Understanding insomnia

■ Anxiety and depression are common causes of insomnia, but they are not the only causes. Neurological disorders such as restless legs syndrome and periodic limb movement disorder are also common. A conceptual model of sleep and wakefulness helps to explain different kinds of insomnia.

Murray Johns

Murray Johns, MB, BS, BSc, PhD, is a consultant in sleep disorders medicine and Director, Sleep Disorders Unit, Epworth Hospital, Richmond, Victoria.

Our knowledge of sleep and its disorders has increased rapidly in recent years, particularly in relation to sleep–related breathing disorders such as obstructive sleep apnoea.[1] To some extent, the developments with sleep apnoea may have overshadowed other advances in relation to insomnia.

Insomnia is a symptom that depends on the subject's perception of difficulty initiating or maintaining sleep, obtaining too little sleep, waking too early or having non restful sleep. Thus, it is highly subjective, depending on the subject's expectations of sleep and wakefulness, and sometimes on the feeling of having too much wakefulness which, if associated with distress, the subject may prefer to avoid by sleeping more.

There are several ways of classifying the disorders that are associated with insomnia, including a new classification published by the American Sleep Disorders Association in 1990.[2] In the author's view, a new pathophysiological concept of sleep and wakefulness, described in more detail elsewhere,[3] is useful in understanding insomnia.

A new conceptual model of sleep and wakefulness

Whether we are awake or asleep at any particular time depends on the relative strengths (not the absolute strengths) of two competing drives — the drives for sleep and for wakefulness (*Figure 1*). Each of these drives is generated by the integrated activity of different groups of neurons in various parts of the central nervous system, not from a single sleep centre or wake centre.[4]

The sleep and wake drives each have two components — the primary and secondary drives for sleep and for wakefulness. The total wake drive (the sum of the primary and secondary wake drives) and the total sleep drive interact continuously by mutual inhibition. The variables Is and Iw (*Figure 1*) reflect the intensity of this mutual inhibition at any particular time.

Primary sleep and wake drives

The primary drives for sleep and for wakefulness vary with the time of day (*Figure 2*). The phase of these circadian rhythms is set by interaction with the environment; for example, the timing of exposure to sunlight and darkness that influences the suprachiasmatic nucleus of the hypothalamus.[5] These drives also vary with ultradian rhythms (periods between 1 and 12 hours), but these will be omitted from further discussion here for simplicity.

The circadian rhythm of the primary wake drive usually reaches its peak at about 1800 to 1900 hours and its trough at about 0400 to 0500 hours (*Figure 2*). This is reflected in body temperature, melatonin secretion from the pineal gland, and the occurrence of rapid eye movement (REM) sleep.

By contrast, the circadian rhythm of the primary sleep drive reaches its peak only a few hours later than that of the wake drive, at 2300 to 2400 hours, and its

trough at 0900 to 1000 hours (*Figure 2*). This is reflected in the circadian rhythm of the occurrence of delta–wave sleep (stages 3 and 4).[6]

Secondary sleep and wake drives

The secondary component of the sleep drive increases progressively during wakefulness and may be influenced by physical exertion. Its effects are dissipated progressively during non–REM sleep as part of sleep's restorative function.

The secondary sleep drive is maximal at the end of the day and its effects are most obvious during the first period of non–REM sleep when the primary sleep drive is at the peak of its circadian rhythm.

The secondary wake drive provides the key to the door of sleep for most people. This component of the wake drive arises from collateral inputs to the reticular activating system and the non–specific thalamic projection system from all sensory afferent tracts, both exteroceptive and enteroceptive, and from the limbic system and other central neuronal populations concerned with attention, cognition and volition.

The secondary wake drive is rapidly responsive to all changes of posture and physical activity and to behavioural and psychological changes of all kinds. Its importance is highlighted by the self–evident fact that most adults do not fall asleep unless they cease major body movements, lie down, stop talking, close their eyes and relax, physically and mentally — all these manoeuvres reduce the secondary wake drive. This process of falling asleep is termed 'sleepening', in contrast to wakening. The first phase of sleepening is usually under voluntary control. We can stay awake later than usual, voluntarily, despite an increasing secondary sleep drive, by keeping physically and mentally active.

Causes of insomnia

Insomnia can be associated with any disorder that involuntarily causes a relative increase in the total wake drive in relation to the total sleep drive during the intended sleep period.

These changes may be either phasic and repetitive or more continuous. The mechanisms by which various disorders produce insomnia may be classified as follows:

1. an absolute increase in the total wake drive: due to increased primary or secondary wake drive or decreased inhibition of the wake drive by the sleep drive (Iw in *Figure 1*)
2. an absolute decrease in the total sleep drive: due to a decreased primary or secondary sleep drive or increased inhibition of the sleep drive by the wake drive (Is in *Figure 1*)
3. circadian rhythm disorders of the

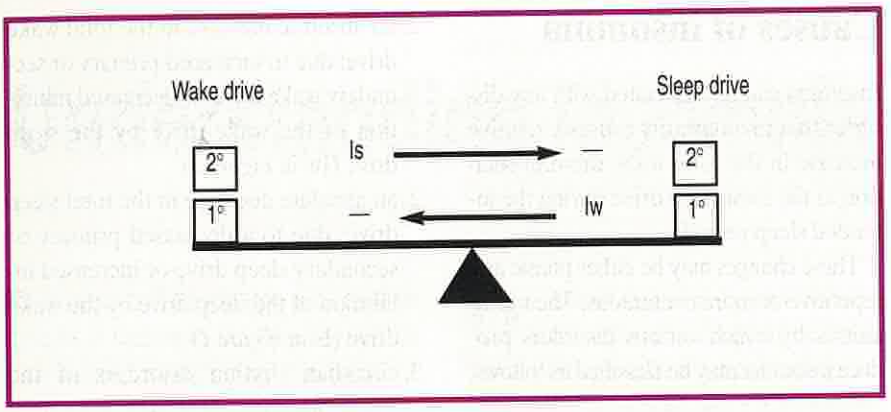

Figure 1. Competing drives for sleep and for wakefulness each have a primary and secondary component and mutually inhibit each other. Their interaction determines the state of sleep or wakefulness at any particular time.

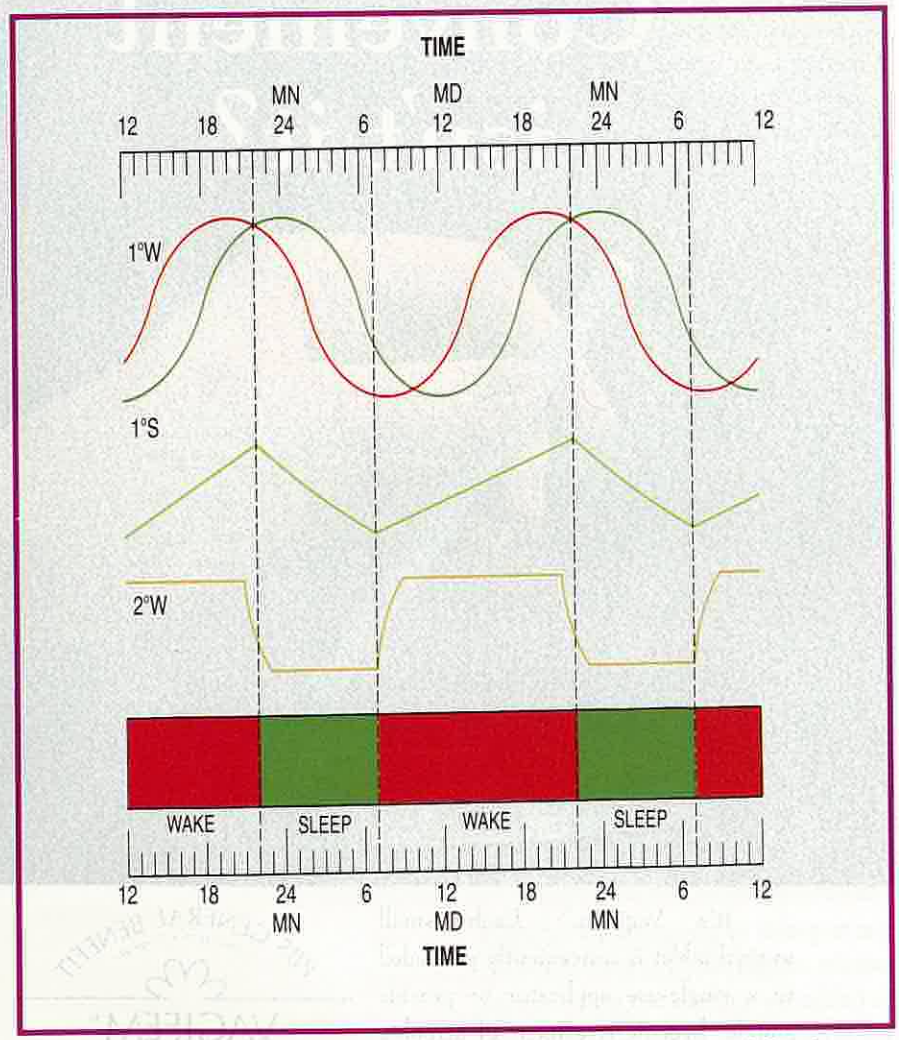

Figure 2. Variations in the primary and secondary components of the sleep and wake drives over a 48–hour period. Ultradian rhythms and the effects of mutual inhibition between the sleep and wake drives have been omitted for simplicity.

sleep and wake drives: due to an inappropriate phase relationship between the circadian rhythms of the sleep and wake drives, cycle lengths much longer or shorter than 24 hours, or abnormal amplitures of one or both circadian rhythms

4. some combination of the above: insomnia in individual patients often seems to involve more than one mechanism.

Clinical types of insomnia

Within this conceptual framework it is possible to explain different types of insomnia that have been recognised clinically. Insomnia with psychiatric disorders that involve affective arousal (anxiety state, mania, schizophrenia, major depressive illness, for instance.) would involve a persistently increased secondary wake drive.

Some patients, however, may also have a decreased total sleep drive manifested as reduced delta–wave sleep. In addition, some may have changes in their circadian rhythms causing early morning wakening and the early onset of REM sleep (within 60 minutes of first going to sleep) which are common with major depressive illness.

Phsychophysiological insomnia is a common form of chronic insomnia that does not involve persistent affective arousal. It usually begins as an adjustment sleep disorder at the time of major stress, such as bereavement, or marriage breakup. The affective arousal caused by this stress settles down after a while as the patient copes. However, an association is formed, consciously or not, between going to bed and not sleeping. The patient begins to fear and be frustrated by insomnia. What used to happen habitually and subconsciously during sleepening is now inhibited by situation–specific cognitive and affective arousal. The harder the patient tries to fall asleep the more the secondary wake drive

is increased. This is sometimes called learnt insomnia.

Idiopathic insomnia begins in childhood and is a lifelong disorder involving difficulty with initiating and maintaining sleep. It seems to be associated with a persistently low sleep drive that may be inherited. It is made worse by an increase in the wake drive such as that which is learnt in psychophysiological insomnia.

Such patients are usually unable to doze during the day, for they are never sleepy but are often fatigued. The increased prevalence of insomnia with age is explained by the well documented decline in delta–wave sleep and hence in sleep drive after early adulthood.

Restless legs syndrome and periodic limb movement disorder are more common causes of insomnia than is currently recognised.

This seems to be due to neuronal degeneration in the central nervous system. There is probably also a decline in the primary wake drive, but this is slower than the decline in the sleep drive. As with idiopathic insomnia, it takes only a small increase in the secondary wake drive in older people to produce insomnia.

Restless legs syndrome is a disorder of sleepening that can be inherited. It usually begins in middle life and can be worsened by pregnancy and iron deficiency anaemia. The patient has a funny feeling or dysaesthesia, typically of the lower legs but sometimes elsewhere, when relaxed and sleepy. Although patients find it hard to describe, some say it is like ants crawling under the skin. It is associated with an urge to move. Any major body movement relieves the symptom temporarily by inhibiting sleepening. However, the feeling may return and prevent sleep onset for prolonged periods.

An associated disorder, periodic limb movement disorder, can also be inherited and involves repetitive twitches of the legs and sometimes of the arms that disturb sleep. This occurs in about 50 per cent of patients with restless legs syndrome, but it can occur independently. These disorders inhibit and fragment sleep much more commonly than is currently recognised. They are found in 15 to 25 per cent of chronic insomniacs when investigated in the sleep laboratory. The pathophysiology of these disorders is unclear, but they may involve failure of sleep–related descending inhibitory mechanisms.

A metabolic disorder such as hyperthyroidism can cause insomnia by both direct and indirect effects on the neuronal systems producing the sleep and wake drives. Hyperthyroidism may increase delta–wave sleep (that is, sleep drive) but also increases the primary and secondary wake drives to produce insomnia.

Delayed sleep phase syndrome can mimic other insomnias by causing great difficulty in falling asleep until very late at night. It differs by way of increased depth of sleep and great difficulty in waking until late next morning. The circadian rhythms of sleep and wakefulness are phase–delayed in the 24 hours. However, there may also be increased mutual inhibition of the sleep and wake drives to explain the unusual difficulty experienced with wakening as well as with sleepening.

Effects of hypnotic drugs and alcohol

Hypno–sedative drugs help the insomniac initially by reducing the secondary wake drive, particularly that part of it associated with anxiety. These drugs may also reduce the sleep drive as an unwanted effect, especially with more prolonged use and higher doses.

The benzodiazepines reduce delta–wave sleep as did the earlier hypnotic drugs such as barbiturates that the benzodiazepines replaced. This explains, at least in part, why most hypnotics become less effective with long–term use. Suddenly ceasing these drugs causes a rebound of the wake drive that makes insomnia temporarily worse and leads many patients to believe that they should continue taking them. Alcohol, too, reduces the wake drive initially and the sleep drive later within the same night.

Hypnotic drugs reduce the wake drive initially but may also reduce the sleep drive later as an unwanted effect.

None of the hypnotics on the market increases the sleep drive as opposed to reducing the wake drive. This may be a new direction for drug research, because some substances (peptides) do increase delta–wave sleep and the sleep drive.

Assessment of insomnia

The first step in the management of insomnia is an accurate assessment of its nature, severity and causes. Only then can appropriate treatment be implemented, sometimes of more than one kind. The mainstay of assessment is a detailed history of sleep habits. The circumstances under which insomnia began are important, particularly in psychophysiological insomnia. A sleep diary kept each day for 2 weeks can be useful for recording the times of sleeping and their variability, which is a feature of many insomniacs.

Restless legs syndrome is diagnosed from the history but, strangely, this may not be volunteered unless the unusual symptoms are specifically sought. Periodic limb movement disorder is seldom diagnosed from history alone and requires overnight polysomnography at a specialist centre, as does sleep apnoea.[7]

However, polysomnography is not required for the management of most cases of insomnia, except where the diagnosis is in doubt or treatment has failed. One reason for such failure can be the coexistence of two or even more kinds of sleep disorder.

Treatment

The treatment of insomnia should be directed initially at its specific causes, when they can be identified (for example, depression, anxiety state, restless legs syndrome, periodic limb movement disorder, inappropriate shift–work rosters, hypnotic/stimulant/alcohol dependence, hyperthyroidism or hypothyroidism).

Hypnotic drugs (which commonly means one of the benzodiazepines) have a role, but this role is diminishing as public attitudes change and as we learn more about sleep disorders. Their long–term use for chronic insomnia should be questioned, if only to be sure there is no better alternative.

Many patients sleep no better after taking benzoidiazepine hypnotics for a few months than they would if they were weaned off them. However, this must be done slowly over 2 to 6 weeks.

The sleep propensity at any particular time depends on the relative strengths of the wake drive and the sleep drive.

Explanation and reassurance can do much to restore a patient's confidence in his or her ability to sleep, but this may not

be sufficient. A behavioural, non–drug treatment programme has been developed and used successfully by Morawetz in Melbourne.[8]

This includes techniques for relaxation, which is necessary but not always sufficient by itself to treat insomnia. Cognitive therapy is another important part of this programme, changing attitudes and behaviour in relation to the time spent in bed awake. It can overcome the learnt component, which is dominant in psychophysiological insomnia, but which is also present in many other types of insomnia. Hypnosis can be useful, too.[9]

These aspects of treatment are aimed primarily at reducing an increased level of secondary wake drive. It may be possible to increase the sleep drive by exercising during the day, but not within a few hours of bedtime because exercise also increases the wake drive initially.

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