

ASPECTS  
OF SLEEP:

ESSAYS EMPHASISING  
RESEARCH  
METHODOLOGY

Kevin Brewer

ISBN: 978-1-904542-27-8

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PUBLISHED BY

Orsett Psychological Services,  
PO Box 179,  
Grays,  
Essex  
RM16 3EW  
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# 1. THE USE OF THE EXPERIMENT TO STUDY SLEEP

- 1.1 INTRODUCTION
- 1.2 EVALUATING TREATMENT
- 1.3 REFERENCES

## 1.1 INTRODUCTION

The experimental method allows the researcher to control the conditions, and to establish cause and effect relationships. The use of a control group compared to an experimental group means that the effect of the independent variable (IV) can be measured.

One example of an experiment in studying sleep is the learning experiment. Participants learn a new task, like a computer game which can be accurately scored, and then are tested on their improvement a few hours later. In the intervening time, some participants will sleep and others will not.

For example, Walker et al (2002) measured the number of times a sequence of computer keys (4-1-3-2-4) could be achieved in thirty seconds 4-12 hours after training the same day, and after a night of sleep. The former saw an average increase of 4% from the baseline, and the latter a 20% increase. The IV here was sleeping or not between learning and testing, and the DV (dependent variable) was the improvement in ability.

Sixty-two participants were allocated into five different groups in terms of training, testing, and sleep or not inbetween (table 1.1). The comparison between group B (no sleep inbetween learning and testing) and group D is the important one.

Table 1.2 gives the full results for each group with an average baseline of 14 sequences in thirty seconds after twelve training trials.

<u>GROUP</u>	<u>TRAINING</u>	<u>TESTING</u>	<u>SLEEP INBETWEEN</u>
A	10am	2pm/4pm/10pm	none
B	10am	10pm	none (but sleep before retesting)
C	10am	10pm*	as B
D	10pm	10am	yes (and retesting 12 hrs later after awake)
E	10pm	10am	yes (in sleep lab)

(\* complete hand rest - hand kept in mitten throughout day to avoid interference with learning) (After Walker et al 2002)

Table 1.1 - Experimental protocol in Walker et al (2002).

GROUP	TESTING	RETESTING
A	small non-significant improvement (1.08)	small non-significant improvement (1.08)
B	(W) 3.9 (0.94)	(S) 18.9 (4.33)*
C	(W) 4.3 (0.90)	(S) 19.7 (4.30)*
D	(S) 20.5 (4.50)*	(W) 2.0 (0.53)
E	(S) 17.9 (4.41)*	-

W = wake inbetween; S = sleep inbetween; \* = significant

Table 1.2 - Percentage improvement in performance from baseline and last testing in each condition (number of sequences improved).

## 1.2 EVALUATING TREATMENT

There are many treatments available for sleep problems, like insomnia, including sleeping pills. The question is whether they work. Any treatment needs to be evaluated by the use of the experiment, and, in particular, a specific type of experiment called the randomised controlled trial (RCT).

A RCT can be designed in a number of ways to compare treatment with no treatment. The most common designs are parallel group design and crossover design (table 1.3; figure 1A).

Berry et al (2006) devised a study, to compare these two types of design, looking at the effects of opiate drugs on deep sleep.

Forty-seven adults without sleep problems were locally recruited by the sleep lab at the University of California. There were three conditions to the study each involving two nights in the sleep lab. The first night in each case was for acclimation to sleeping attached to the measuring equipment of the sleep lab.

Condition 1 was the placebo and condition 2 was the drug. Together these two conditions were the crossover design. In condition 3, participants were randomly divided into two groups and given a different opiate. This was the parallel group design.

All conditions were double-blinded, and there was at least one week between the conditions. Due to drop-outs, data were only usable for thirty-two participants (table 1.4).

Based on statistical analysis, the researchers concluded that the crossover design was four times more statistically efficient than the parallel group design in recognising differences in deep sleep due to treatment or not.

	<u>PARALLEL GROUP DESIGN</u>	<u>CROSSOVER DESIGN</u>
DESCRIPTION	Comparison of two separate groups where 1 group receives treatment and other does not	Participants receive no treatment for a while, then given treatment for same length of time, or vice versa
SIMILAR TO EXPERIMENTAL DESIGN	Independent or unrelated design	Related or repeated measures
ADVANTAGES	<ul style="list-style-type: none"> <li>- Allows comparison between treatment and no treatment</li> <li>- Avoids bias or carryover effects of other design</li> <li>- Study both groups at same time</li> </ul>	<ul style="list-style-type: none"> <li>- Same individuals used both times (treatment/no treatment) makes groups comparable</li> <li>- Less participants needed, which important when time in sleep lab is expensive</li> <li>- Controls for expectation of improvement with treatment</li> </ul>
DISADVANTAGES	<ul style="list-style-type: none"> <li>- Different participants in each group who may not be comparable</li> <li>- Participants may realise they are in the control group and drop-out. This can bias comparison if big difference in size between groups</li> <li>- Need more participants than crossover design; thus expensive</li> <li>- Participants can guess whether treatment or not, and thus effects of expectation of improvement</li> </ul>	<ul style="list-style-type: none"> <li>- Problems of having treatment first, then not having it. This can affect the results, and is known as "carryover effect"</li> <li>- Drop-out of participant affects both conditions as that individual's data have to be removed</li> <li>- Requires participants more than once which increases risk of drop-out</li> <li>- Ethics of giving and removing treatment</li> </ul>

Table 1.3 - Comparison of parallel group and crossover designs in sleep research.

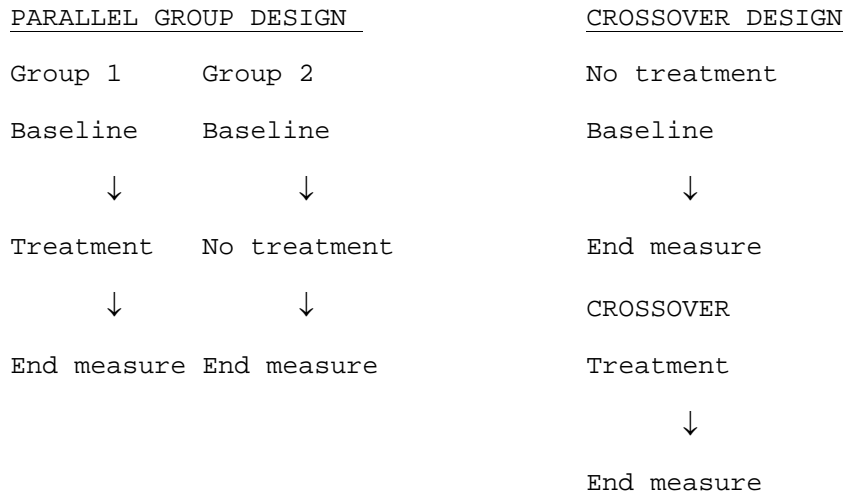


Figure 1A - Parallel group and crossover designs.

Consented to study	47
No show for condition 1	10 (21.3%)
Completed condition 1 but no show for 2	4 (8.5%)
Completed conditions 1 and 2 but no show for 3	0
Technical problems	1 (2.1%)
Sample used	32 (68.1%)

(After Berry et al 2006)

Table 1.4 - Details of drop-outs.

### 1.3 REFERENCES

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## **2. STUDYING SLEEP USING QUESTIONNAIRES: SOME EXAMPLES**

- 2.1 INTRODUCTION
- 2.2 SLEEPINESS
- 2.3 QUALITY OF SLEEP
- 2.4 BEHAVIOUR AND SLEEP
- 2.5 REFERENCES

### **2.1 INTRODUCTION**

Sleep is studied in two main ways - using subjective or objective methods. Subjective methods ask the individual about their experiences of sleep, while objective measures concentrate on physiological changes (eg: EEG).

Questionnaires can be self or other administered. They are used to measure different aspects of sleep.

Table 2.1 summarises the main advantages and disadvantages of the use of questionnaires to study sleep.

<u>ADVANTAGES</u>	<u>DISADVANTAGES</u>
<ul style="list-style-type: none"><li>- Gains insight into the subjective experience of sleep</li><li>- Easier to administer to large numbers of people than physiological measures. Can be used over the internet, and thus cheaper</li></ul>	<ul style="list-style-type: none"><li>- "Self-reporting of symptoms, leaving it open to misinterpretation, unintended bias and outright falsification" (Shen et al 2006 p65)</li><li>- Problems of establishing reliability and validity</li></ul>

Table 2.1 - Main advantages and disadvantages of studying sleep with questionnaires.

### **2.2 SLEEPINESS**

This is how awake/sleepy an individual feels. There are three main measures of sleepiness used in sleep research.

- i) Stanford Sleepiness Scale (SSS) (Hoddes et al 1973)

This contains seven statements for the individual to rate themselves every fifteen minutes: from "(1) feel active and vital; alert; wide awake" to "(7) almost in reverie; sleep onset soon; lost struggle to remain awake". It is an ordinal scale.



ii) Visual Analogue Scale (VAS) (Monk 1987)

This asks individuals to rate themselves on a 100mm line with no words other than "alert" at one end, and "drowsy" at the other. Both these measures rate sleepiness at that moment.

iii) Epworth Sleepiness Scale (ESS) (Johns 1991)

This questionnaire attempts to measure inappropriate sleep onset and daytime sleepiness. Individuals rate 0-3 (never - high chance of dozing) for eight situations; eg: "watching TV" or "in a car, while stopped for a few minutes in the traffic". A score of over ten out of twenty-four is seen as "pathological sleepiness".

## 2.3 QUALITY OF SLEEP

Quality of sleep is how well the individual feels that they sleep. For example, do they awake feeling refreshed? One example of a questionnaire to measure this idea is the recently developed Sleep Quality Scale.

Sleep Quality Scale (SQS) (Yi et al 2006)

This questionnaire originated from interviews with fifty adults attending a sleep laboratory in South Korea. Most of the attendees had some form of sleep problem (eg: seventeen were insomniac). Their views on what constituted "good sleep" and "poor sleep" eventually became the twenty-eight items of the SQS. For example:

- "clear-headed feeling after sleep"
- "increase of mistakes due to poor sleep"
- "wish for more sleep after getting up"

Each item is marked between 0 ("few") and 3 ("almost always").

A well-designed questionnaire must have reliability and validity, which were established for the SQS:

i) Reliability

This is a measure of the consistency of the questionnaire. One form of reliability, test-retest, is achieved by asking the same individuals to fill out the questionnaire at two different points in time. For the SQS, there was a positive correlation of +0.81 for a two-

week interval among 110 students.

## ii) Validity

This is assessing that the questions actually measure what they claim to measure, and there are different types of validity:

### a) Concurrent validity

The comparison of the SQS with a previously validated questionnaire of the same thing (Pittsburgh Sleep Quality Index; Buysse et al 1989). The researchers found a positive correlation (+0.72) between the scores on both questionnaires

### b) Construct validity

This is the relationship of scores on the SQS with expected behaviours. For example, individuals who perceive themselves as insomniacs should score high on the SQS (ie: poor quality sleep). A significant difference was found in answers given by insomniacs and good sleepers (mean 31.1 vs 15.8 respectively).

## 2.4 BEHAVIOUR AND SLEEP

Individuals hold certain beliefs about sleep, and this influences their behaviour (eg: belief that eating cheese before sleeping produces nightmares). In the case of insomnia, an individual's beliefs about the causes are important as to whether their behaviour helps or hinders the problem. This can be measured by the Sleep Belief Scale.

### Sleep Belief Scale (Adan et al 2006)

This is a twenty-item questionnaire developed from the Sleep Hygiene Awareness and Practice Scale (SHAPS) (Lacks and Rotert 1986). For each item the respondent has to decide if the behaviour has a positive, negative, or no effect on sleep. The items include:

- "drinking alcohol in the evening"
- "smoking before falling asleep"
- "going to bed with an empty stomach"

In each case, there is a correct answer as to the effect of that behaviour upon sleep.

Interestingly, Adan et al (2006) found that "morning-type" individuals (those who at best in morning) scored better than "evening-type" individuals among Spanish and Italian students. For example, 92.1% of the former group knew that "drinking coffee or other substances with caffeine after dinner" had a negative effect on sleep, compared to 76.1% of the "evening-type" individuals.

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### **3. THE USE OF ANIMALS TO STUDY SLEEP**

- 3.1 INTRODUCTION
- 3.2 SLEEP DEPRIVATION EFFECTS
- 3.3 REM DEPRIVATION AND MEMORY
- 3.4 PHYSICAL INTERVENTION STUDIES
- 3.5 GENETIC STUDIES
- 3.6 REFERENCES

#### **3.1 INTRODUCTION**

The use of animals in any type of psychological research is open to debate. The advocates point out that many experiments can be tried that would be unacceptable with humans, while those against their use, emphasise the assumptions that have to be made about what the animal is actually feeling or experiencing. This debate continues. Table 3.1 summarises the key arguments for the use of animals to study sleep.

<u>FOR</u>	<u>AGAINST</u>
1. Possible to study in way unacceptable with humans; eg: transgenic and physical intervention studies	1. Animals are not the same as humans, and because they lack language, assumptions have to be made about their behaviour.
2. Easier to control and study; eg: 24 hour environment and light/dark balance	2. Moral argument over rights and wrongs of such experiments, particularly when pain inflicted by the research

Table 3.1 - Key arguments for and against the use of animals to study sleep.

Animals have been used in a number of ways in studying sleep.

#### **3.2 SLEEP DEPRIVATION EFFECTS**

Rechtschaffen et al (1983) developed the method by which to keep animals awake in order to study the effects of long-term sleep deprivation.

Keeping rats from any sleep for up to a month produced physical symptoms like weight loss, body temperate drop, and immune system problems. Ultimately severe sleep deprivation was fatal.

As to the exact reason for death, it is debated. Everson and Toth (2000) believed that it was due to infection from bacteria, normally found in the stomach,

getting into the bloodstream.

Koban and Swinson (2005) deprived eleven male rats of REM sleep for twenty days. They kept the rats from REM sleep using the disk-over-water (DOW) technique. The rats lived in cages where the shallow water level is 1cm below their platform. In REM sleep, the muscles relax and the rats make contact with the water which wakes them. NREM sleep is possible because the muscles do not relax, and there is enough room on the platform to sleep if the rat is unmoved.

The environment was controlled at 12 hours of light and 12 hours of darkness with a steady temperature. After baseline measures, food was freely available as much as the rats wanted. The control rats (n = 5) were in similar cages but with no water.

The key finding was an 11% loss of body weight over the twenty days, while food intake rose to over double (220%) the baseline. This was due to an increase in the metabolic rate (166%). Put simply, REM sleep deprivation caused the body to use up energy at a much greater rate compared to the baseline and controls. Food consumption returned to normal levels immediately after the experiment stopped. None of the rats died during the experiment.

It should be noted that in some experiments the control animal is yoked, on a long lead, to the sleep-deprived animal. This means that the control animal is awakened every time the sleep-deprived one falls into the water. The control animal can, however, sleep as they want otherwise. The purpose of this process is to make the control and experimental conditions as similar as possible. But it does cause the control animals to be partially sleep deprived (Everson and Toth 2000).

In order to facilitate recording of physiological changes, the animals undergo preparatory surgery. Everson and Toth (2000) described what happened to the rats in their sleep deprivation study:

- A low frequency telemetric (radio) transmitter was implanted under the abdominal skin to record core body temperature;
- Electrodes were implanted in the head to record EEG waves. The electrode leads were soldered to head plugs connected to a long recording cable.

These researchers kept the rats awake by rotating the platform when sleep onset occurred, as detected by a microprocessor programmed to recognise changes in EEG waves, rather than the use of water. Everson and Toth felt that this process was "benign" compared to other methods used to keep rats awake. Note that the whole

experiment was automated so that the researchers did not have to miss their sleep.

### **3.3 REM DEPRIVATION AND MEMORY**

This type of study has been used to test the "REM consolidation hypothesis" (ie: REM sleep aids memory consolidation). Both animals and humans have been studied, and post and prior REM deprivation. Post studies involves REM deprivation after learning, and with prior studies, REM deprivation occurs before the learning. REM deprivation is relatively easy because the sleeper is woken only when their EEG shows the unique pattern of REM sleep, but they can have NREM sleep.

#### **i) Post REM deprivation studies**

Animals are taught a behaviour, then deprived of REM sleep, and retested for recall. Studies tend to be equally divided in finding that memory is and is not disrupted by REM deprivation (Vertes and Eastman 2000).

The main concern of these types of studies related to the means of keeping the animals awake. They are placed on a platform in a water tank, and when in REM sleep fall into the water to wake them. The stress, for example, of this procedure is a confounding variable as well as the loss of all sleep (not just REM) with this technique.

#### **ii) Prior REM deprivation studies**

Vertes and Eastman (2000) argued that studies depriving animals of REM sleep before training leads to performance, not necessarily memory, failures. In other words, the animals cannot physically perform the behaviour rather than having forgotten it.

Again the stress of REM deprivation techniques play as important a part as sleep loss itself. For example, van Hulzen and Coenen (1982) used two different techniques to deprive rats of REM sleep for three days. One technique was stressful (a platform in water), the other less so (a platform slightly off the ground, and the rats fall off when in REM sleep). Recall varied depending on the technique of deprivation used - it was worse for the more stressful technique.

### **3.4 PHYSICAL INTERVENTION STUDIES**

This type of study involves directly changing the

brain of an animal either through injection of a substance, or by deliberately destroying an area.

Damaging specific parts of the brain allow the researchers to see the role of that area in sleep. For example, destruction of different parts of the ventrolateral preoptic (VLPO) nucleus (in hypothalamus) reduced REM and NREM sleep. Damage to one area (VLPO cluster) reduced NREM sleep, while destruction of the extended VLPO reduced REM sleep (Saper et al 2005).

But an area of the brain "turned off" by destruction, and then not producing a certain behaviour, is not the same as the healthy part of the brain doing the behaviour. There is always assumption involved, and surgery may have damaged other areas of the brain.

### **3.5 GENETIC STUDIES**

Transgenic studies, with mice in particular, have developed in the last ten years. This allows both specific breeding strains and genetically engineered animals to be studied.

These types of studies, which change specific genes in the embryo, are most useful for understanding pathology. Genes can be manipulated to create the pathology, and thus isolate the genes involved, in a way that is not possible with humans. In other words, to go from healthy to unhealthy.

There are concerns about transgenic studies including the patenting of new genetic versions as if the animals were products. The correct reference number allows ordering of particular types of genetically manipulated animals from a mail order catalogue. Debate about the rights and wrongs of using non-human animals in psychology experiments can be outdated compared to the new areas like this where human genes are placed into mice, for example.

Tafti and Franken (2006) proudly reported their database at a Swiss university of sleep recordings of over 2500 mice, and the "creation" of four new strains of this animal. From these animals, the researchers claimed that 40-60% of variance in amount of sleep was genetic, and 80-95% for differences in EEG patterns (eg: delta wave oscillations in slow wave sleep).

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## **4. USE OF NEUROIMAGING TO STUDY SLEEP**

### 4.1. INTRODUCTION

### 4.2 COMPUTERISED AXIAL TOMOGRAPHY

### 4.3 NUCLEAR MAGNETIC RESONANCE IMAGING

#### 4.3.1 Magnetic Resonance Spectroscopy

### 4.4 POSITRON EMISSION TOMOGRAPHY

#### 4.4.1 Single-Photon Emission Computerised Tomography (SPECT)

### 4.5 FUNCTIONAL MAGNETIC RESONANCE IMAGING

### 4.6 MAGNETOENCEPHALOGRAPHY

### 4.7 EVALUATION

### 4.8 REFERENCES

### **4.1. INTRODUCTION**

Technological advances in brain imaging has made it possible to observe the live brain. There are a number of different types of neuroimaging looking at:

- i) The structure of the brain - CAT scan, MRI;
- ii) The function of the brain - PET scan, fMRI, MEG.

### **4.2 COMPUTERISED AXIAL TOMOGRAPHY (CAT SCANS)**

First used in 1972 (Sadock and Sadock 2003), this method produces a 3D X-ray picture of the static brain based on many X-rays from different angles and then combined together by the computer.

X-ray machines are based on the principle that abnormal tissue absorbs X-rays to a different degree to normal tissue.

There is a small risk from the X-rays if CAT scans are used too often on the same individuals.

### **4.3 NUCLEAR MAGNETIC RESONANCE IMAGING (NMRI or MRI)**

This technique, which entered clinical practice in 1982 (Sadock and Sadock 2003), also shows the static brain, through the use of magnetic fields.

It works by measuring the hydrogen atoms in water. The hydrogen nuclei are exposed to strong magnetic fields and line up like tiny magnets. Then they are hit with radio signals which causes them to move out of alignment. This produces a signal that can be measured.

Compared with CAT scans, MRI provides a better contrast between grey and white matter. The upshot of which is more anatomical detail (Johnstone 1993).

This is no need for radioactive substance to be injected, but there is concern about the effect of the strong magnetic field on the body.

#### **4.3.1 Magnetic Resonance Spectroscopy (MRS)**

MRS is based on the same principles as MRI, and uses magnetic fields - unpaired protons and neutrons aligned with a magnetic field. Radio frequency pulsing causes nuclei to absorb and emit energy. This produces a spectrum of the brain's chemical compounds.

There are different types of MRS: for example, observing the proton nucleus ( $^1\text{H}$ ) in the hydrogen atom ( $^1\text{H}$  MRS), or the stable isotope of phosphorous ( $^{31}\text{P}$ ) (Frangou and Williams 1996).

#### **4.3.4 POSITRON EMISSION TOMOGRAPHY (PET SCANS)**

This technique is able to show the active brain by following the movement of a radioactive substance that has been injected into the bloodstream. Radioactively labelled glucose molecules travel to active areas of the brain. When the radioactive atoms decay, they emit positrons (sub-atomic particles). These encounter electrons (the opposite type of particles) and both are annihilated. This gives rise to gamma rays that travel in opposite directions, and these can be traced to the point of origin.

Its strength is the ability to show blood flow patterns in the brain, which can be affected by, for example, strokes.

Different radioactive tracers (eg: water labelled with oxygen isotope  $^{15}\text{O}$ ) can be used to target different aspects of the brain's activities, like blood flow, glucose metabolism, dopamine receptors, or monoamine oxidase activity (Grasby et al 1996).

Because a small amount of radioactivity is involved, the World Health Organisation recommends one PET Scan per five years. 10 PET scans or 2 SPECT scans are the same as annual background radiation exposure (Liddle 1996).

#### **4.4.1 Single-Photon Emission Computerised Tomography (SPECT)**

This is a more sensitive measure of blood flow in the brain. It makes use of exametazime labelled with technetium isotope,  $^{99\text{m}}\text{Tc}$ , for example (Liddle 1996).

#### 4.5 FUNCTIONAL MAGNETIC RESONANCE IMAGING (fMRI)

This technique detects tissue mass based on blood flow, by measuring changes in deoxyhaemoglobin when neurons are active. Increased neural activity means a reduction in the concentration of deoxyhaemoglobin. In practice, it is possible to localise neuronal activity.

There is no need for a radioactive tracer (Liddle 1996). But acquisition of enough images for study can require the participant's head to remain still in the machine for up to three hours. Small changes in head position can lead to "erroneous interpretations" of brain activation (Sadock and Sadock 2003).

#### 4.6 MAGNETOENCEPHALOGRAPHY (MEG)

This technique makes use of changes in the magnetic fields in cortical neurons, which can be detected by magnets placed on the scalp. Liquid helium coiled superconducting sensors (eg: single superconducting quantum inference devices; SQUIDS) are used to pick up the faint magnetic fields. It is able to detect changes in signals over milliseconds, but it does not have the localised accuracy of MRI scans.

Neuroimaging has been used in a number of ways to study sleep (Nofzinger 2005):

- To measure the normal brain activity during the different phases of sleep - eg: less global brain activity in NREM compared to REM sleep;
- To measure the effects of sleep deprivation on the brain - eg: after 24, 48 and 72 hours sleep deprivation there is less activity (cerebral metabolism) in the frontal and parietal cortices, and in the thalamus;
- To study the brain activity in sleep disorders (table 4.1) - eg: changes in cerebral blood flow in individuals with obstructive sleep apnea syndrome (snoring);
- To study changes in the sleeping brain in other conditions, like depression or schizophrenia;
- To show the biochemical effects on the brain of pharmacotherapy (eg: sleeping pills).

<u>SLEEP DISORDER</u>	<u>EXAMPLE OF FINDINGS</u>
Narcolepsy	Differences in blood flow in parts of the brain in REM sleep of sufferers
Insomnia	Less overall blood flow in brain of insomniacs during NREM sleep
Restless legs syndrome	Differences in dopamine biochemistry in parts of brain of sufferers

Table 4.1 - Examples of findings on three sleep disorders from SPECT studies (Nofzinger 2005).

#### 4.7 EVALUATION

It is important to note that neuroimaging studies still have limits, however perfect they seem (Hobson et al 2000):

i) How to interpret the findings - Neuroimaging can show differences in blood flow, oxygen uptake, and glucose use in parts of the brain, but is an increase or decrease in such activities efficient functioning of the brain?

ii) Most studies use only small samples because of the cost and time involved in neuroimaging.

iii) Focus on overall changes in the brain may miss specific local differences.

iv) General problems of participants sleeping inside these machines.

Table 4.2 summarises the main advantages and disadvantages of each type of neuroimaging.

<u>ADVANTAGES</u>	<u>DISADVANTAGES</u>
<u>CAT SCANS</u> <ul style="list-style-type: none"> <li>- Relatively inexpensive compared to other methods</li> <li>- Limited restrictions on who can have them</li> <li>- Can show if brain tumour cause of sleep disorders</li> </ul>	<ul style="list-style-type: none"> <li>- Risk of radiation</li> <li>- Poor image resolution</li> </ul>
<u>MRI SCANS</u> <ul style="list-style-type: none"> <li>- Not using X-rays</li> <li>- Superior resolution of image over CAT</li> <li>- Able to detect different substances</li> </ul>	<ul style="list-style-type: none"> <li>- Some individuals cannot use; eg: those with pacemakers or metal in body</li> <li>- Expensive</li> <li>- Some health risk of strong magnetic field</li> <li>- Can cause claustrophobia</li> </ul>
<u>PET SCANS</u> <ul style="list-style-type: none"> <li>- Shows how brain uses variety of molecules</li> <li>- Provides absolute measure of regional cerebral blood flow (rCBF) (Banich 2004)</li> </ul>	<ul style="list-style-type: none"> <li>- Health risks of radiation</li> <li>- Time-lag to produce picture; eg: 1½ minutes "averaging" (Banich 2004)</li> <li>- Requires continual infusing of radioactive isotope</li> <li>- Needs large number of individuals and "averaging" of their scans, but problems with "averaging"</li> <li>- Positioning of patient very important and tilt can influence results</li> </ul>
<u>fMRI SCANS</u> <ul style="list-style-type: none"> <li>- Few risks</li> <li>- Multiple scans possible</li> <li>- Shows brain activity over seconds</li> <li>- Good resolution of images</li> </ul>	<ul style="list-style-type: none"> <li>- Does not measure neuronal response directly but response of vascular system to increased need for oxygen of neurons; thus can have 2 second time-lag (Banich 2004)</li> <li>- Some individuals cannot use (as with MRI scans)</li> <li>- Small changes in head position has effect on scan</li> </ul>
<u>MEG</u> <ul style="list-style-type: none"> <li>- Can distinguish between activity in each hemisphere</li> <li>- Gives signals over milliseconds</li> </ul>	<ul style="list-style-type: none"> <li>- Very expensive equipment</li> <li>- Cannot detect activity of cells within certain orientations in brain (Banich 2004)</li> <li>- Not localised accuracy of MRI</li> <li>- SQUID needs to be at temperature of -273 (ie: 4 degrees above absolute zero)</li> </ul>

Table 4.2 - Main advantages and disadvantages of different types of neuroimaging.

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## **5. HUNTING FOR GENETIC CAUSES: THE EXAMPLE OF RESTLESS LEGS SYNDROME**

### 5.1 RESTLESS LEGS SYNDROME

### 5.2 REFERENCES

#### **5.1 RESTLESS LEGS SYNDROME**

Restless legs syndrome (RLS) (or Ekbom's syndrome) (Ekbom 1960) is the involuntary movement of the legs during NREM sleep which disrupts sleep. There is an unpleasant feeling in the legs which is relieved by movement. Up to 15% of the general population may suffer (Phillips et al 2000), though there is a female predominance (Winkelmann and Ferini-Strambi 2006).

Diagnosis is based upon four "essential criteria" (Walters 1995):

- The urge to move the legs due to an unpleasant sensation in them;
- This feeling is evident during rest;
- The feeling is relieved by movement;
- Symptoms are worse at night.

Though full knowledge of RLS is not yet achieved, there seems to be two different versions (early and late onset in life), with the earlier onset type being hereditary (Taheri and Mignot 2002). Winkelmann et al (2000), in a study of 300 patients, found nearly a fifteen-year difference in average age of onset (35.4 years vs 47.2 years).

There are a number of ways of researching the hereditary origins of behaviours:

#### i) Family studies

This type of study is based around the "genetic family tree" of sufferers. Working with a sufferer (proband), the researchers try to discover how many "first degree relatives" (eg: mother, father, siblings) also the same condition. A few of the studies use control groups. The frequency of RLS in families of sufferers varies between 40 - 90% depending on the study (Winkelmann and Ferini-Strambi 2006).

In a "natural experiment", there are a large number of familial cases among French-Canadians in the Quebec area who descended from a small number of 17th century

founders. It is believed to be a recessive gene (Taheri and Mignot 2002).

But there could be common environment factors. The higher prevalence of a behaviour in a family is not a guarantee of a hereditary cause.

#### ii) Twin studies

Identical twins (monozygotic - MZ) have the same genetic make-up, so if a behaviour has a genetic cause, then it should appear in both twins. Ondo and Jankovic (1996) found that ten of twelve pairs (concordance rate 83%) of self-selected MZ twins both had RLS. But the severity and age of onset varied between twins.

It would be better if the twins were raised in separate environments, and this would reduce the risk of environmental factors on behaviour.

#### iii) Molecular genetics

This was developed with the increasing knowledge of genes based around the sequencing of DNA. The aim is to establish the role of specific genes, and to search for differences or patterns of them in diseases.

Linkage studies initially suggested genes on chromosome 12 (known as RLS1) in a South Tyrol family and an Icelandic sample, and then chromosomes 14 (RLS2) (an Italian family), and 9 (RLS3) (2 US families) (Winkelmann 2006; Winkelmann and Ferini-Strambi 2006).

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## **6. A KEY STUDY IN THE HISTORY OF SLEEP RESEARCH: DEMENT AND KLEITMAN (1957)**

- 6.1 INTRODUCTION
- 6.2 STUDY DETAILS
- 6.3 EVALUATION
- 6.4 REFERENCES

### **6.1 INTRODUCTION**

The nature of the relationship between rapid eye movement (REM) and dreaming is now well established. Aserinsky and Kleitman (1953; 1955), and Dement (1955) were the pioneers here: waking participants during REM sleep and finding that they reported dreams compared to no such reports during NREM sleep periods. Dement and Kleitman (1957) developed this research.

But Aserinsky and Kleitman did not initially identify the basic sleep cycle because their EEG readings were samples for a few minutes in every hour to save paper and to allow the researchers to nap between readings (Dement 1993).

Prior to this research, there were various beliefs about dreaming including the link to gastric contractions in the 1920s, and foot twitches in the 1940s. At the beginning of the twentieth century, it was even believed that mental activity ceased during sleep.

The study of sleep and dreaming has two strands:

- Subjective one based on self-reported dream content recall by awakened sleepers
- Objective one using electroencephalograph (EEG) and other physiological measures during sleep.

As it stands, there can only be correlations established between the two (unless a machine is invented to read the dream content as well as the brain's electrical activity).

### **6.2 STUDY DETAILS**

Dement and Kleitman (1957) studied nine individuals (five in detail) in their sleep lab. The length of participation varied from one night to seventeen. Participants had three electrodes attached to the scalp to measure electrical brain activity, and two electrodes near the eyes to record eye movements. The participants were left to sleep in a dark room, and were awakened

intermittently during the night to be asked about dream recall.

Waking involved a doorbell, and participants spoke into a recording device to avoid interaction with the experimenter, and to eliminate "the possibility of unintentional cueing" by the experimenter. There were a total of 351 awakenings from sixty-one person-nights in the sleep lab.

The findings of this study established key aspects of knowledge about sleep and dreaming:

i) Sleep is divided into different phases and types during the night based on EEG patterns. REM is the most distinct. First noted by Aserinsky and Kleitman (1953).

ii) Dream recall occurred more often with awakenings from REM sleep (80%; 152 of 191) than NREM sleep (7%; 11 of 160). The time of waking did not affect the dream recall as there were approximately equal numbers of dream reports from REM sleep in the first and second half of the night's sleep (85% and 78% respectively). Failure to recall dreams from REM sleep was mostly early in the night's sleep (approximately half of such cases).

iii) Participants were able to estimate reasonably accurately the length of the dreaming period (as either five or fifteen minutes): 88% correct for five minutes and 78% for fifteen minutes (total 83% correct).

It was decided to use five and fifteen minutes because it was easier to see when the REM period had begun, and then to wake the sleeper. Otherwise there was no way of knowing the accuracy of the estimates of dream length.

iv) Eye movement patterns seemed to correspond to the direction that the individual was looking in their dreams. The pattern of eye movements were divided into four: vertical, horizontal, both, or neither.

For example, a pattern of vertical eye movements were measured, and the dreamer reported standing at the bottom of a cliff looking up and down. Only thirty-five awakenings were collected here.

### **6.3 EVALUATION**

i) The data collected were based on a large number of awakenings (351) overall, and five individuals were studied in detail (321 awakenings from 55 person-nights). This is a small sample size.

ii) Only two of the nine participants were female,

but it is not clear if they were part of the select five participants studied in detail.

iii) The individuals were asked to describe the dream on waking, and "are thus likely to mix, if not contaminate, the dreaming phenomenology with the phenomenology of waking" (Hobson et al 2000). As it stands, there is no other way.

Furthermore, language is being used to express mental activity that may be beyond language. Attempting to describe in words conscious experience is always open to problems.

iv) The methodology used was a controlled observation rather than a "true" experiment. Controlled observations are halfway between the experiment and naturalistic observation methods. It is an observation in a lab situation. There may be influences on the environment by the researcher, but it is not manipulation of an independent variable as in a "true" experiment. Table 6.1 compares the advantages and disadvantages of the controlled observation, naturalistic observation, and the experiment.

Subsequent studies (eg: Dement et al 1965) have found that sleeping in the sleep lab environment produces not only poor quality sleep (known as "first night effect"), but also the content of dreams were be different (eg: more dreams including references to lab conditions).

v) Subsequently, the findings of Dement and Kleitman (1957) have been adapted and developed or challenged. One area is dreaming during NREM sleep.

It could be that individuals awakened from deep sleep (NREM) struggled to recall the dream rather than not actually dreaming, whereas REM sleep is lighter sleep (Beaumont 1988).

Foulkes and Rechtschaffen (1964) found 62% dream recall from NREM sleep (vs 89% for REM sleep) among twenty-four participants, and Foulkes and Schmidt (1983) 67% and 93% respectively from a similar number of participants.

Nielsen (1999), in a review of previous studies, found average dream recall rates of 81.8% from REM sleep and 42.5% from NREM sleep. But the type of recall from NREM sleep was limited to fragments, and the recall from REM sleep was much more characteristic of what is generally called dreaming (longer, more bizarre, more visual, and more emotional) (Hobson et al 2000).

<u>METHOD</u>	<u>ADVANTAGES</u>	<u>DISADVANTAGES</u>
Controlled observation	Stricter control than naturalistic observation  More flexible than experiment	Low ecological validity  Participant reactivity because they know they being observed
Naturalistic observation	High ecological validity  Participants not aware being observed	Not possible to establish cause and effect  Replication difficult
Experiment	Establish cause and effect relationship between variables  Replication possible because of control over variables	Generally focuses on narrow aspects of behaviour  Risk of demand characteristics and experimenter effects

Table 6.1 - Comparison of advantages and disadvantages of controlled and naturalistic observations, and the experiment.

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## **7. STAYING AWAKE OUT OF CHOICE: VOLUNTARY SLEEP DEPRIVATION**

- 7.1 INTRODUCTION
- 7.2 LAB STUDIES
- 7.3 REAL-LIFE STUDIES
  - 7.3.1 Peter Tripp
  - 7.3.2 Randy Gardner
- 7.4 REFERENCES

### **7.1 INTRODUCTION**

The effects of voluntary sleep deprivation can be studied in two ways: in laboratory studies or in real-life situations (table 7.1).

<u>METHOD</u>	<u>ADVANTAGE</u>	<u>DISADVANTAGE</u>
Lab experiments	- Control over variables and participants	- Artificial situation of sleep lab
Real-life studies	- High ecological validity	- Hard to control and verify sleep deprivation

Table 7.1 - Advantage and disadvantage of two methods of studying voluntary sleep deprivation.

### **7.2 LAB STUDIES**

These involve volunteers who agree to stay awake for certain periods of time.

Lab studies allow more controlled observation of sleep deprived individuals than real-life studies, but usually for shorter periods because of ethical concerns for the participants.

Drummond et al (2000) is a good example of a recent study which makes full use of modern technology in the lab. Thirteen young adults were given functional magnetic resonance imaging (fMRI) scans after thirty-five hours of sleep deprivation. Different parts of the brain were active during memory tests compared to after sleep. The prefrontal cortex and areas of the parietal lobe were more active, and areas of the temporal lobe less active after sleep deprivation. This was interpreted as compensation by the brain. However, average recall of nouns dropped from 4.7 words to 2.8 after sleep deprivation.

Lab studies show that it is not necessarily long sleep deprivation that affects individuals, particularly with higher-order cognitive functions. Killgore et al (2006), recently, noted that 49.5 hours of sleep deprivation was enough to impair judgement and decision-making (particularly in conditions of uncertainty ie no clear correct answer).

This research used thirty-four healthy student volunteers in Washington DC. Participants were asked to abstain from stimulants 48 hours prior to the study, which was verified by a urine sample. The decision-making task used was the Iowa Gambling Task (IGT), which is a computer-based card-gambling game. The study began at 1800 hours on Day 1 with physiological measures and controlled sleep. The sleep deprivation ran from waking on Day 2 until mid-day on day 4.

Sleep-deprived individuals became more risky on the IGT, and this increased with age (ie: older participants more risky choices). The area of the brain involved in such decisions is the prefrontal cortex, and this seems to be vulnerable to sleep loss.

However, it is possible that the participants may have chosen riskier cards in order to stimulate themselves to stay awake. The researchers countered this possibility by telling the participants that they could keep any winnings.

### **7.3 REAL-LIFE STUDIES**

Opportunist studies have been made of individuals voluntarily staying awake for very long periods, as in world record attempts: well-known examples like Peter Tripp (Luce and Segal 1966) and Randy Gardner (Dement 1978).

#### **7.3.1 Peter Tripp**

In 1959, in New York City, WMGM radio DJ, Peter Tripp attempted to stay awake for 200 hours (8 days 8 hours) ("wakethon") to benefit a charity. During this time, he was broadcasting his daily show from a glass-walled booth in Times Square (photographs at <http://www.manfrommars.com/tripp.html>).

Because of the concern for his health, Tripp was well studied throughout the 200 hours by doctors and psychiatrists (Floyd Cornelison and Louis West). There was considerable interest from sleep researchers as the knowledge about sleep (and sleep deprivation) was limited at that time.

After two days, Tripp stated hallucinating - eg: "Specks on the table began to look like bugs. He thought he saw a rabbit in the booth" (Luce and Segal 1966 p91). Some of the hallucinations were linked to paranoia, like a hotel desk was on fire (after 120 hours).

On the last day, he mistook a doctor (Dr.Wolff) with an umbrella as a funeral director come to measure him for a coffin.

Memory and concentration problems increased with time - by 170 hours, he could not say the whole alphabet. By 150 hours awake, he was disoriented. Yet he still managed each day to do his three-hour radio show (5-8pm) effectively. In fact, some listeners had no idea he was not sleeping.

At the end of the 200 hours (201 exactly; Coren 1996), he slept for thirteen hours, and awoke apparently refreshed, though he did have mild depression for three months (Luce and Segal 1966).

As to the long-term consequences of this sleep deprivation, it is unclear. Tripp lost his job at the radio station through a financial scandal soon after, and he had four marriages ending in divorce. These events are probably unrelated to the sleep deprivation. He lived until age 73 ([http://en.wikipedia.org/wiki/Peter\\_Tripp](http://en.wikipedia.org/wiki/Peter_Tripp); accessed 02/04/07).

### **7.3.2 Randy Gardner**

In January 1964, 17 year-old student, Randy Gardner, attempted to stay awake for 264 hours (11 days) as a project for the San Diego Science Fair.

Some textbooks reported that Randy had few problems from the sleep deprivation, but Coren (1996) felt that was inaccurate. Daily examinations were made by Lt. Commander J.J.Ross of the US Navy Medical Neuropsychiatric Research Unit. Here are some of the main observations of problems that came and went (quoted in Coren 1996):

Day 2: Visual problems which made watching television impossible;

Day 3: Mood changes, and physical co-ordination problems;

Day 4: High irritability; memory and concentration problems; hallucinations (eg: street sign was a person) and delusions (eg: believed he was famous US football player);

Day 5: Hallucinations less disturbing as he was now aware what they were; general improvement;

Day 6: Relapse to day 4;

Day 7: Slurred speech, and reappearance of high irritability;

Day 8: Memory and concentration problems most apparent;

Day 9: Fragmented thinking (eg: unable to finish sentences);

Day 10: Paranoia (eg: thought radio DJ who interviewed him was against him);

Day 11: Physical co-ordination reasonable; slurred speech; memory problems.

Afterwards Randy slept for 14 and 3/4 hours, and awoke reasonably refreshed. On the second night, he slept for twelve hours, and 10 hours on the third night. His normal length of sleep (8 hours) returned by the next night. He mostly recovered stage 4 NREM sleep then REM sleep (Horne 1988).

Other claims have been made by individuals in terms of staying awake for very long periods of time. But most of these are unsubstantiated in scientific terms.

For example, a claim of staying awake for over five weeks was made by "Little red Eft" on the "Insomnia Forum"

(<http://www.sleepnet.com/insomnia5/messages/645.html>; accessed 02/04/07). The individual claimed that between 7th January and 13th February 2000 (over 700 hours), they did not sleep for "one minute in the entire time, and eventually was hospitalized".

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## **8. CHANGES IN SLEEP PATTERNS DUE TO BRAIN DETERIORATION**

- 8.1 INTRODUCTION
- 8.2 SLEEP/WAKE PATTERNS
- 8.3 QUALITY OF SLEEP
- 8.4 DIFFERENT TYPES OF BRAIN DETERIORATION
  - 8.4.1 Parkinsons Disease
- 8.5 REFERENCES

### **8.1 INTRODUCTION**

Deterioration in the brain through illness or injury can lead to changes in sleep in two ways:

- i) The sleep/wake (circadian) pattern - Usually this cycle involves one period of sleeping (at night), and activity during the day. Sometimes there is a second (shorter) period of sleep in the afternoon (siesta);
- ii) The quality of sleep - This includes how long the individual sleeps, how they feel about their sleep (eg: waking feeling refreshed), and the make-up of rapid eye movement (REM) and non-REM (NREM) sleep.

### **8.2 SLEEP/WAKE PATTERNS**

Individuals with dementia often have unstable sleep/wake rhythms. This leads to waking at night, and daytime sleeping (Paavilainen et al 2005). Very simply, the normal day/night sleep/wake cycles are disrupted.

There are problems in studying such changes. Keeping individuals with dementia in sleep labs for long periods is very difficult, while using self-reported sleep diaries is not possible with such individuals. This has led to the use of telemetric actigraphy. Individuals wear a small device on their wrists, like a wrist-watch, and this sends a wireless signal to a base station (similar to a mobile phone signal). Thus it is possible to know where or what (ie: active or not) an individual is doing at all times.

Paavilainen et al (2005) tested one such system (IST Virago system) with residents of two private nursing homes in southern Finland. Of the forty-two volunteers aged 56-97 years, 23 were diagnosed with dementia. The data were collected for ten days.

Overall, the study measured bedtime, awakening time, number of nocturnal awakenings, and time in bed (TIB)

with the actigraphy device, as well as subjective measures of quality of sleep.

The study found differences in the time spent in bed between the individuals with and without dementia (mean of 11 and 9 hours respectively), but similar scores for quality of sleep. There was a negative correlation between time spent in bed and cognitive ability.

Despite spending more total TIB in a 24-hour period, individuals with dementia showed no circadian pattern of activity and inactivity. In other words, there were sleeping and waking at different and varied times. The individuals without dementia had the traditional circadian patterns - inactivity during the night, and, for a short period, in the afternoon.

Another aspect of the disruption of the sleep/wake cycle in dementia sufferers is "sundowning" (confusion in early evening) (Ancoli-Israel et al 1993).

### **8.3 QUALITY OF SLEEP**

Case studies of individuals who experience brain injury show changes to the make-up of sleep - for example, damage to the brainstem and vast reduction in quantity of REM sleep (Vertes and Eastman 2000).

Brainstem damage, when not severe enough to produce coma or death, can lead to a condition called "locked-in" syndrome (Plum and Posner 1966). Such individuals are conscious and alert, but physically paralysed and mute. The other type of sleep (NREM) is unaffected in this condition.

In less severe cases of damage to the brainstem, though lacking REM sleep, individuals are able to live normal lives. One twenty-year-old man received gunshot wounds to part of the brainstem, but, after his ten-day coma, continued his life. When examined at thirty-three, he was found to have virtually no REM sleep in a night's sleep (2.25% of total sleep time) (Lavie et al 1984). The usual amount is nearer 20% at that age (Weiten 1995).

The amount of different types of sleep (ie: REM/NREM) also changes with dementia - eg: reduced proportion of sleep as REM in EEG studies of Alzheimer's disease sufferers (Ancoli-Israel et al 1994).

The quality of sleep has a strong subjective element. The perception of good (eg: refreshing) or poor (eg: insomnia) quality sleep is affected by, for example, the expectations associated with sleep. Quality of sleep may have no relationship to TIB.

Tractenberg et al (2006) investigated sleep problems among 662 non-institution-living, elderly adults (in

their 70s and 80s) (399 without dementia) as part of the Oregon Brain Aging Study (OBAS).

The perception of sleep quality was measured using the Sleep Disturbance Symptom Questionnaire (Tractenberg et al 2005). This contained twenty items (symptoms) to be rated "in previous months" from 0 (never) to 4 (nearly every day/night) giving a maximum of eighty. Scores of two or more on individual symptoms or a total score greater than forty were classed as sleep disturbance.

Items included "snores heavily", "restless sleep", "uses alcohol to help get to sleep", and "has restless legs during sleep". The researchers added the item: "awakes feeling well rested". The questionnaire was self-reported, except for individuals with dementia where the assistance of a caregiver was required.

More individuals without dementia (81.5%; 325 of 399) reported sleep problems than those with dementia (27.8%; 73 of 263). Analysis of the four groups (sleep problems/not; dementia/not) showed greater differences between the individuals with or without sleep problems in each case rather than between dementia and not.

In both groups (dementia/not), sleep problems were characterised by taking a long time to fall asleep, lying awake worrying, restless sleep, waking too early, and waking during the night.

The only difference between individuals with or without dementia (irrelevant of sleep problems) was daytime sleeping (ie: equal sleep time during the day and the night). This was higher for individuals with dementia.

It must be remembered that the questionnaires were self-reported for individuals without dementia (in the main) and caregiver-reported for those with dementia. The first group gave a subjective assessment of their quality of sleep, while the latter were probably more objective. However, caregiver reports are not the same as the individual's perceptions. Tractenberg et al (2006) admitted that it was ".. unclear whether any differences between groups.. are due not to actual differences in the sleep disturbance symptoms but instead are due to the level of awareness of the symptoms in the reporters.. " (p102).

#### **8.4 DIFFERENT TYPES OF BRAIN DETERIORATION**

Distinctions can be made between different types of dementia, like Alzheimer's disease (AD) and dementia with Lewy bodies (DLB). In an English study, Grace et al (2000) compared these two groups using two questionnaires. The Epworth Sleepiness Scale (ESS) (Johns

1991) attempts to measure inappropriate sleep onset and daytime sleepiness.

The Pittsburgh Sleep Quantity Inventory (PSQI) (Buysee et al 1988) has seven items measuring sleep disturbance over the previous month on a scale of 0-3. A higher score signifies more sleep problems. The questionnaires took approximately fifteen minutes to fill out.

Both groups showed sleep problems, but the DLB group were worse: more daytime sleepiness, more sleep disturbances at night, and poorer subjective sleep quality (table 8.1).

	AD Group n = 20	DLB Group n = 17
ESS mean score (out of 24)	6.5	12.6
PSQI mean score (out of 21)	4.6	8.5
Subjective sleep quality as "bad" or "fairly bad" (%)	15	53

(After Grace et al 2000)

Table 8.1 - Sleep differences between two types of dementia sufferers.

#### 8.4.1 Parkinson's Disease

Sleep problems are common in individuals with Parkinson's Disease (PD), including sleep fragmentation (frequent waking) and excessive daytime sleepiness (Factor et al 1990).

These researchers questioned 78 US adults with PD, and 43 elderly matched controls about four areas of sleep - sleep initiation (time taken to fall asleep), sleep maintenance (number of awakenings), dream content, and daytime sleepiness. The PD sufferers took longer to fall asleep, and had more awakenings in the night, but no differences in dream content or napping (table 8.2). Though there was no difference in napping, the PD sufferers were more likely to spontaneously doze (48.7% vs 25.6%), and though altered dreaming was the same, PD sufferers had more nocturnal vocalisations (sleeptalking) (30.8% vs 0%). The PD sufferers were more likely to use sleep medication (20.5% vs 2.3% every night).

There is the question of how much the change in sleep quality in PD sufferers is due to medication for their illness.

	<u>PD Group</u>	<u>Control group</u>
Difficulty falling asleep	66.6	53.5
- taking 0-20 mins	29.5	27.9
- taking longer than 45 mins	20.5	14.0
Reported awakening in night	88.5	74.4
- once	16.6	27.9
- more than five times	3.9	0
Napping during day	66.6	65.1
Altered dreaming	9.0	9.3
Use sleep ever in last week	34.6	14.0

(After Factor et al 1990)

Table 8.2 - Percentage of respondents reporting problems with aspects of sleep.

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## **9. NARCOLEPSY, OREXIN, AND COGNITIVE PROBLEMS**

- 9.1 INTRODUCTION
- 9.2 CHARACTERISTICS OF NARCOLEPSY
- 9.3 CAUSES OF NARCOLEPSY
  - 9.3.1 Orexins
- 9.4 COGNITIVE EFFECTS OF NARCOLEPSY
- 9.5 REFERENCES

### **9.1 INTRODUCTION**

Narcolepsy is an example of a sleep disorder from the group called hypersomnia (Mahowald and Schenck 2005). The term narcolepsy was first coined in 1880 in Paris by French doctor, Jean Baptiste Edouard Gelineau (Zeman et al 2001), but patients with symptoms were described from 1862 (Dement 1993). Narcolepsy is best known as suddenly falling asleep.

"The study of narcolepsy has been instrumental in promoting the concept of state dissociation: wakefulness and sleep are not necessarily mutually exclusive states, and furthermore elements of one state of being may intrude inappropriately into another, often with striking consequences" (Mahowald and Schenck 2005 p1280).

Narcolepsy is relatively rare affecting one in two thousand people (Mahowald and Schenck 2005). Though rates vary between 1 in 600 in Japan to 1 in 500 000 in Israel (Siegel 2000) (0.04% of the UK population; Ohayon et al 1996).

It appears most often in the 20s and 30s (Zeman et al 2001).

A questionnaire used to measure narcolepsy is the Ullanlinna Narcolepsy Scale which includes items like:

"When laughing, becoming glad or angry or in an exciting situation, have the following symptoms suddenly occurred?"

- Knees unlocking
- Mouth opening
- Head nodding
- Falling down

Response choices: "never" (0) to "daily or almost daily" (4) (Shneerson 2005).

## 9.2 CHARACTERISTICS OF NARCOLEPSY

Narcolepsy involves four key symptoms:

### i) Sudden sleepiness

In the multiple sleep latency test (MLST), individuals with narcolepsy have a short mean sleep onset time (eg four minutes compared to normal ten minutes), and REM sleep occurs very quickly (known as sleep-onset REM period; SOREMP) (Zeman et al 2001).

SOREMPs also occur after sleep deprivation, withdrawal from REM sleep-suppressing medications, and in rare conditions like Prader-Willi Syndrome (Scammell 2003).

The MLST (Thorpy 1992) involves placing individuals in a quiet, dark room for 20-30 minutes at two-hour intervals over a 24-hour period. The participant is attached to an EEG machine. Sleep onset within five minutes during the day is defined as "pathological sleepiness".

One case of a 23-year-old builder with narcolepsy had a sleep onset time in the MLST of fifteen seconds, and immediate REM sleep (Zeman et al 2001).

### ii) Cataplexy

This is muscle weakness leading to knees buckling, jaw sagging, head dropping, and sometimes resulting in collapse. It is usually brought on by strong emotions. Full consciousness, however, is maintained during such an attack (Taheri and Mignot 2002).

### iii) Hallucinations

These may be either at the onset of sleep (hypnagogic) or on waking (hypnopompic). The latter are a better indicator of narcolepsy (Ohayon et al 1996).

### iv) Sleep paralysis

Feeling awake but paralysed when, in fact, still asleep.

There is also "secondary narcolepsy" which occurs after damage to the hypothalamus, while cataplexy can develop after strokes or tumours in the brainstem (Scammell 2003).

Sufferers do not sleep overall more than the

average, but "they are unable to keep the normal boundaries of wakefulness, NREM sleep and REM sleep" (Mahowald and Schenck 2005). For example, automatic behaviour (eg: unwrapping a sweet, throwing the sweet away and putting the wrapper in the mouth) is a mixture of wakefulness and NREM sleep. While sleep paralysis and cataplexy are the combination of REM sleep and wakefulness.

### 9.3 CAUSES OF NARCOLEPSY

An early psychoanalytic explanation was narcolepsy as avoidance behaviour. Based on Freudian ideas, when something unpleasant (usually from the unconscious mind) was potentially reaching the conscious mind, the Ego would produce the sudden sleepiness to avoid this happening.

More recently, biological explanations have been found. Initially questions focused upon whether the falling asleep was due to an increased sleep drive or due to problems with the arousal system (Scammell 2003). The latter is believed to be the case today.

A genetic link was first observed in litters of Doberman pinchers and Labrador retrievers in the 1970s by sleep researcher, William Dement at Stanford University (eg: Mitler et al 1976). Narcolepsy was evident in these dogs as a recessive gene (known as "canarc-1"; Lin et al 1999). This requires the gene from both biological parents in order for a condition to be inherited.

A narcoleptic dog-breeding colony was set up at Stanford University. This is used to test drugs to combat narcolepsy using the food-elicited cataplexy test (FECT) (Taheri et al 2002). The presenting of food excites the dog which then experiences a bout of cataplexy. The speed of onset of cataplexy and the length of the bout can be measured as evidence of a medication's efficacy.

It seems to be related to a gene on chromosome six (Mignot 1998) for humans. Ninety percent of individuals with narcolepsy appear to be carrying this particular gene which usually is in less than one-third of the general population. However, the risk of inheriting this gene is low (1-2%) (Mahowald and Schenck 2005).

In a MZ (monozygotic) twin study, the concordance rate was around 25% (ie: if one twin had narcolepsy, the other twin also had it in a quarter of cases) (Mignot 1998).

Genetic mutations seem less important for humans compared to animals (Taheri et al 2002).

Also two genes have been found that protect against



the condition (Taheri and Mignot 2002).

### 9.3.1 Orexins

Other animal studies have found a link to the "sleep switch" (areas of the brainstem; Saper et al 2005).

The flipping of the "sleep switch" inappropriately could cause narcolepsy. Recent research has found that this switch is controlled by neurochemicals called orexins or hypocretins (Saper et al 2005).

Two separate research groups were studying this molecule in 1998, and gave it different names: hypocretin (de Lecea et al 1998) and orexin (Sakurai et al 1998). Orexin is now known to exist in two forms, A and B (or hypocretin 1 and 2) (Scammell 2003).

The relationship to sleep was a chance discovery as the focus of research was upon orexin and appetite behaviour by the latter research group. They produced a genetically engineered mouse (known as a knockout) which had no orexin-producing genes, and thus no orexin in the brain (Whatson 2004).

Orexin is only made in the hypothalamus (Siegel 2000). Orexin neurons are active during wakefulness and movement, and they inhibit REM sleep. For example, injections of orexin into the locus coeruleus (brainstem) of sleepy animals increased wakefulness (Hagen et al 1998).

Human sufferers of narcolepsy (with cataplexy) were found to have less orexin neurons (based on post-mortems) (eg: Thannickal et al 2000). Orexin appears to encourage arousal.

One suggestion is that orexin neurons are reduced because the immune systems of narcoleptics are attacking them mistakenly as foreign substances (autoimmune disease) (Siegel 2000).

Smith et al (2004) took a particular antibody marker, that may be involved in autoimmune conditions, from nine narcoleptic and nine non-narcoleptic humans and injected it into healthy mice. The marker from the narcoleptics produced an immune response in the mice, but not from the controls.

But how to explain the fact that there is a selective loss of orexin neurons, and not any damage to neighbouring neurons? (Taheri et al 2003).

The problem with establishing the autoimmune hypothesis is that many individuals are diagnosed with narcolepsy well after the onset of the condition, and any evidence of autoimmune activities may have disappeared (Taheri et al 2002).

## 9.4 COGNITIVE EFFECTS OF NARCOLEPSY

Sleep deprivation generally leads to cognitive problems. For example, reduced recall of nouns by half after 35 hours awake (Drummond et al 2000). Narcolepsy is not the same as sleep deprivation, but does the disruptions in sleeping patterns cause similar cognitive problems?

Naumann et al (2006) set out to answer this question in two experiments. The first experiment looked at attention and concentration in fifteen narcolepsy sufferers and fifteen healthy controls in Germany. Attention was measured in a number of ways over the one and half hour experiment:

- Attention span - listening to a string of digits and repeating them, sometimes backwards;
- Alertness - reaction time to press a computer key at the appearance of a visual target on the screen;
- Sustained attention/concentration - marking all the cases of one letter (eg: "d") in a passage of prose for five minutes;
- Divided attention - watching and listening for the occurrence of certain visual targets or acoustic tones at the same time;
- Working memory - pressing a key if a number appears that is the same as a number two occasions previously.

The individuals with narcolepsy differed in some ways to the controls suggesting slower information processing on complex tasks. Table 9.1 summarises the findings of the different tasks.

<u>TASK</u>	<u>FINDINGS</u>
Attention span	No difference in number of digits repeated correctly; mean 6.5 for both groups
Alertness	No difference in reaction time; around 200 milliseconds each
Sustained alertness	Narcolepsy group marked significantly less letters in time allowed
Divided attention	No difference in hits and errors, but narcoleptics significantly slower reaction time
Working memory	Narcoleptics significantly longer reaction time

Table 9.1 - Summary of results on different tasks to measure attention.

The second experiment studied memory with forty-two German participants (half narcolepsy sufferers). Memory was again tested in a number of different ways over the ninety minute experiment:

- Verbal memory - free recall immediately and thirty minutes after reading a passage of prose;
- Visual memory - draw geographical patterns from memory after seeing for ten seconds (Benton Revised Visual Retention Test; Benton 1955);
- Hayling Sentence Completion Test (HSCT; Burgess and Shallice 1997) - complete the last word of a sentence with either an obvious or an unusual word;
- Verbal fluency - generate examples of categories (eg vegetables, country names).

The narcolepsy group did significantly poorer on the verbal memory test (both immediate and delayed recall) (approximately 15% less); took significantly longer on the HSCT; and produced significantly less words on the verbal fluency test, but there was no difference in visual memory. Overall, the researchers concluded that there existed a "mild verbal memory deficit in patients suffering from narcolepsy" (p334).

An explanation for the cognitive differences by narcolepsy sufferers could be that they have "a reduction of available cognitive processing resources because of the need for continuous allocation of resources to monitoring and maintenance of vigilance" (Naumann et al 2006 p329). This would make sense if a person keeps falling asleep spontaneously.

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## **10. EXTREME INSOMNIA: FATAL FAMILIAL INSOMNIA**

- 10.1 FATAL FAMILIAL INSOMNIA
- 10.2 REFERENCES

### **10.1 FATAL FAMILIAL INSOMNIA**

Many people suffer from problems in falling asleep (between 9-20% of general adult population; Taheri and Mignot 2002). Eventually such individuals do fall asleep, but what if a person could not sleep at all?

A rare type of insomnia is Fatal Familial Insomnia (FFI), which is a genetic condition (on chromosome 20; Taheri and Mignot 2002) leading to degeneration of the thalamus due to the prion protein. It is probably a form of transmissible spongiform encephalopathy (TSE) (eg: Creutzfeldt-Jakob disease: CJD) (National Institute of Neurological Disease and Stroke 2005).

FFI has highlighted the role of the thalamus in regulating the sleep/wake cycle and circadian functions (Montagna et al 2003).

FFI was first reported in two patients in the 1980s (Lugaresi et al 1986), and then fully observed in three different Italian families (Montagna et al 2003).

The malfunctioning of the thalamus stops sleep, disrupts the circadian rhythms, and produces dementia. The result of the disease is death.

An early symptom is loss of stage 2 and stage 4 NREM sleep (Taheri and Mignot 2002). The latter is also called slow-wave sleep and is the deepest type of sleep. There also changes related to circadian rhythms (table 10.1).

<u>CHANGES IN CIRCADIAN RHYTHMS</u>
<ul style="list-style-type: none"><li>- Loss of circadian changes in, for example, blood pressure and heart rate</li><li>- Higher body temperature "sleeping" than awake (opposite to normal)</li><li>- Increased energy expenditure (ie: metabolic rate increased)</li></ul>



Table 10.1 - Examples of changes in circadian rhythms with FFI.

There are four stages to its progression lasting about eighteen months (Akroush 1996-7):

i) Progressive insomnia including panic attacks and bizarre phobias (approximately four months);

ii) Continuing insomnia with hallucinations, panic, and agitation (five months);

iii) Total insomnia (three months);

iv) Dementia, total insomnia, and death (six months).

FFI does not show itself until later adulthood. In an Italian family, where twenty-nine of 288 relatives were affected, the average age of onset was forty-nine (Akroush 1996-7). Both sexes are affected equally.

Because FFI is a rare genetic mutation, it tends to remain in families as in the Italian case, and a Chinese family (Spacey et al 2004). Genetic clusters of the condition have been found in twenty-seven different countries around the world (Montagna et al 2003).

From the original Italian families, there seems to be a short version (death within nine months of onset), and a long version (thirty months until death). The former version showed EEG patterns of "sub-wakefulness" (drowsiness) interspersed with bursts of REM sleep for seconds. In the long version, the NREM sleep is reduced (Montagna et al 2003).

Sufferers of FFI can be studied in a number of ways (table 10.2).

<u>METHOD</u>	<u>ADVANTAGE</u>	<u>DISADVANTAGE</u>
EEG	Measure electrical changes in brain of live patients	Gives general picture of brain activity only
Neuroimaging	Scans of working of live brain	Not able to know why brain functioning in that way
Post-mortems	Detailed examination of brain including dissecting and testing	Patient not alive

Table 10.2 - Three methods of studying FFI sufferers.

A variation on FFI called Sporadic Familial Insomnia (SFI) has been reported. The seven cases found had no family history of FFI, but showed all the symptoms of FFI (Montagna et al 2003).

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## **11. THE CASE OF KEN PARKS AND "SLEEPWALKING MURDER"**

- 11.1 INTRODUCTION
- 11.2 PHYSIOLOGICAL QUESTION
- 11.3 PHILOSOPHICAL QUESTION
- 11.4 CAUSATION QUESTION
- 11.5 REFERENCES

### **11.1 INTRODUCTION**

In the early hours of 24 May 1987, a man drove fifteen miles across town in Ontario, Canada. he fatally stabbed his mother-in-law and injured his father-in-law, and then gave himself up at the nearby police station. This man was Ken Parks, and he performed all these actions in a state of somnambulism (sleepwalking) (Broughton et al 1991).

In court in 1990, he was acquitted of murder under the defence of automatism (first used in English law in the 1950s; McCall Smith and Shapiro 1997). This legal defence uses the argument that "individuals are not fully accountable for what they do in such states" (Levy and Bayne 2004).

Parks was 23 years old at the time of the murder, and he had a good relationship with his in-laws. He had gambled excessively, embezzled money, and lost his job before the murder.

The murder took place the night before he and his wife were due to visit the in-laws to confess about these events. At 1.30am, Parks fell asleep in front of the television, and then awoke in the house of the in-laws after the murder (Braisby 2002).

Parks had a family history of sleepwalking. On the night in question, he was suffering from sleep deprivation, was stressed, and had done a lot of physical exercise that day (three factors in sleepwalking) (Braisby 2002).

Cases of behaviour while sleepwalking, of which Ken Parks is one of the most extreme, raise three main issues:

- How is the individual able to do something like drive a car while sleeping (physiological question)?
- How can the individual not be accountable for their actions (philosophical question)?
- Where did the motivation come from to kill the in-laws (causation question)?



## 11.2 PHYSIOLOGICAL QUESTION

This is a question of how Ken Parks drove across town while asleep. He must have been awake to do such behaviour (and thus lying about not remembering the murder) says commonsense.

But this assumes that sleep and waking is a simple on/off process. Sleep is a complex phenomenon (eg: REM and NREM sleep), and waking is not one event, but many variations (eg: drowsiness, focused attention). There are many different states of consciousness in the 24 hour period. This is the conscious state hypothesis (Hobson 2005): consciousness changes as the brain state changes during the sleep/wake cycle.

Sleepwalking is an example of a parasomnia, which are complex behaviours that occur unusually during sleep. It happens during deep sleep (stages 3 and 4 NREM sleep), not during light sleep as commonsense would predict.

Studies of sleep and sleep disorders have shown that the normally distinct phases of wakefulness, NREM and REM sleep can overlap and interfere ("state dissociation"; Mahowald and Schenck 2005) (figure 11A). The relevant case here is sleepwalking as NREM sleep mixed with wakefulness.

It is also possible that different parts of the brain are in different conscious states (eg: left occipital lobe EEG showing NREM sleep and right central EEG REM sleep in narcoleptic patient; Mahowald and Schenck 2005).

	intrudes	
NREM →	WAKEFULNESS = automatic behaviour	eg: driving car on familiar road
WAKEFULNESS →	NREM = sleepwalking	
REM →	WAKEFULNESS = cataplexy (loss of muscle tone)/"waking dream"	
WAKEFULNESS →	REM = sleep paralysis (feeling awake and paralysed when actually dreaming)	
REM + NREM =	"ambiguous sleep" in narcolepsy	

Figure 11A - Intrusions of different conscious states.

Often there is an abrupt waking (not completely) from deep sleep. So, in a situation like Ken Parks, the brain is enough awake/aware to monitor the driving behaviour, but still asleep in terms of conscious awareness of what doing. This is further developed in the next section in terms of the philosophical question of

such "zombie" states (Braisby 2002).

There are a number of scientific documented cases of complex behaviour during sleepwalking (including playing a musical instrument, and eating). Schenck and Mahowald (1995) detailed a case of a 43-year-old man with a history of lifelong sleepwalking activities including frenzied running and violence. He was reported to have driven a car during a sleepwalking episode ("somnambulistic driving").

### 11.3 PHILOSOPHICAL QUESTION

The commonsense view is that individuals choose to do something or not. This choice (or agency) carries consequences and responsibilities. But Levy and Bayne (2004) saw agency as having four types rather than the simple present/absent distinction:

- i) Deliberative agency - consideration of the consequences before the action is performed;
- ii) Conscious agency - awareness of the decision to do something without complete assessment of the consequences;
- iii) Automatic agency - behaviour that does not require full concentration, usually because it is habit;
- iv) Automatistic agency - "a class of conditions in which one acts without being fully conscious of what one is doing". It can be sub-divided into "global automatisms" (affecting the whole of consciousness) like somnambulism or trance states, and "local automatisms" ("a description of consciousness and control over a particular kind of action"); eg: "automatic writing".

Levy and Bayne (2004) further distinguished these four types of agency in three respects:

- Deliberation - opportunity to reflect upon the actions;
- Character - actions as a reflection of a person's character;
- Control - ability to control behaviour.

Table 11.1 summarises the different types of agency.

<u>TYPE OF AGENCY</u>	<u>EXAMPLE</u>	<u>DELIBERATION, CHARACTER, CONTROL</u>
DELIBERATIVE	Decide to leave job after careful consideration	Yes to all 3
CONSCIOUS	Choose to do something but more "spur of moment"	Some deliberation; yes to other 2
AUTOMATIC	Driving car along well known road	Limited deliberation; control if required
AUTOMATISTIC	Sleepwalking	No to all 3

Table 11.1 - Four types of agency.

Putting these ideas together for Ken Parks, somnambulism is a global automatism that "leaves the individual confused and without the resources of conscious control" (Levy and Bayne 2004). In this state, Parks acted out of character (he had no history of violence), with no control over his behaviour, and no ability to reflect upon the action.

The difference between automatistic agency and automatic agency is that with the latter, the individual can quickly resume conscious control. Automatic agency involves indirect monitoring. Neither of these occur with automatistic agency.

In the legal (and moral) sense, Parks therefore has no responsibility for his actions. This type of event opens up many questions about consciousness and responsibility which normally go unaddressed.

#### 11. 4 CAUSATION QUESTION

Even if Ken Parks could drive across town and commit murder while asleep, and he was not legally responsible because of the automatic nature of the behaviour, why did he do it?

Parks himself had no explanation. Any suggestion must be speculation about the motives. Psychoanalysis has a possible explanation.

It is argued that during automatic actions, the individual is in a form of autopilot controlled by non-conscious parts of the brain. In psychoanalysis, this would be the unconscious mind (and the Id) (Freud 1923). Here live the "true" desires that are not acceptable in society. In normal consciousness, the unconscious desire to harm his in-laws is kept in check by the Ego, which also keeps such knowledge from the conscious mind. This

is why Parks would not be aware of his desire to harm the in-laws. In Freudian theory, individuals do not consciously know such things, it is not that they are lying. Defence mechanisms used by the Ego, like repression, keep such information from the conscious mind (Freud 1936) (figure 11B). This is crucial because conscious knowledge of these unacceptable desires could lead to major psychological problems.

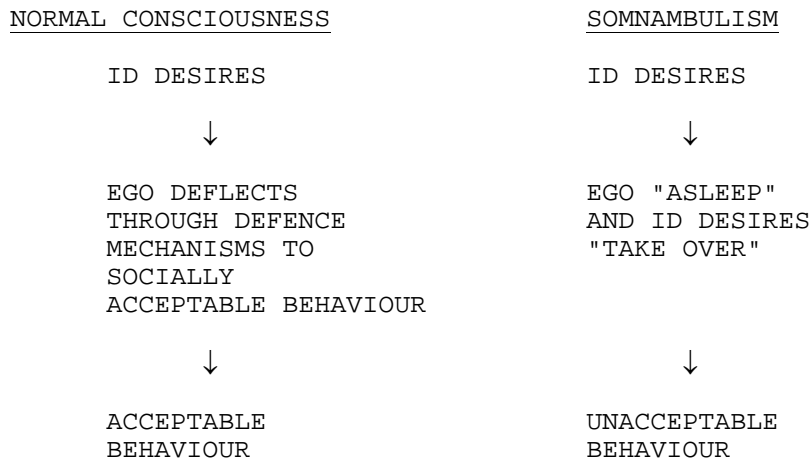


Figure 11B - Id and ego in two states of consciousness.

In the state of somnambulism, the Ego is temporarily unable to be in control, and the Id "takes over", leading to Parks acting out the unconscious desires. The conscious mind is protected as afterwards he had little memory of the actions.

Because the murder took place on the night before Parks was going to admit to his in-laws about his gambling and embezzlement, the Id desired to remove the in-laws so he would not have to face the shame. But, if so, why did he only kill the mother-in-law, and strangle the father-in-law unconscious?

Psychoanalysis places great emphasis upon the role of the unconscious mind as the cause of behaviour, but there are many psychologists who disagree (eg: Eysenck 1985).

An alternative explanation for Park's behaviour is that of dissociation: "A person acting in a state of dissociation is not unconscious in the normal sense of the term; he is aware of his surroundings and responds to them, but does not relate these surroundings and his reaction to them to his normal self" (McCall Smith and Shapiro 1997 p48). It is a separation of consciousness as in multiple personalities (Dissociative Identity Disorder), for example, or in hypnosis. But this does

explain why Parks did what he did, it only describes it.

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## **12. SLEEP PARALYSIS: AN UNUSUAL SLEEP DISORDER**

- 12.1 INTRODUCTION
- 12.2 FREQUENCY OF SLEEP PARALYSIS
- 12.3 EXPERIENCES OF SLEEP PARALYSIS
- 12.4 REFERENCES

### **12.1 INTRODUCTION**

Imagine opening your eyes shortly before dawn, attempting to roll over in your bed, and suddenly realizing that you are entirely paralyzed. While lying helplessly on your back and unable to cry out for help, you become aware of sinister figures lurking in your bedroom. As they move closer to your bed, your heart begins to pound violently and you feel as if you are suffocating  
(McNally and Clancy 2005 p114).

This is an example of what is experienced in sleep paralysis (SP).

SP is a state of "involuntary immobility" due to the intrusion of wakefulness into REM sleep. The upshot is that individuals perceive themselves as awake but paralysed, when in fact they are dreaming. It is a highly vivid dream, or put another way, it is the "maintenance of REM consciousness when waking" (Girard and Cheyne 2006).

### **12.2 FREQUENCY**

The lifetime prevalence of SP occurring once is between 2.3 and 40%. About 5% of the population have SP with a full range of visual, tactile, and auditory hallucinations (McNally and Clancy 2005).

While individuals who have experienced severe trauma reported a higher rate of SP - eg: 42% among Cambodian refugees (Hinton 2003 in McNally and Clancy 2005). The rate is also higher among narcolepsy sufferers (49%) (Sturzenegger and Bassetti 2004).

There is a very rare version of SP that is inherited by women via the X-chromosome, and it occurs at sleep onset (Shneerson 2005).

For regular sufferers (up to 10% of the general population), SP can occur at the same times during the night (Girard and Cheyne 2006).

Girard and Cheyne (2006) set up an online study with

individuals who had suffered an episode of SP in the previous twelve months (n = 410). For 28% of respondents, SP occurred within the first hour after Bedtime (45% in first two hours), and 26% experienced an episode five hours or more after Bedtime. This is a bimodal distribution.

This study is typical of an increasing number in that the internet is used as the means to collect questionnaire data. There are a number of advantages and disadvantages of using this method (table 12.1).

ADVANTAGES	DISADVANTAGES
<ul style="list-style-type: none"> <li>- Cheaper than postal or face to face-to-face questionnaires</li> <li>- Allows anonymity of respondent apart from email address</li> <li>- Wider geographical sample (anywhere in world)</li> <li>- Individuals can fill out questionnaire in their own time</li> </ul>	<ul style="list-style-type: none"> <li>- Self-selecting sample of those who visited website and signed up for study</li> <li>- Under-representation of individuals from certain backgrounds eg without internet access, poorer, older</li> <li>- No independent confirmation of information</li> <li>- Misunderstanding questions and no interviewer to clarify details</li> </ul>

Table 12.1 - Advantages and disadvantages of internet-based questionnaires.

Girard and Cheyne (2006) were able to draw from 1876 individuals who had visited their website, but the majority (89%) were from North America. The sample of respondents was also biased in Caucasian descent (84%), female (71%), age (mean 31.3 years old), and students (23%).

### 12.3 EXPERIENCES OF SLEEP PARALYSIS

The feeling of being awake but paralysed is frightening, particularly if it is associated with something/somebody (malevolent presence) in the room. A sensed presence in the room is reported thus: "My eyes pop open and I sense something ominous. Then something comes over me and smothers me, as if with a pillow, I fight but I can't move" (internet respondent in Cheyne 2001).

Not knowing that such experience is SP leads the experiencer to search for meaning: "hallucinated intruders menacing the sleeper have been interpreted as male (incubus) or female (succubus) demons, as witches,

and, most recently, as alien beings" (McNally and Clancy 2005 p114).

SP has been proposed as an explanation for many historical and cultural accounts of visitations in the night. These include (McKechnie 1998):

- "Old Hag" in Newfoundland, Canada trying to strangle victims. Described by one respondent as "this old lady, that was sitting on top my chest beating the living daylights out of my head" (Cheyne 2001);
- "Kanashibari" in Japan;
- "Ghost oppression" in China.

In Laos (South East Asia), it is the "Grey Ghost". Chiao Song described the experience thus: "You cannot move at that time, cannot even move your hand or leg or body. They just come and press on your chest and then sit on top of your body. Sometimes you wake up after that goes, but sometimes maybe you deep sleep" (Horizon 1994: transcript p21).

SP has also been suggested as an explanation for modern stories of alien abduction. Pat Cross believed that 25% of individuals, who responded to her newspaper ads about alien abduction experiences, were describing SP: "Well typically that they were in their beds, woke up in the middle of the night, found they were unable to move, frequently described a feeling of being pushed down, or held down by some unseen force. The standard description focused on the eyes as being very big, very dark" (Horizon 1994: transcript p23).

Beliefs and cultural values play a role in whether the SP experience is interpreted as angels or demons, or as aliens.

McNally and Clancy (2005) reported interviews with ten US abductees and compared them to twelve matched controls. Key differences emerged in how the former group interpreted their experiences of SP as shown by significantly higher scores on three questionnaires (table 12.2):

- Dissociative Experiences Scale (DES) (Bernstein and Putnam 1986) - This scale measures feelings of altered consciousness, like not recognising own reflection in the mirror;
- Absorption Scale (Tellegen and Atkinson 1974) - The ability to become absorbed in imagination;
- Magical Ideation Scale (Eckbald and Chapman 1983) - This measures the belief in paranormal phenomena.



<u>QUESTIONNAIRE</u>	<u>ABDUCTEES</u>	<u>CONTROL</u>
DES	8.4	3.3
Absorption Scale	21.6	9.6
Magical Ideation Scale	9.2	2.6

Table 12.2 - Mean scores on three questionnaires.

What is being shown by this research is that individuals interpret the experience of SP differently depending upon their prior beliefs. Individuals who believe in the paranormal and can become absorbed in imagination were more likely to interpret their SP experience as alien abduction today in the US.

Other individuals will interpret the experience differently; eg: dream, haunted by a ghost, or something physically wrong (second study respondents; McNally and Clancy 2005).

Two of Cheyne's (2001) respondents reported: "During my first experiences I believed a ghost was sitting on my back, pressing me down", and "I believed that the devil or an evil spirit had me pinned down and was fondling me" (p144).

The experiencer of SP is convinced that they are awake in all these cases. One interviewee from Newfoundland said: "Oh my eyes are open, yes. I've seen my mother walk in the room when I've had the Old Hag" (Horizon 1994: transcript p22).

The experience interpreted as alien abduction is real enough such that McNally and Clancy (2005) found that their sample showed symptoms of PTSD in relation to it.

Cheyne (2005) used the Waterloo Unusual Sleep Experiences Scale website (<http://watarts.uwaterloo.ca/~acheyne/spquest01.html>) to investigate the types of hallucinations during SP. Between March 2001 and February 2004, there were 12 942 respondents (of which 5799 were used in the study). The type of hallucinations were similar between single or regular SP experiencers.

The types of hallucination in SP can be divided into three (Cheyne 2005):

- Intruder - sense of a threatening presence, even humanoid apparitions and the sense of being touched;
- Incubus - feelings of suffocation, and thoughts of impending death;
- Vestibular-Motor - feelings of floating, flying or falling.

Part of the combination of REM sleep and wakefulness in SP is that a "hypervigilant defensive state" (usually when threatened while awake) clashes with the paralysed muscles of REM sleep (Cheyne 2001). One part of the brain (limbic system) is activating an emergency response while the sleeper is still paralysed. This produces the feelings of terror often associated with SP.

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## **13. SLEEP AND LEARNING**

### 13.1 INTRODUCTION

### 13.2 PROCEDURAL LEARNING

#### 13.2.1 Visual Texture Discrimination

#### 13.2.2 Motor Sequence Task

#### 13.2.3 Motor Adaptation Task

#### 13.2.4 Serial Reaction-Time Task

### 13.3 ODOUR LEARNING

### 13.4 REFERENCES

### **13.1 INTRODUCTION**

The benefits of sleep for memory and learning has only recently become a topic of research interest again since Karni et al (1994). This has become known as "sleep-dependent memory enhancement" (Stickgold 2005). The simple finding was that participants' recall and learning after a night of sleep is better than after the equivalent time awake.

Research has shown that the relationship between sleep and memory is complex, and depends on what is learnt, and the stages of sleep. The studying of sleep and learning and memory makes use of a number of different methods and techniques:

i) Learning experiments - Participants learn a task and then they are retested later after sleep or not;

ii) Molecular studies - The increasing knowledge of genes, particularly from animal studies, has shown that certain genes are active in the hippocampus at night. The hippocampus is a key area of the brain related to learning and memory;

iii) Deprivation studies - Studies, again, on animals, deprived of light, for example, showed that cells in the brain changed during sleep (eg: Frank et al 2001);

iv) Neurophysiological studies - Measurements of brain activity (eg: EEG) showed that cells used in learning were active during sleep (eg: song learning in Zebra finch; Dave and Margoliash 2000);

v) Brain imaging studies;

vi) Dream studies - Subjective reports of dreams suggested that dreams are not just replaying episodic memories of the day, but are involved in learning: for example, Stickgold et al (2000c) analysed the dreams of

participants after playing the computer game "Tetris".

## **13.2 PROCEDURAL LEARNING**

This is the type of learning and memory used in perceptual and motor skills (like driving a car). Generally it improves with practice. It is studied by different tasks in experiments.

### **13.2.1 Visual Texture Discrimination**

This task requires participants to identify diagonal lines among horizontal lines on the computer screen. It is a foreground-background discrimination task. It is the speed of identification that is measured, and this will improve with time.

Performance improves even more with sleep between training and testing compared to waking, but particularly the amount of slow wave sleep in the first quarter of the night (SWS1) and the amount of REM sleep in the last quarter (REM4) (Stickgold et al 2000a).

Calculations can be made by multiplying the percentage of these two types of sleep together. For example, when  $SWS1 \times REM4 = 100$ , improvements in task time was an average of 15 milliseconds compared to 30 msec where  $SWS1 \times REM4 = 200$  (Stickgold 2005).

Conversely, sleep deprivation the night after training stops improvements in performance, and this effect will continue after subsequent normal sleep (Stickgold et al 2000b). For example, the general improvement after three nights of normal sleep after training was around 20 milliseconds, but only an improvement of about five milliseconds for participants who had one night of sleep deprivation followed by two nights of normal sleep (Stickgold 2005).

The improvement in performance due to sleep does not stop after one night, but continues for 48-96 hours afterwards if normal sleep occurs. For example, after the first night of sleep, there is an improvement of about 10 milliseconds, another 5 milliseconds after the second night of sleep, and the same for the third and fourth night (and then the benefits peak) (Stickgold 2005).

### **13.2.2 Motor Sequence Task**

This type of task includes asking participants to perform a particular sequence on a computer keyboard (eg: 7-9-A-Q-1), and their speed and accuracy are measured.

Sleep after learning produces a better performance than remaining awake for the same period.

Also a ninety-minute daytime nap after training produced a 16% improvement in performance (Walker and Stickgold quoted in Stickgold 2005). Again improvements continued after the second and third nights of sleep, and were linked to a particular stage of sleep - in this case, stage 2 NREM in the last quarter of the night (Stickgold 2005).

Stickgold et al (2000b) found that sleep also reduced the number of errors (ie: increased accuracy) by 36% on these types of tasks compared to an increase of 9% for errors after twelve hours of daytime waking.

### **13.2.3 Motor Adaptation Task**

This type of task involves activities like moving a cursor on a screen to draw a line, but the cursor moves independently of the controls. The participants must adapt to the unusual behaviour of the cursor. The number of errors are usually measured, and these are reduced by sleep after training. Improvements in performance are linked to slow wave sleep (Ghilardi et al 2000).

### **13.2.4 Serial Reaction-Time Task**

Participants are instructed to press keys on a computer in a particular sequence, which has a complex order that the participants are not told about. PET scans of sleeping individuals found that during REM sleep, regions of the brain active during learning of the task were also active (Maquet et al 2000).

Sometimes the participants are warned that there is a pattern to the sequence of keys. They showed improvements in performance after sleep. But, interestingly, participants who did not know about the pattern showed improvements during waking as well as after sleep (Robertson et al 2004).

## **13.3 ODOUR LEARNING**

A different area of learning that has gained interest recently is odour recognition. Killgore and McBride (2006) tested odour recognition accuracy in thirty-eight adults volunteers at the Walter Reed Army Institute of Research in Maryland, USA. Participants were tested on recognition of forty odours after sleep and after 24 hours of wakefulness. Recognition ability declined with increasing sleepiness.

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## **14. STAYING AWAKE NOT OUT OF CHOICE**

### 14.1 INSOMNIA

#### 14.1.1 Anti-depressants and Insomnia

### 14.2 OCCUPATIONAL DEMANDS: HOSPITAL STAFF

### 14.3 REFERENCES

### **14.1 INSOMNIA**

About one-third of people have some form of insomnia, of which half of those are chronic (Taheri and Mignot 2002).

Insomnia occurs in three forms (either independently or together):

- Onset - unable to fall asleep
- Maintenance - waking frequently
- Termination - waking early and unable to return to sleep.

One problem with insomnia is that individuals report less sleep than they actually had. This is because individuals notice being awake more. For example, 83% of insomniacs and 50% of controls reported being awake when aroused from EEG-verified NREM sleep (Moore et al 1981). The perception of not sleeping is more distorted with middle of the night awakenings (Bonnet and Moore 1982). This sleep state misperception is called "Paradoxical Insomnia" in the International Classification of Sleep Disorders (American Academy of Sleep Medicine 2005).

There is a rare opposite condition where individuals perceive themselves as asleep when they are not ("reverse sleep state misperception") (Attarian 2007).

Semler and Harvey (2005) led insomniac students to believe that they had a bad night's sleep when they had not. These students showed behaviour as if they had not slept well (eg: negative thoughts, feeling sleepy). The researchers argued that it is the anxiety about not sleeping well that can be as important as actually not sleeping well for some insomniacs.

#### **14.1.1 Anti-depressants and Insomnia**

Depression is one of the causes of insomnia, and insomnia itself can cause depression (Mahowald and Schenck 2005). But anti-depressants also have an effect upon sleep (in a different way). They suppress REM sleep (Vertes and Eastman 2000). Though there are three main

types of anti-depressants, they all have this same effect.

- Monoamine oxidase inhibitors (MAOI)

Individuals taking this type of anti-depressant for a long period of time can gradually end up with no REM sleep. In fact, one case study found a patient taking phenelzine with no REM sleep for 226 days. But there seemed to be "no adverse psychological effects" (Wyatt et al 1971). However this study was only with seven individuals.

- Tricyclic anti-depressants (TCA)

TCAs reduce the amount of REM sleep by an average of 50%, but do not eliminate it. There is an immediate loss of REM sleep for the first few nights after starting the TCA, and this evens out (Vertes and Eastman 2000).

- Selective serotonin reuptake inhibitors (SSRI)

The newest type of anti-depressants, SSRIs produce a similar effect to TCAs, but slightly stronger. For example, Staner et al (1995) compared the long-term use of paroxetine (SSRI) and amitriptyline (TCA) in depressed individuals. The former showed a reduction in REM sleep of about half, and the latter one-third.

## **14.2 OCCUPATIONAL DEMANDS: HOSPITAL STAFF**

A number of jobs require individuals to work long hours, but for hospital staff, this involves sleep deprivation or disturbed/limited sleep (particularly when on call). The loss of a small amount of sleep is recoverable, but the effects of chronic partial sleep loss are cumulative (Veasey et al 2002).

Veasey et al (2002) reviewed thirty-three studies of sleep loss and hospital interns and residents (house staff). These are junior staff who work long hours including multiple nights on call.

For example, one study found that surgical residents after sleep loss made more mistakes and took longer to complete tasks. While naps of thirty minutes every three hours was an effective counter-measure to sleep loss. Table 14.1 shows some examples of studies included in the review.



<u>STUDY</u>	<u>DETAILS</u>
Grantcharov et al (2001)	14 surgical residents: 2- fold increase in procedural errors and 38% increase in time to complete laparoscopic tasks after sleep loss
Friedman et al (1971)	14 medical interns' interpretation of electrocardiogram: 9 errors after 2 hrs sleep vs 5 after 7 hrs sleep
Lingenfelser et al (1994)	40 junior doctors showed significant impairment on Stroop test after sleep loss (24 hrs on call vs 6 hrs sleep)
Hart et al (1987)	30 medical interns less story recall (90% vs 77%) after night on call than after sleep

(After Veasey et al 2002)

Table 14.1 - Four studies showing negative effects of sleep loss on medical staff.

In table 14.1, two of the studies used real-life tasks as the measure of performance, and two used common psychological tests. There are advantages and disadvantages to using either type of test (table 14.2).

	<u>REAL LIFE TASKS</u>	<u>COMMON PSYCHOLOGY TESTS</u>
ADVANTAGE	Ecologically valid measure of effects of sleep loss on medical staff	Standardised test with comparable scores to other groups
DISADVANTAGE	Sometimes difficult to measure accurately if hospital busy	Participants may not take as seriously as real life tasks

Table 14.2 - Advantages and disadvantages of using different measures of behaviour.

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