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Treatment of sleep disorders in adults

Sue Wilson & David Nutt

One-third of our lives is spent asleep, but the reasons why we sleep are not yet fully understood. Sleep is a state of inactivity accompanied by a loss of awareness and a markedly reduced responsiveness to environmental stimuli. When a recording is made of an electroencephalogram (EEG) and other physiological variables such as muscle activity and eye movements during sleep (a technique called polysomnography) a pattern of sleep consisting of five different stages emerges. This pattern varies from person to person, but usually consists of four or five cycles of quiet sleep alternating with paradoxical (active) sleep, with longer periods of paradoxical sleep in the latter half of the night. A representation of these various stages over time is known as a hypnogram, and one of these derived from a normal control subject is shown in Figure 1. The quiet sleep is divided further into four stages, each with a characteristic EEG appearance, with progressive relaxation of the muscles and slower, more regular breathing as the deeper stages are reached. Most sleep in these deeper stages occurs in the first half of the night.

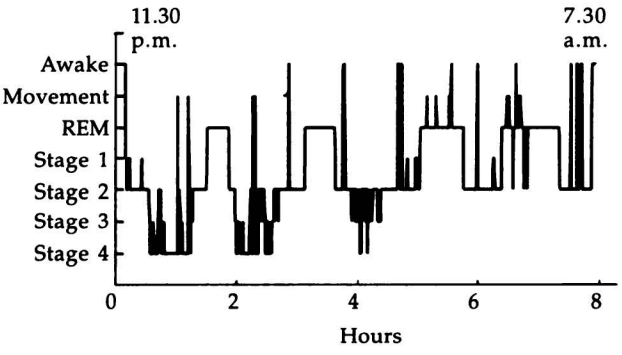

Fig. 1 Hypnogram of a person with a normal sleep pattern (total time sleeping 448 minutes)

During paradoxical sleep, the EEG appearance is similar to that of waking or drowsiness. There is irregular breathing, complete loss of tone of the skeletal muscles and frequent phasic movements, particularly of the eyes, consisting of conjugate movements which are mostly lateral, but can also be vertical. This stage of sleep is called rapid eye movement (REM) sleep and is the stage when most dreaming takes place.

The total length of sleep varies between three and 10 hours in normal people with an average in the 20- to 45-year age group of 7–8 hours. Sleep time is reduced in older people, down to about six hours in those aged over 70 years, with increased daytime napping further reducing the actual time spent asleep at night. The amount of time spent in each of the five stages will vary between subjects, particularly with age, with much less slow-wave sleep in older people. Another sleep variable which increases with age is the number of awakenings after the onset of sleep. Sleep continuity is the term used to describe the maintenance of sleep. Sleep efficiency is the percentage of time spent asleep. Thus:

$$\frac{\text{Total sleep time}}{\text{Total time in bed}} \times 100 = \text{sleep efficiency}$$

Sleep efficiency reflects time spent trying to get off to sleep, waking during the night, and early morning wakening.

As can be seen from Fig. 1, a normal person will have several short awakenings during the night, most of which are not perceived as awakenings unless they last more than about two minutes. Probably there will not be clear consciousness, but there may be an occasional brief thought of how comfortable the person is or how pleased they are that it is not time to get up yet followed by an immediate return to sleep. If during the short period

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of waking there is some factor which causes anger or anxiety such as aircraft noise, partner's snores or a dread of being awake, it is much more likely to cause full awakening and be remembered. The more times this happens, the more subjects complain of an unrefreshing night's sleep. One of the most common ways in which insomnia develops is by 'clock-watching'; sufferers check the time on awakening, remember it, and repeat this cycle many times during the night. Remembering a time of transient awakening reinforces the patient's belief that they sleep poorly and also produces anger and frustration which, in turn, delays their return to sleep and may promote subsequent awakenings.

Diagnosis

In order to aid diagnosis of sleep disorders it is important for the patient to keep a diary of when they slept and how they felt about their sleep. A simple record of retiring and waking times and unusual happenings, with a qualitative visual analogue score for time to fall asleep, amount of intra-sleep awakening and feeling refreshed is very useful for understanding the disorder and monitoring its treatment.

When the symptoms are fully assessed it may be that objective recording of sleep is necessary to differentiate between disorders occurring in different sleep stages. A summary of sleep disturbances is shown in Table 1.

Table 1 Types of sleep disorder

Symptom	Disorder
Not enough sleep or sleep of poor quality	Insomnia
Too much sleep (i.e. in daytime as well)	Hypersomnia or excessive daytime sleepiness *Narcolepsy* *Respiratory, e.g. obstructive sleep apnoea syndrome*
Sleeping at wrong time (i.e. in day instead of night)	Sleep–wake cycle disturbance
Unusual happenings in the night (may not subjectively disrupt sleep)	Parasomnias *Night terrors* *Sleep-walking* *Nightmares* *REM behaviour disorder* Sleep paralysis Periodic leg movements Panic attacks

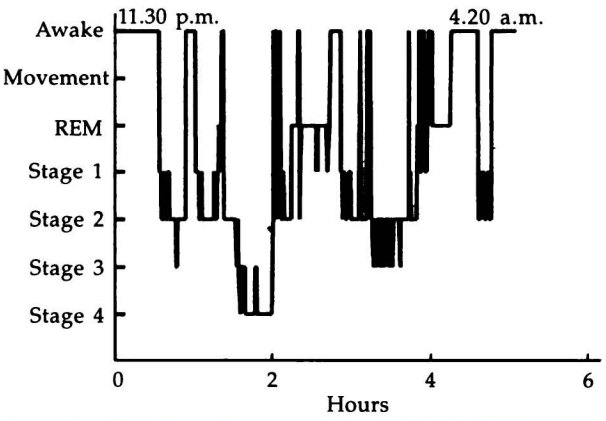

Fig. 2 Hypnogram of a person with psychophysiological insomnia (total time sleeping 220 minutes)

Insomnia

People with insomnia complain of poor sleep, and sufferers report that the duration or subjective quality of sleep is unsatisfactory. Insomnia may consist of difficulty in falling asleep at night (initial insomnia) or difficulty remaining asleep (maintenance insomnia) with either frequent awakenings or waking too early in the morning. Many patients have all of these symptoms and may report that their sleep does not refresh them.

Insomnia may be accompanied by daytime fatigue, but is not usually accompanied by sleepiness during the day. The feeling of constant tiredness and poor performance is experienced by many patients with insomnia. As a result their poor sleep is a major health problem which has a considerable effect on their quality of life.

Figure 2 shows the hypnogram of a typical patient from our clinic with severe insomnia of all three types. There is a lengthened interval before sleep is initiated, more awakenings than usual and early morning awakening without return to sleep. However, this pattern is not always present. Often the patient will misperceive their sleep, and when it is recorded objectively sleep may be of normal appearance and length, with a reasonable number of night-time awakenings. In these cases it seems that there is abnormal awareness of these brief intra-sleep awakenings and lack of awareness of having slept.

Insomnia may be classified according to duration: transient (lasting a few days), short-term or long-term (lasting more than a few weeks).

Transient insomnia is usually situational, for example when subjects are sleeping in a novel

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environment (e.g. hospital). Stress caused by factors such as examinations, impending surgical operations or situations requiring alertness and readiness, such as being 'on-call', can also cause this transient complaint. Withdrawal from hypnotic or anxiolytic drugs, particularly benzodiazepines may cause 'rebound' insomnia, usually not lasting more than a few days. These are all self-limiting conditions.

Treatment of short-term insomnia

The first and most important step is explanation and reassurance. Patients should be informed that if there is an overwhelming physical need for sleep, this will occur whatever the circumstances, and that serious harm will not occur by them sleeping less than they think they should. This is particularly important where fear of insomnia is a factor. If the patient fears waking up unrefreshed because of poor sleep, this attitude alone may lead to a wakeful night (see Box 1).

The precipitating factors should be removed or ameliorated where possible.

Psychological causes

Psychological causes of insomnia include hyperarousal due to: stress; the need to be vigilant at night because of sick relatives or young children; or being 'on-call'.

These can sometimes be modified by improved coping strategies, but the impact on sleep of substantial stress, such as that caused by a major life event such as bereavement or separation or by a stressful period at work, is difficult to reduce.

Pharmacological causes

These include the use of non-prescription drugs such as caffeine or alcohol. Alcohol reduces the time to onset of sleep, but disrupts sleep later in the night. Regular and excessive consumption disrupts sleep continuity; insomnia is a key feature of alcohol withdrawal. Excessive intake of caffeine and theophylline, either in tea, coffee or cola drinks, also contributes to sleeplessness.

Starting treatment with certain antidepressants, especially selective serotonin reuptake inhibitors (e.g. fluoxetine or fluvoxamine) or monoamine oxidase inhibitors, can cause sleep disruption. This is usually resolved after 3–4 weeks of treatment.

The use of other drugs which increase central noradrenergic and serotonergic activity, including stimulants such as amphetamines, cocaine and methylphenidate and sympathomimetics such as the β-adrenergic agonist salbutamol and associated compounds, can also cause sleep disruption. Additionally, withdrawal from hypnotic drugs can cause problems – these are usually short-lived.

Treatment with beta-blockers sometimes causes disruption of sleep (Betts & Alford, 1983), perhaps because of their serotonergic action. In this case an alternative beta-blocking drug which does not cross the blood–brain barrier so readily should be used.

Physical factors

These are listed in Box 2.

Psychiatric disorders

Patients with depressive illnesses often have difficulty falling asleep at night and complain of restless, disturbed and unrefreshing sleep and early morning waking. When their sleep is analysed by polysomnography, time to sleep onset is indeed prolonged, and there is a tendency for more REM

Box 1. Short-term insomnia – summary of treatment

Education and reassurance
Treat precipitating cause
Establish good sleep hygiene
Consider hypnotic medication

Box 2. Physical factors in short-term insomnia

Pain – in which case adequate analgesia will improve sleep
Pregnancy
Coughing or wheezing – adequate control of asthma with stimulating drugs may paradoxically improve sleep by reducing waking due to coughing
Respiratory and cardiovascular disorders
Need to urinate – this may be affected by timing of diuretic medication
Neurological disorders – for example, stroke, movement disorders
Periodic leg movements of sleep (frequent jerks or twitches during the descent into deeper sleep) – rarely reduce subjective sleep quality (Mendelson, 1996) but are more likely to cause insomnia in the subject's sleeping partner

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sleep to occur in the first part of the night, with reduced deep quiet sleep in the first hour or so after sleep onset, and increased awakenings during the night. They may wake early in the morning and fail to get back to sleep again.

Anxiety disorders may cause patients to complain about their sleep, either because there is a reduction in sleep continuity or because normal periods of nocturnal waking are somehow less well tolerated.

Patients with bipolar disorders in the hypomanic phase sleep less than usual and sometimes changes in sleep pattern can be an early warning that this phase is imminent.

Symptomatic treatment of psychiatric disorders usually improves sleep. Antidepressant drugs cause major changes in the structure of sleep when given to normal subjects and patients with depression, some being relatively sedating and improving continuity of sleep, others causing more awakenings (Gillin *et al*, 1997). However, it is clear that when patients with depression improve in mood they report an improvement in their sleep, whether they are treated pharmacologically or by other methods (Buysse *et al*, 1992; Satterlee & Faries, 1995; Gillin *et al*, 1997). However, objective sleep abnormalities predict a poor response to psychotherapy (Thase *et al*, 1996).

Disruption of circadian rhythm

This can cause insomnia, in that patients cannot sleep when they wish to. Causes of this disruption include shift work, jet-lag and irregular routine. These are described more fully under 'Sleep–wake schedule disorders'.

Once precipitating causes have been dealt with as far as possible, educating the patient in good sleep hygiene measures (see Box 3) is important. The patient should stick to these simple measures for 2–3 weeks, keeping a diary as above, before being seen again.

If these measures are not adequate, then short-term treatment with a hypnotic drug is appropriate (see Box 4). There are safe and effective remedies available. All hypnotic drugs will decrease the time taken to fall asleep, awakenings and Stage 1 sleep. There are also effects on sleep architecture, that is, the amounts and distribution of stages of deeper sleep, which vary according to the drug used. Generally, older drugs have greater effects on sleep architecture and the newer drugs very little effect. Some unwanted effects are shown in Table 2. Because of their relative freedom from unwanted effects, zopiclone or a short-acting benzodiazepine should be used for 1–2 weeks, or if only initial insomnia is the problem, the shorter acting zolpidem may be more appropriate.

Box 3. Sleep hygiene

- Keep regular bedtimes and rising times
- Reduce daytime napping
- Take daytime (but not evening) exercise and exposure to daylight
- Avoid stimulants, alcohol and cigarettes
- Establish bedtime routine ('wind down') – a milk drink may be helpful

Box 4. Drug treatment in insomnia

- Treat precipitating factors first
- Relief of short-term insomnia when precipitating causes cannot be improved
- Prevention of progression to long-term problem by establishing good sleeping habits
- Reduction of vicious cycle of anxiety about sleep itself

Treatment of long-term insomnia

The original precipitating factor is usually a cause mentioned in the short-term section and for various

Table 2 Unwanted effects of hypnotic drugs

Drug	Effect
Chloral hydrate	Dependence Low therapeutic ratio/lethal in overdose Gastric irritation
Barbiturate	Misuse/dependence Low therapeutic ratio/lethal in overdose
Chlormethiazole	Misuse/dependence Low therapeutic ratio/lethal in overdose Nasal irritation
Antihistamines	Long-acting – may cause daytime sedation
Benzodiazepines	Dependence Diversion (temazepam)
Cyclopyrrolones (zopiclone)	Metallic taste experienced by 40–50% of patients
Imidazopyridines (zolpidem)	Possibly too rapid/short effect

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reasons this becomes a long-term problem. Usually poor sleep hygiene is established and maintained and the patient becomes excessively anxious about their sleep, spending much time and effort on 'trying' to sleep. Care should be taken to identify and treat depressive and anxiety disorders in this group of patients.

'Psychophysiological insomnia' has a strong element of conditioning: the whole sleeping environment and preparation for bed becomes associated with poor sleep and subsequent anxiety which, in turn, raises levels of arousal. Patients with psychophysiological insomnia will often sleep better in a strange environment which is not associated in their experience with poor sleep, such as the first night on holiday or in a sleep laboratory (this is the reverse 'first-night effect').

The most contentious area in insomnia treatment is whether it is acceptable to use hypnotic drugs continuously. There is a Government intiative to reduce the prescription of benzodiazepines, including hypnotics, but in practice many patients find it difficult – if not impossible – to stop taking them. When forced to do so by their doctor, many experience severe insomnia with secondary anxiety and depression that can significantly increase the overall burden of health costs. There is little evidence that long-term use of hypnotics causes significant medical problems or that tolerance develops. Patients rarely misuse these drugs, daytime sedation is not a problem with the short-acting hypnotics, and dose escalation (indicative of tolerance) is very rare. Our advice would be that forcing patients to stop taking long-term hypnotics can cause more problems than it solves and the main goal should be to prevent long-term use from the outset.

In patients who wish to stop taking medication and in the group with long-term insomnia who have rebound insomnia on stopping, certain strategies may be useful. One is to use treatment on an intermittent basis, so that the patient knows that he or she is guaranteed a good night once or twice a week with medication. It is sensible to use newer hypnotics such as zopiclone and zolpidem whose pharmacology makes them less likely to cause dependence and withdrawal. Stopping treatment by slow reduction is generally more successful than 'cold turkey'; in fact, the latter should never be used because of the risk of seizures.

One of the main thrusts of treatment for long-term insomnia is to change the patient's behaviour, thoughts and beliefs about their sleep. This may require a structured programme and take some time, but some suggestions for inclusion are shown in Box 5 and the subject is dealt with comprehensively in Espie (1994).

Despite all these approaches there will always be some patients who continue to suffer with insomnia and who only respond to hypnotic drugs. In these cases after judiciously weighing up with the patent the risks and benefits of long-term medication, considering the likelihood of alcohol being used as an alternative, and probably a trial of an antidepressant, it may be appropriate to prescribe a hypnotic drug.

Box 5. Changing behaviour and thoughts about sleep

Behaviour

Simple sleep hygiene plus:

(a) Actively restrict the time spent in bed to the same or less than desired duration of sleep (this maximises sleep efficiency)

(b) Schedule a time in early evening for going over events of the day, planning next day, resolving worries, so that these do not encroach on bedtime

(c) Keep bedroom for sleeping – no food and drink, television, books, put light out straight away

(d) Only go to bed when sleepy – learn to recognise sleepiness

Thoughts

It should be obvious from sleep diaries that disasters do not necessarily follow after a night's poor sleep – emphasise positive achievements

Going to sleep is an effortless process, so trying very hard will not help – in general it makes things worse

Think pleasant thoughts when you lay down and close eyes – e.g. summer's day by a river

If thoughts are racing, use mantra-type techniques for stopping them

Disorders of daytime sleepiness

Narcolepsy

Narcolepsy is a disorder with a prevalence of about 0.05%, with onset from childhood to middle age with a peak in the second decade (Guilleminault, 1994). It is a disorder of REM sleep and genetic factors are strongly involved in its aetiology. Patients with narcolepsy have an overwhelming urge to fall asleep

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several times during the day, not only during sleep-promoting activities, but also at strange times such as while eating or holding a conversation. They also have cataplexy, the sudden loss of muscle tone during strong emotion, for example, laughter or fear. This happens without loss of awareness; it can result in slight stuttering or head-dropping for a second or two, through to falling to the ground with complete lack of movement for up to 30 minutes. As well as daytime sleepiness and cataplexy, hallucinations in drowsiness and sleep paralysis exist (the so-called narcolepsy syndrome tetrad). Treatment is usually carried out in specialist sleep centres where stimulant drugs or modafinil are used to treat the daytime sleepiness, and the cataplexy is often treated with antidepressants such as selective serotonin reuptake inhibitors.

Breathing-related sleep disorders

By far the most common of these is obstructive sleep apnoea syndrome, which is prevalent especially in middle-aged men, particularly those with thick necks, and is strongly associated with loud snoring. The airway tends to narrow in normal people during sleep, and in patients with obstructive sleep apnoea syndrome it totally collapses. These patients are sleepy during the day because they have awoken as many as several hundred times each night when their breathing has become obstructed and oxygen concentration in the blood falls. Often they are unaware of their poor sleep and present to health services after falling asleep while driving or when their sleeping partner complains that they snore very loudly or stop breathing at night. Treatment is usually carried out at a specialist sleep-disordered breathing centre and often involves the administration of continuous positive airway pressure via a mask worn at night.

Atypical depression and chronic fatigue syndrome may also cause excessive sleepiness in the daytime.

Sleep–wake schedule disorders

The time of day at which the usual long period of sleep takes place seems to be determined by an internal clock, influenced by the light cues of day and night. When these cues are missing, humans tend to maintain an approximately 25-hour cycle, which helps to explain why it is less disruptive to lengthen our day on an east to west air trip than shorten it when travelling in the other direction. Many other factors, including length of time since the last sleep period, as well as social and environmental factors, determine the tendency to fall asleep. It is when sleep propensity due to these factors does not coincide with the body's innate circadian sleep–wake rhythm that these disorders occur.

Jet-lag

Patients with disturbances in their sleep–wake schedule complain that they sleep during the day and are awake all night. This may have been precipitated by an aeroplane journey over time zones or may date back to a period of night work or other need to be awake at night. Re-establishing a more acceptable sleep–wake rhythm can be achieved by the use of appropriately timed light. Use of the simple sleep hygiene measures, two hours of bright light in the early morning and none in the evening (daylight is best) and rigid adherence to regular routines for eating and exercise should be rapidly effective.

Shift work

Shift workers have to sleep at different times on different days and often have difficulty getting enough sleep or returning to a normal routine on days off. There are ways in which a routine more conducive to sleep may be arranged:

(a) Improved regularity of shift changes, for example, equal number of days on each schedule.
(b) Shifts should rotate clockwise, as lengthening the time between successive sleep periods is easier than shortening them. The best rotation is morning shift, afternoon shift, night shift followed by at least two days off (Monk, 1994).
(c) Dividing sleep so that there is a long main sleep period and a short nap before work (Gillberg *et al*, 1996).

Other sleep–wake schedule disorders

Some patients with irregular routine, or disorders in which light or environmental cues are reduced, such as schizophrenia, learning disabilities or visual impairment, have difficulty sleeping at night. For treating this group a methodical systematic shift of sleeping time over a long period used to be employed, by gradually going to bed and being woken 30 minutes later each day until the optimum time schedule was reached. More recently the use of bright morning light has proved a quicker and more effective aid to resetting the body clock (Duffy *et al*, 1996), accompanied by a conscious reinforcement of environmental and social cues for waking and sleeping.

Melatonin, the hormone produced by the pineal gland during the hours of darkness, has been used recently to try to reset the circadian clock in subjects with sleep–wake schedule problems such as jet-lag.

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It has proved effective at resetting circadian rhythm in these disorders (Sack *et al*, 1997) usually at a dose of 0.5 mg at the appropriate phase of the cycle, that is, at night to bring on sleep and in the morning to delay it. However, the use of melatonin as a hypnotic agent has been less successful, with contradictory effects (Zhdanova *et al*, 1997).

Parasomnias

Night terrors and sleep-walking

Both of these phenomena arise from slow-wave sleep, and they are often coexistent. There is usually a history dating from childhood, and often a family history. Exacerbations usually coincide with periods of stress, and alcohol will increase the likelihood of them occurring.

During a night terror the patient usually sits or jumps up from deep sleep (most often early in the night) with a loud cry, looks terrified and moves violently, sometimes injuring himself or herself or others. He or she appears to be asleep and uncommunicative, often returning to sleep without being aware of the event. These terrors are thought to be a welling up of anxiety from deep centres in the brain, which is normally inhibited by cortical mechanisms. There have been some reports of successful treatment with benzodiazepines (Schenck & Mahowald, 1996) and more recently paroxetine (Wilson *et al*, 1997).

Nocturnal panic attacks may be distinguished from night terrors by the fact that the patient awakens fully from light sleep before the panic symptoms have reached a peak, and is fully aware of the attack.

Nightmares

These arise out of REM sleep and are reported by the patient as structured, often stereotyped dreams which are very distressing. Usually the patient wakes up fully and remembers the dream. Psychological methods of treatment may be appropriate, such as a programme of rehearsing the dream and inventing different endings. In a small number of cases where adverse events such as angina have been provoked by recurrent nightmares it may be appropriate to consider drug treatment, such as a monoamine oxidase inhibitor which suppresses REM sleep (Akindele *et al*, 1970). Nightmares of a particularly distressing kind are a feature of post-traumatic stress disorder and, although there have been a small number of case reports showing various pharmacological agents to be effective in ameliorating these nightmares, there are no clear indications for any particular drug's efficacy. However, serotonin (5-HT_2) blockers such as trazodone or nefazodone are precribed by many psychiatrists.

Sleep paralysis

This is another phenomenon which arises from REM sleep, and is experienced by the sufferer as a state of being fully conscious and aware without being able to move. It usually only lasts a few seconds, but because of its distressing nature is perceived as lasting longer. It is usually aborted by auditory or somatosensory stimuli. Attacks of sleep paralysis seem to be due to an incomplete arousal from REM sleep, so that consciousness is restored but the atonia of REM persists. It is more likely to occur at times of sleep deprivation, during naps, and is one of the tetrad of symptoms of the narcolepsy syndrome. Reassurance is very important in this disorder as it is extremely frightening; it may be associated with panic disorder (Allen & Nutt, 1993) and can lead to alcohol dependency where alcohol is used to reduce fear and anxiety.

REM behaviour disorder

This was first described by Schenck *et al* (1988) and consists of lack of paralysis during REM sleep, which results in acting-out of dreams, often vigorously with injury to the self or others. It can occur acutely as a result of drug or alcohol withdrawal, but the chronic manifestation can be idiopathic or associated with neurological disorder (about 50% of each). It is much more common among older patients. Successful treatment has been described with clonazepam (Schenck *et al*, 1996), and clonidine at night can also be helpful.

Periodic limb movements of sleep

These are jerking or kicking movements of the limbs, usually the legs, during sleep which can occur in normal subjects, but increase with age. In some people they become excessive and distressing, often to the bed partner more than the subject. They tend to arise from Stage 2 sleep, and there is a strong association between these and the restless leg syndrome in the daytime. Both of these disorders have been described in association with medical disorders such as uraemia, anaemia and heart failure, and there is a marked increase in incidence

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with increasing age. If they are associated with arousals from sleep, or occur more than 25 times per hour then referral to a specialist sleep centre is indicated. Effective treatment with dopaminergic drugs such as carbidopa/levodopa (25/110 or 50/200) has been reported (Kaplan *et al*, 1993).

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Multiple choice questions

1. Regarding drugs and sleep:
   a beta-blockers greatly improve sleep
   b selective serotonin reuptake inhibitors suppress night terrors
   c tricyclic antidepressants are the preferred treatment for REM behaviour disorder
   d salbutamol increases sleep latency
   e dopamine agonists may help periodic limb movement disorder.

2. The following are disorders of REM sleep:
   a night terrors
   b nightmares
   c sleep paralysis
   d periodic leg movements
   e nocturnal panic attacks.

3. Causes of daytime sleepiness are:
   a insomnia
   b narcolepsy
   c obstructive sleep apnoea syndrome
   d tricyclic antidepressants
   e nightmares.

4. Key elements in the management of short-term insomnia are:
   a education
   b good sleep hygiene
   c treatment of underlying cause
   d full blood work up
   e family therapy.

5. The following are true of hypnotic drugs:
   a new hypnotics (zopiclone and zolpidem) cause less rebound
   b zopiclone has a shorter half-life than zolpidem
   c the half-life of temazepam can extend to greater than 10 hours in some people
   d hypnotics induce slow-wave sleep
   e chlormethiazole is safer than benzodiazepines in overdose.

MCQ answers

1	2	3	4	5
a F	a F	a F	a T	a T
b T	b T	b T	b T	b F
c F	c T	c T	c T	c T
d T	d F	d T	d F	d F
e T	e F	e F	e F	e F

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