

# 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



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



