavioral treatment (CBT) of insomnia, which focuses on psychologic and behavioral factors that play a role in maintaining sleep-related problems, has been shown to be of value in producing long-term benefits in primary insomniacs [52]. This treatment may represent an alternative to pharmacotherapy or be complementary during discontinuation of hypnotic medication. The CBT of insomnia may include different components, such as stimulus control, sleep restriction, relaxation, cognitive restructuring, and sleep hygiene [53]. For people who are dependent on benzodiazepines or other hypnotic medication, a supervised tapering based on attaining successive objectives is generally added to the CBT of insomnia. CBT has been used successfully in individual and group approaches. In a recent study, 45 adults suffering from primary insomnia received CBT implemented either in a group therapy format, in individual face-to-face therapy, or through individual telephone consultations. The results indicate that CBT was effective in improving sleep parameters with all three methods of treatment implementation, and there was no significant difference across methods of implementation. All three treatment modalities produced improvements in sleep that were maintained for 6 months after treatment completion [54]. These results suggest that group therapy and telephone consultations represent cost-effective alternatives to individual therapy for the management of insomnia.

The use of bright light may also be an effective treatment for insomnia resulting from circadian rhythm sleep disorders, as reported by Postolache and Oren elsewhere in this issue.

Pharmacologic treatment of insomnia

Hypnotics have a central role in the treatment of insomnia. In the western countries, approzimately 20% of insomniacs and 40% of severe insomniacs are occasionally or regularly taking sleeping pills. The efficacy of the most commonly prescribed treatments in improving sleep latency and continuity has been shown by meta-analyses [6–55]. However, there is a public health concern about the potential abuse and misusage of hypnotics [4]. Thus, the pharmacologic approach of insomnia is generally framed by several consensual recommendations.

General recommendations on the prescription of hypnotics

1) Generally, prescribe hypnotics for short-term use (no more than 3 or 4 weeks). There are very few data assessing the efficacy of these treatments on a long-term pattern.
2) Inform patients about the recommended medication (and alternatives) and the duration of the treatment, and urge them to follow sleep hygiene techniques associated with the treatment.
3) Start low; the lowest dose should be tried first.
4) Intermittent dosing (several nights a week) has proven efficient for some treatments [56].
5) Discontinuation should be made gradually to avoid rebound insomnia.
6) Adverse effects on short-term memory and alertness should be considered before prescribing, and then must be monitored thereafter.

The hypnotics used in insomnia

The most commonly prescribed treatments for insomnia are γ-aminobutyric acid (GABA) agonists; specifically, receptor-selective-agonists such as zolpidem and zopiclone. These agents are selective compounds that interact preferentially with omega 1 receptors to produce sedative effects, whereas benzodiazepines interact with omega 2 receptors and produce adverse effects on cognitive performance and memory.

Zolpidem is a receptor-selective imidazopyridine GABA-agonist hypnotic with a short-term half-life of about 2.6 hours. The efficacy of zolpidem on sleep latency and sleep efficiency was assessed in 1997 by a meta-analysis on 16,944 subjects, and adverse effects were reported by only 1.1% [55]. Zolpidem has also been shown to be an effective treatment for insomnia when used intermittently (ie, on an as-needed basis) with no tendency to increase the dose prescribed [56].

Zopiclone is a cyclopyrolone short-acting GABA-specific agent with an elimination half-life of approximately 5 hours. Zopiclone has been shown to be efficient in short-term insomniacs (28 days) compared with triazolam, flunitrazepam, and placebo [57]. Sleep latency decreased and TST increased by 20% with zopiclone. There was also a decrease in the number of awakenings, a feeling of being more refreshed in the morning, and less daytime impairment. A long-term (8 weeks) study has also shown a good efficiency and lack of rebound after withdrawal [58]. Zaleplon is an ultrashort-acting agent (elimination half-life of 1.2 hours) and a pyrazolopyrimidine GABA receptor-selective agonist that is used in patients who experience nocturnal awakenings.

Older and less selective classic BZDs (eg, triazolam, flunitrazepam, lormetazepam, temazepam) are still used in many countries to treat insomnia. The efficacy of BZD on reducing night awakenings and inducing sleep has been repeatedly demonstrated. However, BZDs have side effects, including daytime sedating (depending of the half-life), alteration of short- and long-term memories, cognitive impairments, rebound insomnia, and dependence [3–7].

Other agents

Certain antidepressants, such as trazodone, nefazodone, mirtazipine, and paroxetine, decrease sleep disturbances associated with depression but they have not been tested for treatment of insomnia. Trazodone is often used for sleep independent of a depression diagnosis. The aim of antidepressant treatment is to avoid the side effects of BZD, including dependence in cases of continuous treatment for chronic insomniacs, and to try to prevent the appearance of depression. Antidepressants are prescribed at lower doses for insomnia than for depression. However, no controlled clinical trial could be found that assesses the efficacy of this method for use in insomnia. The longer half-life of these medications results in daytime sleepiness.

Melatonin is a hormone secreted at night by the pineal gland that can shift the circadian pacemaker in an opposite direction from light. In clinical trials, the indication of melatonin is more focused on circadian disorders (eg, caused by shift work, jet lag, phase delay) [59]. However, a recent study of 517 insomniacs showed that low nocturnal melatonin production is associated with insomnia in patients older than 55 years and identifies patients who are somewhat more likely to respond to melatonin replacement at the dose of 2 mg [60]. Q15

Sedating antihistamines (eg, diphenhydramine) are the main components in most over-the-counter sleeping pills. There are few studies of their effect on sleep architecture. Patients usually describe a better sleep continuity, but a residual sedation effect seems to be a common side effect.

How to use hypnotics in athletes who experience insomnia

If an athlete complains of regular insomnia before competition, a treatment should be tried days before competition, rather than the night before an important event. Athletes who have transient insomnia should be treated with zolpidem or zopiclone at the first raw. Zopiclone is preferred if a consolidated night of 6 to 8 hours of sleep sis required, and zolpidem preferred if there is difficulty inducing sleep or a need for only 5 to 6 hours of sleep. Zaleplon may be indicated in cases involving night awakenings where there is difficulty going back to sleep. Stress management and sleep hygiene should be strongly recommended and explained. Q16

Summary

Managing insomnia in athletes is not an easy task. As sleep is essential for a good performance, experiencing insomnia may affect an athlete's subjective feeling before competitions. Although pharmacologic management of insomnia is not the ideal solution, it is sometimes necessary. It is often difficult to apply nonpharmacologic recommendations in the context of competition, especially on a short-term basis. However, our experience indicates that sleep hygiene, sleep education, and relaxation may greatly help athletes, not only before competitions but also during the regular training periods. Sport physicians must interview young athletes about their sleep patterns to detect subjects who are vulnerable to

insomnia [11], and should consult a specialist when insomnia persistently interferes with competition or training. This will help prevent episodes of insomnia during acute stress situations.

References

[1] Ohayon MM. Epidemiology of insomnia: what we know and what we still need to learn. Sleep Med Rev 2002;6:97–111.

[2] Leger D, Guilleminault C, Dreyfus JP, et al. Prevalence of insomnia in a survey of 12,778 adults in France. J Sleep Res 2000;9:35–42.

[3] Roth T, Hajak G, Ustun TB. Consensus for the pharmacological management of insomnia in the new millennium. Int J Clin Pract 2001;55:42–52.

[4] World Health Organization. Insomnia: report of an international consensus conference. Versailles, Paris: October 13–15, 1996. Geneva, Division of Mental Health and Prevention of Substance Abuse, WHO, 1998. Publication MSA/MND/98.2.

[5] Sateia MJ, Doghramji K, Hauri PJ, et al. Evaluation of chronic insomnia. An American Academy of Sleep Medicine review. Sleep 2000;23:243–308.

[6] Kupfer DJ, Reynolds 3rd CF. Management of insomnia. N Engl J Med 1997;336:341–6.

[7] Schenck CH, Mahowald MW, Sack RL. Assessment and management of insomnia. JAMA 2003;19:2475–9.

[8] American Psychiatric Association. Diagnostic and statistical manual of mental disorders. 4th edition. Washington, DC: American Psychiatric Association; 1994.

[9] Diagnostic Classification Steering Committee. International classification of sleep disorders: diagnostic and coding manual. Rochester, MN: American Sleep Disorders Association; 1990.

[10] Morin CM, Rodrigue S, Ivers H. Role of stress, arousal, and coping skills in primary insomnia. Psychosom Med 2003;65:259–67.

[11] Drake C, Richardson G, Roehrs T, et al. Vulnerability to stress-related sleep disturbance and hyperarousal. Sleep 2004;27:285–91.

[12] Ohayon MM, Caulet M, Priest RG, et al. DSM-IV and ICSD-90 insomnia symptoms and sleep dissatisfaction. Br J Psychiatry 1997;171:382–8.

[13] Hohagen F, Rink K, Kappler C, et al. Prevalence and treatment of insomnia in general practice. A longitudinal study. Eur Arch Psychiatry Clin Neurosci 1993;242:329–36.

[14] Pilowski I, Crettenden I, Townley M. Sleep disturbance in pain clinic patients. Pain 1985;23:27–33.

[15] Shoenut JP, Yamashiro Y, Orr WC, et al. Effect of severe gastroesophageal reflux on sleep stage in patients with aperistaltic esophagus. Dig Dis Sci 1996;41:372–6.

[16] World Health Organization Regional Office for Europe. European technical meeting on sleep on health; January 22–24, 2004; Bonn, Germany.

[17] Edinger JD, Stout AL, Hoelscher TJ. Cluster analysis of insomniacs' MMPI profiles: relation of subtypes to sleep history and treatment outcome. Psychosom Med 1988;50:77–87.

[18] Hohagen F, Kappler C, Schramm E, et al. Sleep onset insomnia, sleep maintaining insomnia and insomnia with early morning awakening–temporal stability of subtypes in a longitudinal study on general practice attenders. Sleep 1994;17:551–4.

[19] Katz DA, McHorney CA. Clinical correlates of insomnia in patients with chronic illness. Arch Intern Med 1998;158:1099–107.

[20] Leger D, Guilleminault C, Bader G, et al. Medical and socio-professional impact of insomnia. Sleep 2002;25:625–9.

[21] Leger D, Scheuermaier K, Philip P, et al. SF-36: evaluation of quality of life in severe and mild insomniacs compared with good sleepers. Psychosom Med 2001;63:49–55.

[22] Brooks 3rd JO, Friedman L, Bliwise DL, et al. Use of the wrist actigraph to study insomnia in older adults. Sleep 1993;16:151–5.

[23] Krahn LE, Lin SC, Wisbey J, et al. Assessing sleep in psychiatric inpatients: nurse and patient reports versus wrist actigraphy. Ann Clin Psychiatry 1997;9:203–10.
[24] Buysse DJ, Reynolds CF, Monk TH, et al. The Pittsburgh Sleep Quality Index: a new instrument for psychiatric practice and research. Psychiatry Res 1989;28:193–213.
[25] Buysse DJ, Reynolds CF, Monk TH, et al. Quantification of subjective sleep quality in healthy elderly men and women using the Pittsburgh Sleep Quality Index (PSQI). Sleep 1991;14:331–8.
[26] Singh NA, Clements KM, Fiatarone MA. A randomized controlled trial of the effect of exercise on sleep. Sleep 1997;20:95–101.
[27] King AC, Oman RF, Brassington GS, et al. Moderate-intensity exercise and self-rated quality of sleep in older adults. A randomized controlled trial. JAMA 1997;277:32–7.
[28] Spitzer RL, Williams JB, Kroenke K, et al. Utility of a new procedure for diagnosing mental disorders in primary care. The PRIME-MD 1000 study. JAMA 1994;272:1749–56.
[29] Bonnet MH, Arand DL. Physiological activation in patients with Sleep State Misperception. Psychosom Med 1997;59:533–40.
[30] Piccione P, Tallarigo R, Zorick F, et al. Personality differences between insomniac and non-insomniac psychiatry outpatients. J Clin Psychiatry 1981;42:261–3.
[31] Stepanski EJ, Koshorek G, Zorick F, et al. Characteristics of individuals who do or do not seek treatment for chronic insomnia. Psychosomatics 1989;30:421–7.
[32] Practice parameters for the use of actigraphy in the clinical assessment of sleep disorders. American Sleep Disorders Association. Sleep 1995;18:285–7. Q17
[33] Elbaz M, Roue GM, Lofaso F, et al. Utility of actigraphy in the diagnosis of obstructive sleep apnea. Sleep 2002;25(5):527–31.
[34] Vallieres A, Morin CM. Actigraphy in the assessment of insomnia. Sleep 2003;26:902–6.
[35] Edinger JD, Hoelscher TJ, Webb MD, et al. Polysomnographic assessment of DIMS: empirical evaluation of its diagnostic value. Sleep 1989;12:315–22.
[36] Edinger JD, Marsh GR, McCall WV, et al. Sleep variability across consecutive nights of home monitoring in older mixed DIMS patients. Sleep 1991;14:13–7.
[37] Wauquier A, van Sweden B, Kerkhof GA, et al. Ambulatory first night sleep effect recording in the elderly. Behav Brain Res 1991;42:7–11.
[38] Mamelak A, Hobson JA. Nightcap: a home-based sleep monitoring system. Sleep 1989;12:157–66.
[39] Partinen M, Putkonen PT, Kaprio J, et al. Sleep disorders in relation to coronary heart disease. Acta Med Scand Suppl 1982;660:69–83.
[40] Bonnet MH, Arand DL. Heart rate variability in insomniacs and matched normal sleepers. Psychosom Med 1998;60:610–5.
[41] Riedel BW, Lichstein KL. Insomnia and daytime functioning. Sleep Med Rev 2000;4:277–98.
[42] Stepanski E, Zorick F, Roehrs T, et al. Daytime alertness in patients with chronic insomnia compared with asymptomatic control subjects. Sleep 1988;11:54–60.
[43] Bonnet MH, Arand DL. Activity, arousal, and the MSLT in patients with insomnia. Sleep 2000;23:205–12.
[44] Mougin F, Simon-Rigaud ML, Davenne D, et al. Effects of sleep disturbances on subsequent physical performance. Eur J Appl Physiol Occup Physiol 1991;63:77–82. Q18
[45] Meney I, Waterhouse J, Atkinson G, et al. The effect of one night's sleep deprivation on temperature, mood, and physical performance in subjects with different amounts of habitual physical activity. Chronobiol Int 1998;15:349–63.
[46] Souissi N, Sesboue B, Gauthier A, et al. Effects of one night's sleep deprivation on anaerobic performance the following day. Eur J Appl Physiol 2003;89:359–66.
[47] Souissi N, Gauthier A, Sesboue B, et al. Circadian rhythms in two types of anaerobic cycle leg exercise: force-velocity and 30-s Wingate tests. Int J Sports Med 2004;25:14–9.
[48] Fulda S, Schulz H. Cognitive dysfunction in sleep disorders. Sleep Med Rev 2001;5:423–45.
[49] Bonnet MH, Arand DL. The consequences of a week of insomnia. Sleep 1996;19:453–61.
[50] Hauri P. Treating psychophysiologic insomnia with biofeedback. Arch Gen Psychiatry 1981;38:752–8. Q19

603 [51] Spielman AJ, Saskin P, Thorpy MJ. Treatment of chronic insomnia by restriction of time in
604 bed. Sleep 1987;10:45–56.
605 [52] Morin CM, Culbert JP, Schwartz SM. Nonpharmacological interventions for insomnia: a meta-
606 analysis of treatment efficacy. Am J Psychiatry 1994;151:1172–80.
607 [53] Bastien CH, Morin CM, Ouellet MC, et al. Cognitive-behavioral therapy for insomnia:
608 comparison of individual therapy, group therapy, and telephone consultations. J Consult Clin
609 Psychol 2004;72:653–9.
610 [54] Belleville G, Belanger L, Morin CM. Traitement cognitivo-comportemental dans l'insomnie et
611 son utilité dans le sevrage des hypnotiques [Cognitive-behavioral treatment of insomnia and
612 its use during withdrawal of hypnotic medication]. Sante Ment Que 2003:87–101 [in French]. Q20
613 [55] Nowell PD, Mazumdar S, Buysse DJ, et al. Benzodiazepines and zolpidem for chronic insom-
614 nia: a meta-analysis of treatment efficacy. JAMA 1997;278:2170–7.
615 [56] Hajak G, Bandelow B, Zulley J, et al. "As needed" pharmacotherapy combined with stimulus
616 control treatment in chronic insomnia–assessment of a novel intervention strategy in a primary
617 care setting. Ann Clin Psychiatry 2002;14:1–7.
618 [57] Hajak G, Clarenbach P, Fischer W, et al. Zopiclone improves sleep quality and daytime well-
619 being in insomniac patients: comparison with triazolam, flunitrazepam and placebo. Int Clin
620 Psychopharmacol 1994;9(4):251–61.
621 [58] Terzano MG, Rossi M, Palomba V, et al. New drugs for insomnia: comparative tolerability of
622 zopiclone, zolpidem and zaleplon. Drug Saf 2003;26:261–82.
623 [59] Sack RL, Lewy AJ, Hughes RJ. Use of melatonin for sleep and circadian rhythm disorders.
624 Ann Med 1998;30:115–21.
625 [60] Leger D, Laudon M, Zisapel N. Nocturnal 6-sulfatoxymelatonin excretion in insomnia and
626 its relation to the response to melatonin replacement therapy. Am J Med 2004;116:91–5.
627