

The Neurological Sleep Clinic – Part 2

Insomnia and parasomnias

Virtually everyone has experienced short-term insomnia from time to time, usually in the context of some stressor or the anticipation of an exciting event. However, chronic insomnia is also very common, affecting up to 10% of the population. It is rather blandly defined as the perception of inadequate or insufficient sleep for a period of three weeks or more, with most insomniacs having a history that dates back for many years. Most commonly, the problem is one of both sleep onset and subsequent sleep maintenance although some have only one or other of these elements. In typical chronic or so-called ‘psycho-physiological insomnia’, a trigger or adverse life event can usually be identified at the start of symptoms. Subsequent insomnia and concerns over poor sleep seem to fuel further symptoms although it is usually presumed there is also some ill defined constitutional predisposition or central ‘wiring problem’ combined with varying degrees of maladaptive habits developed by the sufferer. Examples of the latter include frequent checking of the clock through the night or using the bedroom for activities other than sleep. Although psychological or even psychiatric factors are clearly important in most forms of insomnia, additional elements of more interest to neurologists are often relevant as will be discussed.

Parasomnias, literally ‘events during sleep’, can almost be considered normal in children. However, abnormal behaviours arising from sleep, invariably with reduced awareness, are not uncommon in adults, usually in those with a childhood background of sleep-walking. It can be important to diagnose these sleep-related phenomena confidently from history alone, especially since tests are rarely helpful. A mis-diagnosis of nocturnal epilepsy is not rare and can lead to unnecessary treatment and restrictions.

Insomnia

At best, the majority of UK sleep centres deal with insomniacs poorly. At worst, they refuse even to see them. This is mostly because the best recognised treatment for primary insomnia, certainly that occurring at sleep onset, is cognitive behaviour therapy for which it is extremely difficult to find interested practitioners with expertise, at least in the NHS. However, not infrequently, secondary causes of insomnia can be recognised and successfully treated with relative ease. An algorithm is shown in Figure 1.

If symptoms are not volunteered, RLS can be missed as a relatively common and treatable trigger for sleep onset insomnia or, indeed, poor sleep maintenance. Associated periodic limb movements during sleep may also be worth treating even if the diagnosis is not expected from a bed partner’s history and movements are subsequently picked up with overnight recording. In addition, it can be appropriate to address pain or discomfort arising from musculoskeletal disorders including fibromyalgia and other general medical conditions such as reflux oesophagitis which can act as a significant ‘hypnotoxin’.

Another category of sleep disorder that merits addressing as an explanation for some forms of insomnia is delayed sleep phase syndrome (DSPS), especially in young populations. In this under-recognised phenomenon, a subject’s internal ‘clock’ appears to run a few hours behind the average, making it difficult to settle before 2am and very difficult to wake up before, say, 10am. This latter feature is very unusual in simple chronic insomnia. The genetics of DSPS are an active area of research and many such subjects may have specific polymorphisms or mutations of genes

intimately involved in central clock mechanisms.

Some authorities are enthusiastic about the use of diaries or wrist actigraphy in the assessment of insomnia. Although the former may give valuable insight into an individual’s habits, some of which may be maladaptive, the latter is only rarely helpful in documenting the severity of insomnia. Since it is only a surrogate measure of actual sleep, if a subject remains completely still although awake, misleading information may be obtained.

A number of neurological conditions, both common and rare, may have insomnia as a prominent disabling symptom, assuming it is picked up from the history. Somnolent parkinsonian patients frequently have fragmented overnight sleep with early wakening as key elements of their disturbed sleep-wake cycle. This presumably directly reflects brainstem pathology although drugs and mood disorder may be additional factors. The ultimate rare neurodegenerative cause of insomnia, namely fatal familial insomnia, probably reflects the result or relatively specific thalamic dysfunction caused by prion protein accumulation.

Several rare autoimmune or paraneoplastic syndromes such as limbic encephalitis may also produce severe insomnia with or without hallucinatory intrusions as part of the clinical spectrum. Indeed, a good sleep history is often a useful diagnostic marker in such conditions.

Parasomnias

Parasomnias are usually classified according to the sleep stage from which they arise and are broadly divided into REM and non-REM types. The latter are extremely common in children and form a spectrum of night terrors, confusional arousals and actual sleepwalking. Events occur when a subject arouses abnormally and incompletely from deep non-REM sleep usually within 90 minutes of sleep onset. It is not uncommon for such phenomena to persist into adulthood in which case their nature may change. Complex behaviours such as cooking or even driving are well described and violent parasomnias are increasingly seen in medico-legal contexts. If parasomnias start to occur in adults with a distant childhood history of



Dr Paul Reading, MA, FRCP, PhD, is a consultant neurologist based at the James Cook University Hospital, Middlesbrough. He has trained in Cambridge, Edinburgh and Newcastle. Over the last seven years he has developed an academic and clinical interest in sleep medicine and been secretary of the British Sleep Society for four years. Narcolepsy and the sleep disorders associated with neurodegenerative disease are particular areas of interest.

Correspondence to:

Dr Paul Reading,
Department of Neurology,
The James Cook University
Hospital,
Middlesbrough, TS4 3BW, UK.
Email: Paul.Reading@tees.nhs.uk

Figure 1: Algorithm for assessing subjects with insomnia.
RLS – restless legs syndrome;
OSA – obstructive sleep apnoea

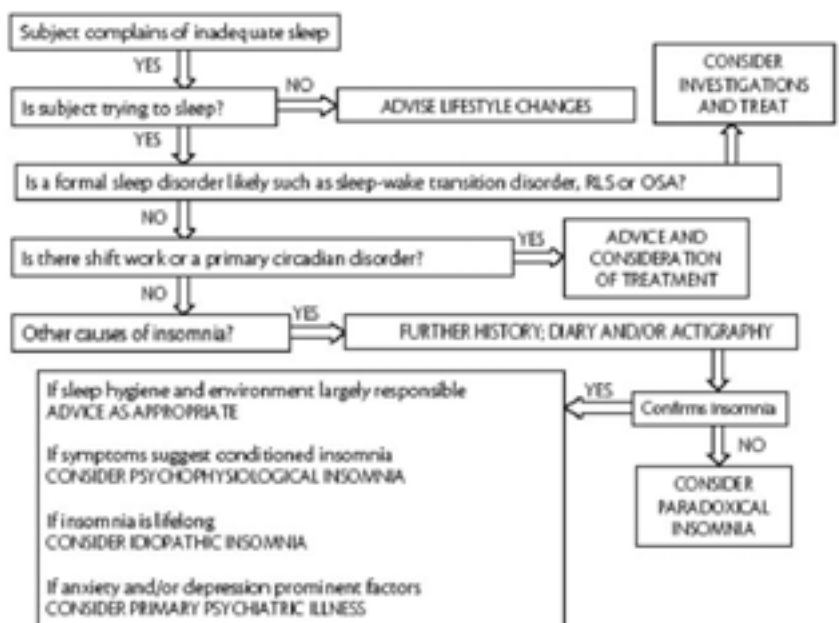


Table 1: A summary of some key differences between non-rapid eye movement (non-REM) parasomnias and nocturnal epilepsy.

	Non-rapid eye movement sleep arousal disorder	Nocturnal (bilateral lobe) epilepsy
Age at onset	Early childhood	Adolescence or later
Positive family history	30–50%	<4%
Number per month	1–3	Usually > 10
Number per night	1	Usually several
Semiology	Complex and non-stereotyped	Stereotyped
Duration	Minutes	Seconds
Timing	First third of night	Random
Sleep stage	Non-rapid eye movement stage II or IV	Most often stage II
Local EEG	High amplitude delta activity	Epileptic activity rarely seen
Triggers	Commonly identified	Rare
Natural history	Spontaneous remission	Persistent

similar phenomena, it is always worth considering whether another sleep disorder such as sleep apnoea is present and acting as a trigger, potentially fuelling partial arousals from deep sleep. It is fairly common for parasomnias to be confused with nocturnal epilepsy although a good history usually suffices for a confident diagnosis. Some key pointers for distinguishing the two entities are given in Table 1.

Occasionally, there is considerable doubt as to the nature of abnormal nocturnal behaviours and investigations may be considered. Unfortunately, it is relatively rare to capture non-REM parasomnias with overnight monitoring and recording between events is generally normal or non-specific. Providing a patient with video equipment for home monitoring may be cost effective.

In a neurological setting, violent parasom-

nias arising from sleep in the context of REM sleep behaviour disorder are not uncommon. Most subjects are elderly males, often with a parkinsonian syndrome already present or in sub-clinical evolution. Injuries, especially to bed partners, may be significant and more than justify long term treatment. The diagnosis is usually clear from history alone. Typically the subject lashes out in deep sleep with brief upper limb movements and vocalisations. The eyes are usually closed and complex behaviours including mobilisation are rare. Dream recall is the norm and subjects usually describe defensive manoeuvres as an explanation for the violent behaviour. Since REM sleep occurs at intervals through the night, events may also recur with a particularly high incidence at around 4am. If overnight polysomnography is performed, the loss of the normal muscle atonia

seen in REM sleep is diagnostic and is present even in the absence of frank movements. Long term treatment with clonazepam is usually successful with melatonin gaining a reputation as a useful second line agent.

Conclusion

In summary, as one commentator famously noted: "sleep is by the brain and for the brain". Although many fundamental questions about sleep remain unanswered, it is crucial for normal brain function and, indeed, mental health. With a broad knowledge of what can go wrong with sleep, both in normal subjects and those with neurological problems, a confident diagnosis to explain sleep-related symptoms can usually be made without recourse to sophisticated tests. Investigations and, more importantly, their proper interpretation, have an important role, however, and it is strongly hoped that neurologists will have an increasing profile in sleep medicine, a discipline that deserves and needs their attention.

Key references for further reading

- American Academy of Sleep Medicine. The International Classification of Sleep Disorders, 2nd Edition. AASM. Rochester 2005.
- Espie C. Overcoming Insomnia and Sleep Problems. London: Constable and Robinson, 2006.
- Morin C, Hauri PJ, Espie C, et al. Non-pharmacologic treatment of chronic insomnia. An American Academy of Sleep Medicine review. Sleep 1999;22:1134–56.
- Reading PJ. Parasomnias: the spectrum of things that go bump in the night. Pract Neurol 2007;7:6–15.

Read ACNR free on-line

You can read every issue of ACNR free of charge by downloading PDFs from our website at www.acnr.com

Simply register by sending an email to Rachael@acnr.co.uk, and we will notify you every two months when new issues are uploaded to the site.

*Do you have an idea for an article?
Are you organising an event,
can we help with publicity?
Would you like to review a book,
perhaps you're writing one?*

Please contact

Patricia McDonnell
ACNR
(Advances in Clinical Neuroscience & Rehabilitation)

T/F. +44 (0)288 289 7023
E. Sales@acnr.co.uk

www.acnr.com

