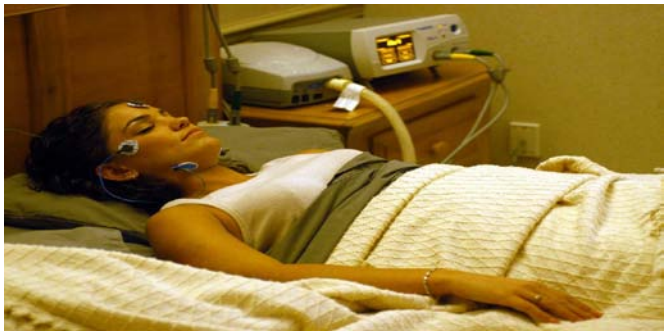
THE NATURE OF SLEEP, INCLUDING STAGES OF SLEEP AND LIFESPAN CHANGES IN SLEEP

As noted previously, **Loomis, Harvey & Hobert (1947)** found that contrary to popular belief, the brain is not uniformly active/inactive over the course of a night's sleep. Instead, there are times when electrical activity is high, and other times when it is low. The activity of the brain is measured in term of the *frequency* of the electrical activity. The four types of brain wave that occur during sleep are called **beta**, **alpha**, **theta**, and **delta**.

As well as using the EEG to measure the electrical activity of the brain, measures can also be taken of how tense the muscles are. This is called an **electromyogram (EMG)**. Measures can also be taken of eye movements during sleep, beneath the closed eye-lids. This is called an **electro-oculogram (EMG)**.Both of these indicate that interesting things go on during sleep.



The EOG is the electrode closest to the eye. The EMG is the electrode on the chin. EEG recordings are taken from the scalp

Rechtschaffen & Kales (1968) were the first to try and classify brain activity over the course of a night. To do this, they used the predominant frequency of the brain's electrical activity to distinguish between different stages of sleep.

The stages of sleep

In the waking state, the EEG is fast and desynchronised, the EMG indicates the muscles are tense, and the EOG indicates eye movements. When we are relaxed, the EEG shows slower, more synchronised alpha activity. The EMG indicates reduced muscle tension, and EOG activity is lessened. We are technically asleep when alpha activity gives way to theta activity.

Rechtschaffen and Kales identified **4 stages of sleep** based on the electrical activity the brain was showing. We can add a fifth stage to the four. This is called **REM sleep**. The other four stages are collectively known as **NREM sleep**.

In **REM sleep**, the EEG is **desynchronised**, and similar to the waking state. For this reason, REM sleep is sometimes called '*paradoxical sleep*'. As well as the irregular EOG in REM sleep, the EMG indicates no muscle tension at all, indicating that during REM sleep we are in a state of *virtual paralysis*. This is because the part of the cortex that deals with movement is inhibited during REM sleep. The function of this inhibition is believed to be to prevent us from acting out a **dream** we are having.

REM sleep and dreaming

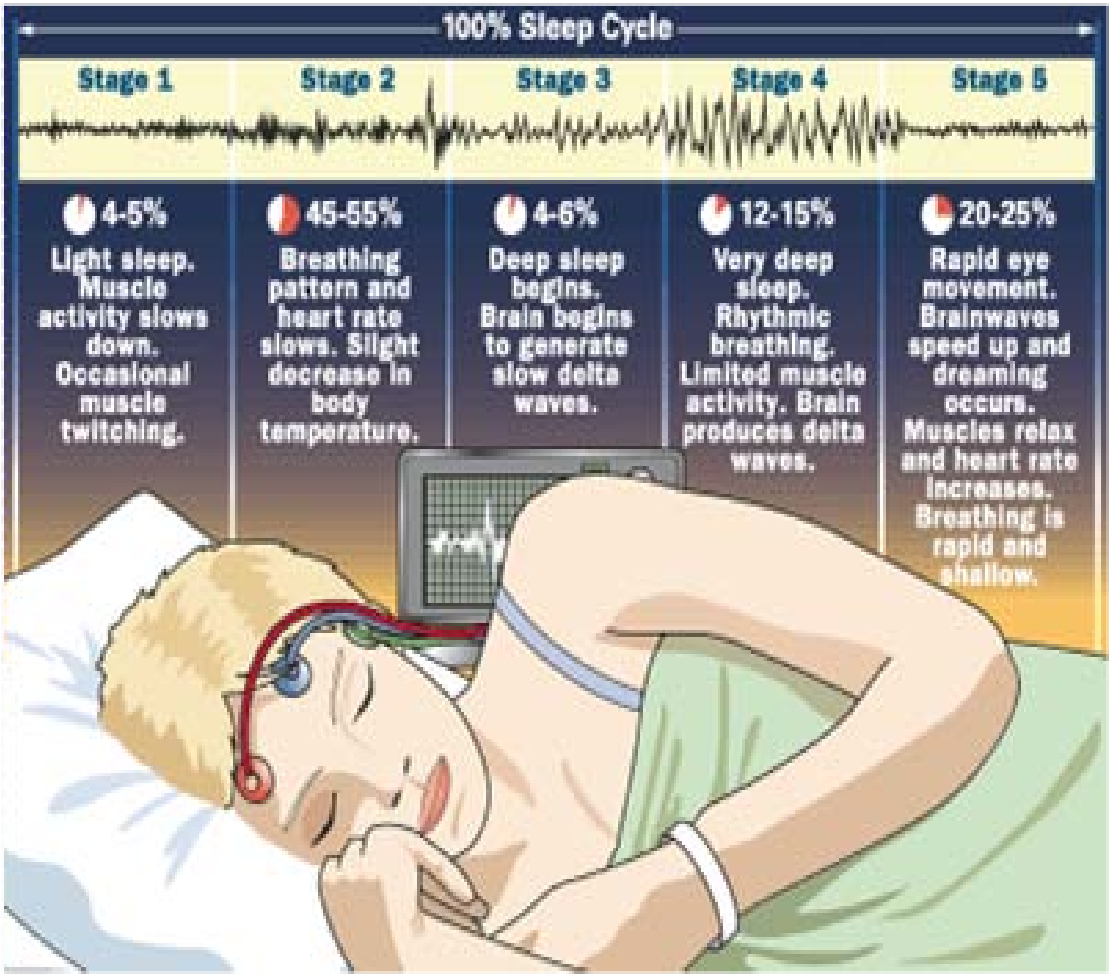
Aserinsky & Kleitman (1953) found that every 90 minutes or so, there is intense EOG activity during sleep. They called this **Rapid Eye Movement (REM)** sleep. A few years later, **Dement & Kleitman (1957)** were able to correlate REM sleep with ***dreaming***. They did this by waking up participants who were in their **sleep laboratory** and saying to them: "Tell me about your dream." They found that people awakened in this way reported dreaming 80-90% of the time. However, if people were woken at any other time of the night (called **Non-Rapid Eye Movement (NREM) sleep**), dreaming was reported less than 10% of the time. Research indicates that everyone dreams, and this has led to the proposal that dreaming must serve an important function, and is therefore an important part of the cycles of sleep.

The claim that REM sleep is when we dream and NREM sleep is when we don't is too simplistic. Some dreaming does occur in NREM sleep. However, whereas REM sleep dreams are typically bizarre, NREM dreams tend to be about mundane activities. The reasons for this are not known.

It is also impossible to be sure from which state a dream is being recalled. For example, if a person is woken in REM sleep and reports a dream, is that because the dream was occurring in REM sleep or because it has been remembered from NREM sleep?

It is not clear what the significance of dreaming is, and why it has to occur. However, it does seem to be necessary, because if a person is deprived of REM sleep for one night, they make up for 'lost time' when allowed to sleep normally. Freud saw dreaming as "the royal road to the unconscious" and therefore very important. Other researchers disagree, and see dreaming as the brain's attempt to make sense of the random activity that occurs across the brain when we enter REM sleep.

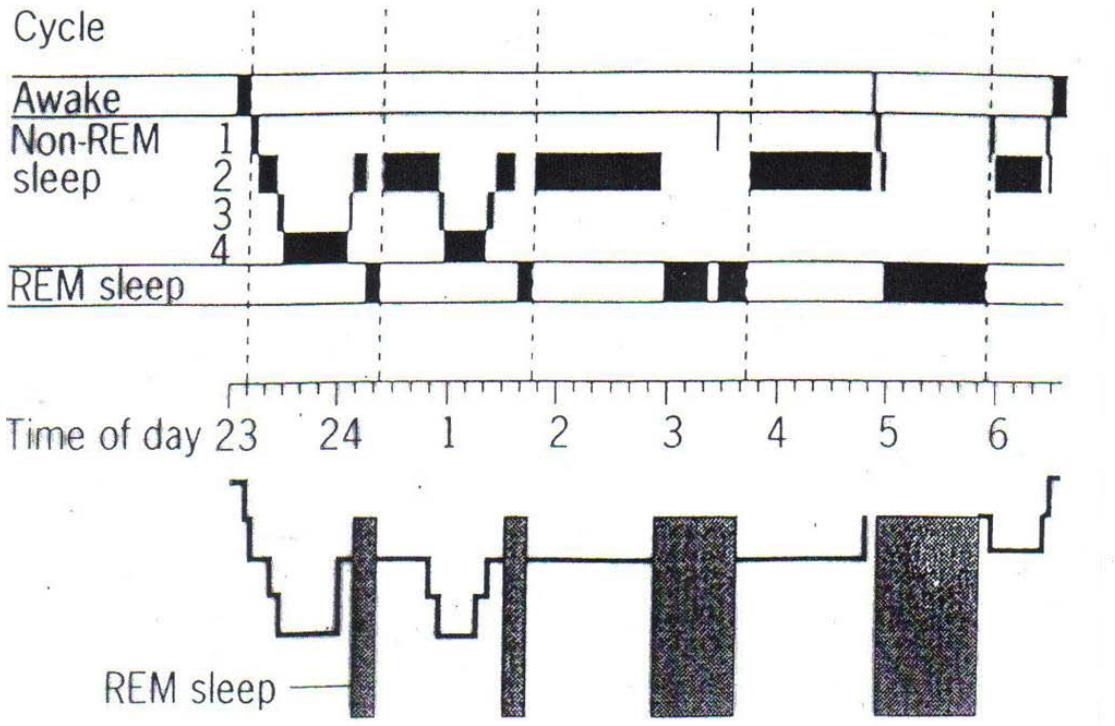
It would be interesting if the eye movements that occurred during REM were related to what the dream was about. However, whilst the eye movements *can* correspond to a dream's content, this is the exception rather than the rule.



The four stages of NREM sleep are each characterised by a particular kind of electrical activity. In REM sleep, the electrical activity is desynchronised, and the musculature paralysed.

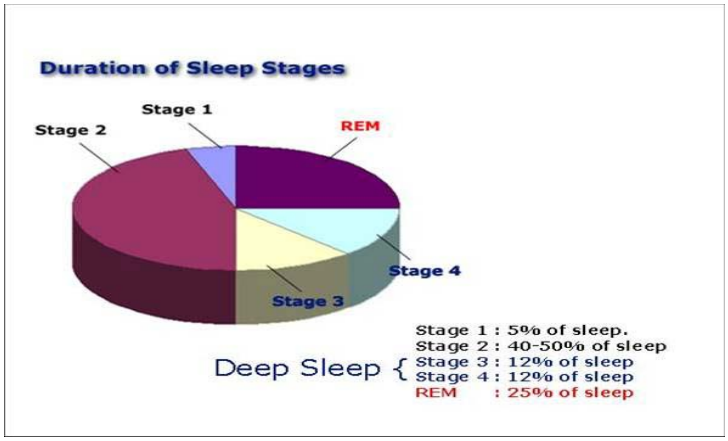
Note that there is strong evidence to support the view that the function of the paralysis in REM sleep is to prevent a dream being acted out. Research has shown that the structure involved in REM sleep is the **pons**, and in particular a group of cell bodies within it called the **locus coeruleus**. The locus coeruleus interacts with another group of cells in the pons called the **gigantocelluar tegmental field** to control the onset and offset of REM sleep. We know that these structures are important because (a) destruction of part of the pons causes REM sleep to disappear, and (b) destruction of another part maintains REM sleep but keeps muscle tension high. Thus, a non-human animal will carry out behaviours even though it s fast asleep. The human equivalent of this is called **REM Sleep behaviour Disorder**.

Rechtschaffen and Kales discovered that sleep is cyclical, with the first two cycles alternating between light, deep, light, and REM sleep, and the remaining cycles alternating between light sleep and REM sleep, as shown overleaf:



Notice that deep sleep (Stages 3 and 4) only occurs in the first two stages, and that only around 10 minutes or so is spent in REM in the first two stages. In the remaining stages, Stage 2 alternates with REM sleep, and REM sleep increases to around 45 – 60 minutes.

The total amount of time spent in the various stages is shown below:



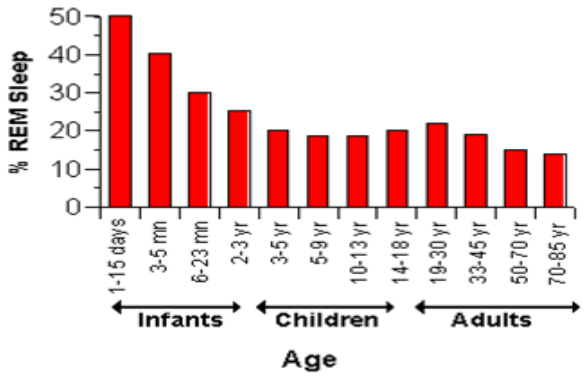
Dement and Kleitman found that whilst there are some individual differences in the length of each cycle of sleep, most people have a cycle of around 90 minutes, and that pretty much everyone shows a sleep pattern very similar to that illustrated above. This would suggest that the cycles of sleep themselves are determined by some kind of endogenous pacemaker.

Exogenous zeitgebers and the cycles of sleep

Remember that when external events play a role in rhythmic activities they are called *exogenous zeitgebers*. A good example of an exogenous zeitgeber that affects the cycles of sleep is *drugs*. For example, *alcohol* suppresses REM sleep, as do *anti-depressants*. However, when a person does not have alcohol having previously consumed a lot of it, their subsequent sleep involves *more* REM sleep time, as though the brain is trying to make up for the REM sleep that was lost when alcohol was consumed. This is called a '*rebound effect*'. When a person stops taking anti-depressants, there is *no* rebound effect. Given that anti-depressants work by increasing the availability of the neurotransmitter *serotonin*, this suggests that serotonin may be produced during REM sleep.

Lifespan changes in sleep

Another interesting finding is that sleep is not uniform across the lifespan. For example, newborns spent about 50% of their total sleep time in REM sleep, whereas in young adults about 20% is REM sleep. In the elderly, only about 14% of total sleep time is REM.



Presumably, there is some mechanism which determines how long we spend in REM sleep. Exactly why this change occurs is not known. However, it has been suggested that REM sleep serves an important function. According to Oswald's restoration theory the function of REM sleep is to *'psychologically restore'* us. Oswald argues that purpose of REM sleep is to replenish brain processes and repair the brain, and consolidate learning and memory.

Oswald believes that the reason new-born infants spend a long time in R.E.M. sleep is because they need to continually update their memory files so that they can acquire a language and learn to discriminate between different faces, as well as learning other things about the world. As we get older, the need to update memory files lessens because, for example,

we have now acquired a language. Therefore, the need for R.E.M. sleep becomes less as we grow older.

Another lifespan change that occurs in sleep is that the amount of time spent in Stage 4 sleep increases up to the age of 18, and then gradually decreases. In fact, by the time we get to very old age, Stage 4 sleep has virtually disappeared. Our total sleep time also decreases.

Oswald believes that stage 3 and 4 sleep decreases over the lifespan because those stages have 3 main functions: (1) they restore depleted energy resources, (2) they eliminates waste products from muscles, and (3) they enable physical abilities to be recovered. Therefore, the function of stage 3 and 4 sleep ('deep sleep') is to repair the damage done to the body during the waking day.

This is supported by the finding that the pituitary gland releases ***growth hormone*** during stage 4 sleep. This is important in tissue growth and red blood cell formation (i.e. the growth process). It has also been found that cell repair occurs during stage 4 sleep through a process called 'protein synthesis' (for which growth hormone is necessary). Oswald believes that more time is spent in Stage 4 sleep when we are younger because our need for growth hormone is greater (and therefore less time is spent in Stage 4 when we get older).