Sleep Disorder Following Traumatic Brain Injury: An Investigation of the Predictors of Sleep Disorder 12 Months or More Following Traumatic Brain Injury.

Margaret C. Couston

Doctorate in Clinical Psychology
Edinburgh University and East of Scotland Training Course

2002
For
"The Telly Man"

My Dad
Robert Brown Couston
(1942 – 2000)

I got there, eventually!
ACKNOWLEDGEMENTS

I would like to thank my supervisor and colleagues from the Neuropsychology department of Astley Ainslie Hospital: Anthony Prior for his helpful suggestions and for providing access to subjects and to Sue Turnbull, Andrew Harrison and Marion Murray for their comments on earlier drafts. I am also grateful to Ken Laidlaw for his assistance and academic supervision of this work throughout the last year. Thanks must go to Dr. Brian Pentland and Dr. Stephen Smith, consultant neurologists at The Charles Bell Pavilion, Astley Ainslie Hospital, for advice and help recruiting participants. I am, of course, indebted to all the individuals who agreed to take part and who were so willing to help. Finally, I am very grateful to my partner Gavin, for his constant patience and support, without whom the last three years would have been impossible.
DECLARATION

I hereby declare that the work enclosed herein is my own except where otherwise stated.
Abstract

Objective: The purpose was to identify the characteristics predictive of sleep disorder in a sample of traumatic brain injury (TBI) patients, twelve months or more following trauma.

Design: A between-subject design explored the relationship between the participant’s sleep disturbance and the severity of TBI. A within-subjects design investigated reliability of the sleep disorder self-report and explored differences between ratings of the participant and a significant other. In addition, qualitative analysis based on content analysis, investigated themes relating to sleep experiences generated by a semi-structured interview.

Participants: Eighteen males and 15 significant others were recruited from patients who were admitted to The Scottish Brain Injury Unit (SBIRS) between June 2002 and June 1997. The participants were predominantly in the severe TBI category.

Measures: The following were the factors measured, and the instruments used for this purpose: sleep quality (The Pittsburgh Sleep Index; PSQI), psychological distress (Hamilton rating Scale for Depression, HRSD; Hospital Anxiety and Depression Scale, HADS) and fatigue (Bentall fatigue inventory and a Visual Analogue Scale, VAS-F). Significant others completed only the PSQI.

Results: Fifty per cent of the sample reported poor sleep quality and 22 per cent of the participants had insomnia. Among the demographic, affective and injury variables examined, the strongest relationship with sleep quality was linked to depression. The significant other ratings were no different to the participant’s self-ratings. Interestingly, sleep quality rather than TBI severity appeared linked to depression.

Conclusions: This sample has reported slightly lower rates of sleep disturbance than a comparable post acute population but this is still more than double the rate of sleep disturbance in the normal population. There was evidence that links may be between poor sleep quality and depression, perhaps even depression secondary to insomnia, rather than TBI. In addition TBI is considered as a model for depression. The importance of evaluating treatments for insomnia in this group is discussed.
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1. INTRODUCTION

1.1 WHAT IS TRAUMATIC BRAIN INJURY?
Traumatic brain injury (TBI) is a neurological condition of relatively high incidence, particularly in males; head trauma is two or three times more common in males than in females (Rosenthal, Griffith, Kreutzer & Pentland, 1999). Head injuries most commonly occur in individuals up to twenty-four years of age. Rimel, Jane & Bond (1990) showed that two age groups are at risk of head injury. They are the ten to thirty year-olds and those over the age of sixty-five years. TBI's are often sustained as a result of road traffic accidents, falls and sports related injuries (Echemendia & Julian, 2001). In Scotland, in a study of men between fifteen to twenty four years of age, who were treated in accident and emergency departments, it was found that assaults were twice as common as road traffic accidents (Jennet & MacMillan, 1981). Studies from the United States reported motor vehicle accidents as the predominant cause of injury (Rimel et al. 1990).

McGregor & Pentland (1997) reported that between two to three hundred people per 100,000 in the UK require hospitalisation each year after sustaining a TBI. These figures suggest that TBI is a problem of great concern to public health. Some researchers have indicated that the incidence of TBI is at least three times greater than schizophrenia, mania and panic disorders (Yudofsky, Silver & Hailes, 1992). In America, TBI results in significant morbidity and cost; studies have estimated the cost as high as thirty-seven and a half billion dollars a year (Arciniegas, Adler & Topoff, 1999).

A recent cohort study (Thornhill, Teasdale, Murray, McEwan, Roy & Penny, 2000) provided a picture of current features of the Scottish population. They estimated that in Glasgow, annually, one thousand and four hundred young people and adults are still disabled one year after head injury. The study reported Scottish figures for head injury requiring hospitalisation: one thousand two hundred and fifty five (forty two per cent) were men aged forty years or less, five hundred and seventy five (nineteen per cent)
were men and women aged sixty five years or more, and most (ninety per cent) were classified as having a mild injury. The most common causes of injury were falls (forty three per cent), assaults (thirty four per cent) and road traffic accidents (ten per cent); alcohol was often involved (sixty one per cent) and one quarter reported treatment for a previous head injury. Severity ratings (Glasgow Coma Scale) for this sample were reported as ninety per cent mild; five per cent moderate; three per cent severe and a further two per cent unclassified. The characteristics of this cohort agreed with previous studies (McMillan, Strang, Jennet, 1979; Miller & Jones, 1985).

Thornhill et al. (2000) obtained outcome information on patients at follow-up and these figures indicated that around twelve per cent were dead or in a vegetative state one year following injury, twenty one per cent had a severe disability, twenty six per cent a moderate disability and about thirty nine per cent went on to have a good recovery. The outcomes for these patients related to initial severity of head injury: dead or vegetative, mild (eight per cent), moderate (twenty two per cent) and severe (twenty nine per cent); severe disability: twenty per cent, twenty two per cent and twenty nine per cent (respectively); moderate disability: twenty eight per cent, twenty four per cent and nineteen per cent, and good recovery: forty five per cent, thirty eight per cent and fourteen per cent.

1.1.1 Brain Injury Severity
The primary area responsible for conscious function (sleep versus wakefulness) is generally reported to be in the part of the brain named the reticular formation. Any damage to the brainstem at the reticular formation may lead to a lowering of conscious levels. Additionally, bruising and swelling cause intra-cranial pressure and compression of the brainstem at the foramen magnum consequently lowers conscious levels. The extent of the damage must be in some way assessed and monitored. If the brain swelling is life threatening, then intra-cranial pressure needs to be monitored invasively, usually in an intensive care unit. Although radiological scanning procedures (CT, MRI) can provide objective indicators of intra-cranial pressure after a brain injury, behaviour has been regarded as the most important measure of the integrity of the central nervous
system (Kolb & Wishaw, 1996). In the immediate post injury period there are two common measures of behaviour: coma and amnesia, the Glasgow Coma Scale (GCS) and Posttraumatic amnesia (PTA), respectively.

The GCS was designed to provide an objective indicator of the degree of unconsciousness (Jennet & Teasdale, 1977; Teasdale and Jennett, 1974). The coma scale measures severity from three to fifteen on an index of wakefulness that falls within the three categories: 1) eye opening; 2) motor response and 3) verbal response (see Table 1.1 below). A score of less than eight meets criteria for a severe head injury, nine-to-twelve is generally considered as moderate, Thirteen and above is a mild head injury. The majority of brain injuries are mild (Rosenthal, et al, 1999)

**TABLE 1.1 Glasgow Coma Scale**

<table>
<thead>
<tr>
<th>GCS Score</th>
<th>Severity</th>
<th>Mortality</th>
</tr>
</thead>
<tbody>
<tr>
<td>13-15</td>
<td>Mild</td>
<td>4%</td>
</tr>
<tr>
<td>9-12</td>
<td>Moderate</td>
<td>4%</td>
</tr>
<tr>
<td>3-8</td>
<td>Severe</td>
<td>40%</td>
</tr>
</tbody>
</table>


PTA is another measure of severity of injury first suggested by Russell in the 1930s. PTA is considered to last from the time the injury took place to the time when the person has full and continuous awareness (Wilson, Shiel, Watson, McLellan, 1994). Very mild concussion would be indicated by a PTA measure of less than ten minutes, mild by a PTA of ten to sixty minutes, moderate by a PTA of one hour up to a day, severe would be greater than one to seven days and very severe by a PTA of more than seven days (Kolb & Wishaw, 1996, p575).

1.1.2 Neuropathological Processes Associated with Traumatic Brain Injury

Brain injury, characterised by structural damage and neurological dysfunction lasts from hours to weeks or years after the initial insult. The brain injury is a combination of
mechanical forces and physiological events often referred to as mechanism of injury. Mechanism of injury can be described as the relationship between the impact of mechanical forces to the head and the resultant physical and physiologic effects on the brain. As a result of initial mechanical forces a cascade of neural and vascular events occur, which lead to the clinical syndrome of TBI (Graham, 1999).

In a TBI, damage to the brain originates by way of three main factors: 1) diffusion of energy from the impact, 2) shearing and stretching of neurones, and 3) focal damage at the injury site. All the following sequelae are possible: contusions (tears); haemorrhaging and haematomas (bleeding and bruising) of the brain underneath the site of the impact (coup injury); opposite sided contusions (contrecoup injury); contusions of the orbital surfaces of the frontal lobes and the tips of the temporal lobes are particularly common. The frontal lobes and anterior temporal lobes are particularly vulnerable to contusions, haemorrhages and haematomas associated with acceleration and deceleration forces (Lezak, & O'Brien, 1990). Long-term consequences include shrinkage of the brain and corresponding enlargement of the ventricular system, although in many moderate and severe TBI’s CT scanning does not demonstrate an abnormal appearance.

Diffuse axonal injury (DAI) is non-focal damage from shear-strain effects on neural pathways and is associated with obstructed blood flow and oedema or swelling of the brain. DAI is the predominant mechanism of injury in forty-to-fifty per cent of TBI’s requiring hospital admission in the US (Meythaler, Peduzzi, Eleftheriou & Novack, 2001):

“The pathology of DAI in humans is characterized histologically by widespread damage to axons in the brain stem, parasagittal white matter of the cerebral cortex and corpus callosum and is consistent with features of TBI.” (Meythaler et al. 2001,p1462).

DAI is believed to be present in all road traffic accidents (RTAs) where the patient has lost consciousness (Graham, 1999). DAI is most frequently associated with coma of
immediate onset after brain injury, but the diagnosis of DAI can only be established at autopsy (Meythaler et al., 2001). DAI has also been associated with sports injuries where there is a risk of high speed collisions (e.g. football, rugby, hockey) and these athletes have been reported as suffering many of the same medical and neurocognitive deficits as those involved in high speed RTAs (Powell & Barber-Foss 1999; Echemendia & Julian, 2001).

1.1.3 Common Complaints Following Traumatic Brain Injury
TBI is highly variable in its long-term effects and although a common neurobehavioural syndrome cannot be identified, in individual cases it can be associated with a range of cognitive impairments (Dikmen, Reitan & Temkin, 1983). Bigler (1990) stated that neurological consequences depend on the severity of the injury. The most common and reliable complaints following traumatic brain injury are of poor concentration and problems with memory (Dikmen, Machamer, Winn & Temkin, 1995). Other generalisations are difficult because the nature and severity of the brain damage will be different in any two patients, e.g. focal damage may lead to specific symptoms; for instance, damage to the left-hemisphere language areas may cause aphasia. Many studies suggest that traumatically brain-injured patients are more handicapped by personality and behavioural disturbances than by cognition and physical disabilities (Lezak & O’Brien, 1990). In addition, neurobehavioural impairments were more common in patients after TBI with sleep complaints that in those who did not report sleep disturbances, and occupational outcome was poorer (Cohen, Oksenberg & Snir, 1992).

Frontal lobe damage is associated primarily with executive dysfunction: in severe injuries of the frontal lobes the dysfunction can be quite debilitating, as the individual experiences greatly impaired flexibility in problem solving and adaptability (Lezak, 1995). Damage to the frontal lobes has been associated with disturbance of fine movements (Kuypers, 1981), loss of strength (Leonard, Jones & Milner, 1988), poor movement planning (Roland, Larsen, Lassen & Skinhoj, 1980); poor voluntary eye gaze (Guitton, Buchtel & Douglas, 1982); loss of divergent thinking with reduced spontaneity (Jones-Gotman & Milner, 1977) and poor strategy formation (Shallice & Evans, 1978).
In addition, risk taking and rule breaking (Miller, 1985), impaired associative learning (Petrides, 1982), poor temporal memory (Milner, 1964), impaired social behaviour (Blumer & Benson, 1975), altered sexual behaviour (Walker & Blumer, 1975) and impaired olfactory discrimination (Potter & Butters, 1980) were also found following frontal lobe damage.

Temporal lobe damage is associated with the loss of certain functions that can have devastating consequences for behaviour. There may be an inability to perceive or to remember events, including language, in the environment. There may be a loss of normal affective response. Major symptoms associated with temporal lobe damage are: disturbance of auditory sensation (Vignolo, 1969; Hécaen & Albert, 1978); disturbance of selection of visual and auditory input (Sparks, Goodglass & Nickel, 1970); disorders of visual perception (Milner, 1968); disorders of auditory perception (Samson & Zatorre, 1988); impaired organisation and categorisation of material (Read, 1981), poor contextual use (Milner, 1958), disturbance of language comprehension (Hécaen & Albert, 1978), poor long-term memory (Blumer & Benson, 1975), changes in personality and affect (Pincus & Tucker, 1974) and changes in sexual activity (Blumer & Benson, 1975).

Mild head injuries, regardless of foci, are most commonly associated with memory and attention deficits (Lezak, 1995). Speeded performance is also likely to be adversely affected (Putnum & Fichtenberg, 1999). In most cases of moderate head injury, symptoms can vary widely: with most individuals continuing to experience significant impairment three months after the injury (Lezak, 1995) and Dikmen et al. (1995) found significant impairments one year after injury. However, this finding was largely related to the severity of the injury. Nevertheless, in neuropsychological terms even a mild head injury can cause a high level of cognitive impairment particularly when an individual has to function at consistently high levels (Rosenthal, Bond, Griffith, & Miller, 1999).
1.1.4 Traumatic Brain Injury and Co-morbidity

Psychiatric syndromes are common in patients with TBI. Patients may develop typical disorders of mood, anxiety or psychosis, in addition to changes in personality and cognition (Lezak & O'Brien, 1990).

The Diagnostic and Statistical Manual of Mental Disorders, fourth edition (DSM-IV; APA, 1994) has described organic personality disorder (OPD) following TBI as a personality change occurring secondary to a general medical condition. The emotional and personality changes, which may include neurosis and psychotic disorder, are not necessarily associated with severe cognitive impairments (Franulic, Horta, Maturana, Scherpenisse & Carbonell, 2000). In a recent study, Franulic et al. (2000) reported that thirty two per cent of their sample met the criteria for diagnosis of OPD. They also reported a clear preponderance of frontal lesions in both groups (those with both TBI and OPD diagnosis and the group with TBI alone): there was no strict relationship with cognitive impairment, but the OPD group showed significantly lower psychosocial adaptation.

During the first year following TBI, a percentage of patients noted changes in their personality. Brooks, McKinlay, Symington, Beattie & Campsie (1987) reported that nearly fifty per cent of family members noted changes in the first three months, and approximately sixty six per cent noted changes in the patient between six to twelve months following the TBI. These personality changes have been described as an increase in irritability, frustration, aggressiveness, egocentricity, impulsivity, impairment of judgment & insight and inappropriate expression of affection. Other researchers have reported that the most salient changes were indicated by lowered tolerance to frustration and increased irritability in trivial situations (Prigatano, 1992). These manifestations tend to generate greater distress in patients after six months, rather than sooner, following TBI (McKinlay & Watkiss, 1999).

The literature reports wide-ranging figures for rates of post TBI depression. Originally post TBI depression was given little attention and was thought to be a minor
consequence of TBI. More recent studies still vary a great deal: two studies reported twenty six per cent to sixty one per cent fulfilling the DSM-III-R criteria for depression respectively (Jorge, Robinson & Arndt, 1991; Van Reekum, Bolago & Finlayson, 1996, respectively). In an American sample Kreutzer, Seel & Gourlay (2001) reported forty two per cent of patients met the DSM-III-R (APA, 1994) diagnostic criteria for major depression. Fatigue (forty six per cent), frustration (forty one per cent) and poor concentration (thirty eight per cent) were the most commonly cited manifestations of depression. Certainly, more rigorous methodology and the use of standardized measures and diagnostic criteria has provided more robust evidence for depression following TBI. However, the more recent literature continues to report huge variations in the occurrence of depression in this population. This may raise doubt regarding the effective use of standardized depression measures with the post TBI population. Perhaps, more sensitive measures require further research and development for post TBI depression.

Psychotic syndromes occur more frequently in individuals who have had a TBI than in the general population (McAllister, 1998). Psychotic disorders following a TBI can present in the period of posttraumatic amnesia, in association with posttraumatic epilepsy, in conjunction with TBI-related mood disorders and as a chronic schizophrenia-like syndrome. In addition, individuals with schizophrenia have a higher frequency of prior TBI than individuals with other psychiatric disorders (Salcido & Costich, 1992).

However, in comparison to evidence for post TBI development of psychoses, Sbordone & Liter (1995) reported that mild TBI does not produce posttraumatic stress disorder (PTSD). This has led to some controversy as to whether PTSD can develop following brain injury. The debate has evolved around the question regarding whether a person can be traumatised after experiencing an event he or she cannot recall. According to Bryant, Marosszeky, Crooks & Gurka, (2000) posttraumatic stress disorder (PTSD) most probably can develop after TBI. They assessed ninety-six TBI patients for diagnosis of PTSD six months after the injury. PTSD was diagnosed, using an interview schedule based on DSM-III-R criteria, in twenty seven per cent of the sample, whilst only
nineteen per cent who were diagnosed reported intrusive memories of the trauma, ninety six per cent reported emotional reactivity. They concluded that the predominance of emotional reactivity and the relative absence of traumatic memories, in patients with PTSD who suffered impaired conscious levels during trauma, suggested that traumatic experiences could mediate PTSD at an implicit level.

This research is supported by the findings of Turnbull, Campbell & Swann (2001) who identified three groups of TBI survivors from an accident and emergency sample. The three groups were those with traumatic memories of the injury, with untraumatic memories and a group with no memories of the index event. Groups with no memories or traumatic memories of the index event reported higher levels of psychological distress than the group with untraumatic memories. Ratings of PTSD symptoms were less severe in the no memory groups compared to those with traumatic memories. Therefore, although the reporting of intrusive memories may be less frequent in this population this does not protect people from developing PTSD following a brain injury. In a recent literature review of PTSD following a TBI, Bryant (2001) posits possible mechanisms of PTSD development after a brain injury. These possible mechanisms involve theories of: implicit processing, fear conditioning, memory reconstruction, post-amnesia trauma, neurobiological factors, posttrauma stressors and impaired treatment stressors.

In a longer-term follow-up study, where the mean follow-up was carried out fourteen years following severe TBI (mean coma was fourteen days), Hoofien, Gilboa, Vakil & Donovick (2001) reported primarily severe long-term psychiatric problems. Depression, anxiety and hostility were among the most conspicuous symptoms. Similar rates of obsessive-compulsive disorder and PTSD symptoms were found. In addition, there were higher than previously reported rates of psychoticism and paranoid ideation. Psychiatric symptomatology was related to objective measures of behaviours (i.e. relatives, reports) and non-acceptance of the disability is related to depression and psychiatric symptomatology.
1.1.5 Long Term Outcomes Following Traumatic Brain Injury

Oddy, Coughlan, Tyerman (1985) investigated the long-term outcomes following TBI. They reported an overall decrease, after six months, in the reported social network and one third of their sample were unable to resume leisure activities. At one to two years they received fewer visits and had fewer social encounters. At seven years, loneliness was reported as the greatest difficulty. However, within that sample, those who returned to work had improved social adjustment.

Brooks et al. (1987) investigated a sample of people who had sustained a very severe TBI (median PTA was three weeks) and found that only one third returned to work in the seven years after their TBI. Brooks et al (1987) found less than one third had returned to work. Amongst those returning to work, their post injury duties were often more basic than previous employment, as a result of their neuro-behavioural difficulties. These neurobehavioural problems were: poor verbal memory, poor sustained concentration and mental speed, behavioural problems, poor emotional regulation, reduced self-care skills (e.g. personal hygiene) and communication problems. Other researchers have reported similar findings following return to work in this population: memory problems (Hoofien et al, 2001), emotional-behavioural problems and lack of insight (Newman, Garmoe, & Ziccardi, 2000; Ponsford, Willmott, Rothwell, Cameron, Kelly, Nelms, Curran & NG, 2000)

Compared to ill or injured individuals who did not have a traumatic injury, TBI survivors may have more areas of physical and emotional challenge and more unmet need. The unmet needs and goals include: work, socialisation, parenting, learning, self-expression, sexuality and leisure activities (Brown & Vandergroot, 1998). The ability to return to work has been a critical factor in predicting quality of life (QOL), social integration and return to home and leisure activities (O’Neil, Hibbard & Brown, 1998). In studies tracking long-term outcomes the social aspect of community integration proved to be significantly related to the level of subjective QOL (Koskinen, 1998). Long-term recovery and the QOL at five years after the injury could be predicted by age, injury severity, artificial respiration, severity (GCS) and length of time in coma and
duration of posttraumatic amnesia (PTA during the initial period of intensive care at the time of the traumatic injury; Asikainen, Kaste & Sarna, 1998).

In their long-term follow-up Hoofien et al (2001) reported that TBI participants frequently suffer permanent difficulties in all areas of their lives. In accordance with other studies of recent TBI’s, they found psychomotor slowness and difficulties in information processing. This study showed that these specific disabilities, which were previously reported shortly after injury, are sustained in the long-term. The employment rate among the participants was higher than premorbidly. Low-level technological and clerical professions were found to be the most frequent fields of employment. Correspondence was found between a fairly good evaluation of the participants’ functioning within the family and high levels of burden as reported by family members. This parity was explained in terms of adaptation of expectations.

Previous studies including Hoofien et al (2001) found ongoing loneliness and social withdrawal to be the major problem in the lives of the persons with TBI. Physical dependence and activities of daily living (ADL) functioning were repeatedly assessed as quite reasonable at the very long-term post-injury stage. Out of four functional areas: vocational, family, social functioning and independence in ADL, the first three were related to psychiatric illness symptomatology and not intellectual abilities (IQ), whereas only independence in ADL was related to IQ and not to symptomatology. The authors conclude that these results support the idea that the main sources of chronic disability are psychiatric and behavioural symptoms rather than cognitive aptitude and abilities.

Hoofien et al. (2001) went on to recommend that long term survivors of TBI (ten to twenty years later) required continued aid particularly in the following domains: emotional reactions (both of the identified patient and family members), the vocational domain (with regard to potential unemployment, low level of employment and associated financial problems), and in the social domain (with regard to potential isolation, withdrawal and narrow support networks). Further long-term outcome studies
are needed in order to validate the limited studies so far. Prospective follow-up studies, using within-participants, repeated measures designs, could provide such evidence.
1.2 SLEEP

1.2.1 Normal sleep architecture.

Sleep is generally regarded as a lowered consciousness state, but it is also a behaviour. Carlson (1994) stated,

“What characterizes sleep is that the insistent urge of sleepiness forces us to seek out a quiet, comfortable place, lie down and remain there for several hours. Because we remember very little about what happens while we sleep, we tend to think of sleep more as a state of consciousness than as a behavior. This change in consciousness is undeniable, but it should not prevent us from noticing the behavioral changes.” Carlson, 1994, p254.

Active brain mechanisms cause us to engage in the sleep behaviour. The stages of non-REM sleep, stages one through to four, are defined by EEG activity. Slow-wave sleep (stages three and four) is the deepest stage. Alertness consists of desynchronised beta activity (13-30 Hz); relaxation and drowsiness consist of alpha activity (8-12 Hz); stage one sleep consists of alternating periods of alpha activity, irregular fast activity and theta activity (3.5-7.5 Hz); the EEG on stage two sleep lacks alpha activity but contains sleep spindles (short periods of 12 - 14 Hertz activity) and occasional K complexes; stage three sleep consists of twenty to fifty per cent delta activity (less than 3.5 Hz); and stage four sleep consists of more than fifty per cent delta activity. About ninety minutes after the beginning of sleep, people enter REM sleep. These cycles of REM and slow-wave sleep alternate within a period of approximately ninety minutes (Wolstenholme & O'Conner, 1961).

The two main theories for why we sleep are (a) that sleep serves as an adaptive response and (b) that it provides a period of restoration (Oswald, 1980; Mendelson, 1987; Borbély, 1986). Sleep as an adaptive response ensures that small vulnerable animals sleep when they are not attending to their survival needs (foraging for food or reproducing); this keeps the animals safe and conserves vital energy resources. The
restorative hypothesis posits that sleep is necessary for the animal to recharge its hypothetical “battery” and to rejuvenate by way of tissue repair and growth. Comparative studies of animals point out that a species’ degree of safety and rate of metabolism are related to the amount of sleep it engages in. This would support the adaptive hypothesis, but the fact that all vertebrates sleep does not. Predators would not need to sleep if its function was solely adaptive: also, some species of dolphin have evolved so that separate brain hemispheres sleep alternately (Carlson, 1994). These studies emphasise the diverse evolutionary developments that have associated survival and fitness with a species-specific sleep pattern.

The effect of several days without sleep is not devastating to humans. The primary finding is intense sleepiness, difficulty with performing tasks that require sustained concentration and perceptual distortions (sometimes mild hallucinations). These effects suggest that sleep deprivation does impair cerebral functioning (Borbély, 1986). Deep slow-wave sleep appears to be the most important stage and it has been suggested its function is recuperative (Oswald, 1980). Animals who are sleep deprived eventually die. Researchers cannot be sure whether it was the stress (caused by the lack of sleep) or the procedure needed to keep the animals awake that caused their premature deaths (Jouvet, 1999).

Horne & Moore (1985) showed that exercise can increase the amount of slow-wave sleep a person receives, but the effect only appears to occur if the brain temperature rises. Cooling the person’s head when exercising can abolish the increased need for slow-wave sleep or warming the head can induce it. Perhaps, then, the fundamental cause is an increase in brain metabolism. Growth hormone is normally secreted only during slow-wave sleep, but the significance of this phenomenon is uncertain (Carlson, 1994).

The function of REM sleep is even less well understood. It may promote vigilance (Snyder, 1966), learning (Greenberg & Pearlman, 1974), deleting useless information from memory (Newman & Evans, 1965; Crick & Michison, 1983), species typical
reprogramming (Jouvet, 1980) or brain development. So far the evidence is inconclusive, although several studies have shown a moderate relationship between REM sleep and learning. Experiments with laboratory animals suggest that REM sleep performs functions that facilitate learning (for a review see Smith, 1985). Experiments have shown that when animals are deprived of REM sleep after participating in a training session, they learn the task more slowly. In addition, when the animals learn a new task, the amount of time they spend in REM sleep increases, as if the learning increases the need for this stage of sleep (Bloch, Hennevin & Leconte, 1977).

In contrast to the animal experiments, studies with humans show that REM sleep deprivation has only a small effect on a person’s ability to learn or remember what was previously learned. However, several studies have reported that children with learning disabilities engage in less REM sleep than children with average intellectual abilities and that intellectually gifted children engage in more still (Dujardin, Guerrien and Leconte, 1990). In addition, Smith & Lapp (1991) found that REM sleep in college students increased during exam time, when they were presumably spending more time learning new information.

Some studies suggest that learning related to emotionally significant material might be affected by REM sleep deprivation. Greenberg, Pillard & Pearlman (1972) showed subjects a film that was anxiety producing (a particularly gruesome circumcision rite performed by members of an American South Sea Island tribe). People who watched the film tended to exhibit less anxiety during the second viewing. The investigators found that those who were permitted to engage in REM sleep between the first and the second viewings showed less anxiety the second time compared with the subjects who were deprived of REM sleep. Breger, Hinter & Lane (1971) additionally reported that the anxiety-producing media affected the dream content of the film viewers. The studies might suggest that REM sleep and perhaps the dreaming that occurs thereafter somehow assist people to process information that has had some emotional impact on the sleeper.
Nevertheless, the calming effects of REM sleep appear to be contradicted by clinical demonstrations of the experiences of people with mental illnesses and their experience of REM sleep. The symptoms of people with severe, psychotic depression are reduced when they are deprived of REM sleep. In addition, treatments that reduce the symptoms of depression, such as antidepressant medication and electroconvulsive therapy, also suppress REM sleep. If REM sleep helps people assimilate emotionally relevant information then why does sleep deprivation relieve the symptoms of people who are suffering from serious emotional disorder? Perhaps because the emotional reprocessing task is so large as to be disturbing and distressing in people who are depressed and/or psychotic.

However, a TBI case reported by Lavie, Pratt, Scharf, Peled & Brown (1984) of a thirty-three-year-old survivor of a shrapnel injury to the head twenty-three years earlier, indicated that whatever the purpose is of REM sleep, it was not necessary for survival. In the sleep laboratory, this man slept an average of four and a half hours per night. On three out of eight nights he engaged in no REM sleep and the average of REM sleep on the other five nights was six minutes. The pons appears to be the brain area that controls REM sleep. The shrapnel in the survivor’s brain damaged the pons, left temporal lobe and left thalamus. The almost complete lack of REM sleep did not appear to cause any serious side effects. After sustaining his injury, he completed high school and went on to become a lawyer.

1.2.2 Biological clocks & physiological mechanisms of sleeping and waking

Our daily lives are characterised by cycles in physical activity, sleep, body temperature, secretion of hormones and many other physiological changes. Circadian rhythms (those with a period of approximately one day) are controlled by biological clocks in the brain. The principal biological clock appears to be located in the suprachiasmatic nuclei (SCN) of the hypothalamus; lesions of these nuclei disrupt most circadian rhythms and the activity of neurons located there correlates with the day-night cycle (Moore and Eichler, 1972; Stephan and Zucker, 1972). Light serves as a zeitgebers (German for “time giver”) for most circadian rhythms (Czeisler, Kronauer, Allan, Duffy, et al., 1989). The sight of
sunlight in the morning is conveyed from the retina to the SCN, resetting the clock to the start of a new cycle, the light synchronising the endogenous rhythm (Ashcoff, 1979).

The fact that the amount of sleep is regulated suggests that sleep-promoting substances (produced during wakefulness) or wakefulness-promoting substances (produced during sleep) may exist. These substances may accumulate in the brain, but attempts to find them have been unsuccessful (Borbély and Tobler, 1989). The release of growth hormone (a hormone that promotes protein synthesis) occurs primarily during slow-wave sleep, drugs that disrupt this hormone also disrupt slow-wave sleep.

Three systems of neurons appear to be important for alert, active wakefulness: the acetylcholinergic system of the pons, the noradrenergic system of the locus coeruleus and the serotonergic system of the raphe nuclei. The particular roles played by each system are still not understood (Carlson, 1994).

Slow-wave sleep occurs when neurons in the basal forebrain become active. These neurons are also involved in temperature regulation, leading some investigators to suggest that an important function of slow-wave sleep is to lower brain temperature (and permit the brain to rest). REM sleep occurs when the activity of the acetylcholinergic neurons in the dorsolateral pons increases; pons, geniculate and occipital waves (PGO waves, the first manifestations of REM sleep), some PGO waves initiate cortical arousal and others produce rapid eye movements (Sakai & Jouvet, 1980). Atonia (muscle paralysis that prevents acting out dreams) is produced by a group of acetylcholinergic neurons located in the subcoerulear nucleus that activate neurons in the magnocellular nucleus of the medulla, which in turn produce inhibition of motor neurons in the spinal cord (Jouvet, 1999; Sakai, 1985). REM sleep is also related to temperature: it occurs only after the brain temperature has been lowered by a period of slow-wave sleep (Jouvet, 1999).

The noradrenergic neurons of the locus coeruleus and the serotonergic neurons of the raphe nuclei have inhibitory effects on the acetylcholinergic neurons of the pons that are
responsible for REM sleep (Lydic, McCarley and Hobson, 1983; Delinger, Patarca & Hobson, 1988). Bouts of REM sleep begin only after the activity of the noradrenergic and serotonergic neurons stops. Whether this event is the only one to trigger REM sleep or whether direct excitation of the acetylcholinergic neurons also occurs is not yet known (Sakai, 1985).

1.2.3 An integrated psychobiological model of normal sleep
Espie (2002) posits an integrated psychobiological model of normal sleep. He presented different factors that appear to play some part in insomnia: human development, quality of sleep, predisposing, precipitating & perpetuating factors, mental disorder, faulty conditioning, poor chronobiological timing, physiological hyper-arousal, cognitive hyper-arousal, dysfunctional thinking and paradox & ironic control. Espie (2002) then integrated this evidence into a conceptual framework; A Psychobiological model of good sleep (see Figure 1).

The psychobiological model of good sleep proposes that good sleep is the natural condition for humans, its “default state”. The biological clock (circadian processes) and physiological mechanisms (homeostatic processes) of sleeping and waking, under normal conditions, default to good sleep, not insomnia. Central tenets of this model are the automatic and well-orchestrated interaction between homeostat and timer, which is linked with the self-perception of good quality sleep (Espie, 2002).

Comparable to other neurobiological / behavioural models the assumption is that good sleep has both functional flexibility (plasticity) and involuntary, habitual (automaticity), formats. Plasticity means that the sleep-wake system has the capability to accommodate to various situational and personal factors. In this default state, variabilities are tolerated and minimised because the homeostat drives sleep related behaviours efficiently.
**Figure 1.** A Psychobiological Model of Good Sleep. Insomnia is proposed as resulting from chronic inhibition of one or more of the component processes.

**Automaticity** means that there is an involuntary basis of a well-adjusted schedule, nightly habits, a stimulus control paradigm driven by conditioned associations, combined with the sleeper’s implicit expectations and assumptions regarding what constitutes a good sleep. However, the good sleeper is considered as effectively passive in this process, internal and external cues being the automated setting conditions for sleep.

"The good sleeper sleeps just as he walks and talks - without thinking about it." (Espie, 2002, p228)

The properties of **plasticity** and **automaticity** are presented as protective properties, which defend basic "core" processes of homeostasis, circadian timing and the associated experience of sleep quality. These properties are maintained by four interacting subsystems: 1) sleep-stimulus control, 2) physiological de-arousal, 3) cognitive de-arousal and 4) daytime facilitation. In essence the model proposes:
that the good sleeper accurately interprets physiological and mental signs of sleep readiness, and once in a bedroom the stimulus environment further reinforces de-arousal. Active information processing recedes as the wake system disengages and the sleep system engages.

1.3 INSOMNIA AND SLEEP DISORDER
The Diagnostic and Statistical Manual of Mental Disorders (DSM-IV) defines primary insomnia as a complaint lasting for at least one month of difficulty initiating and/or maintaining sleep or of non-restorative sleep (American Psychiatric Association, 1994). The International Classification of Sleep Disorders-Revised (ICSD-R) uses the term “psychophysiological insomnia” for such a complaint and associated decreased functioning during wakefulness (American Sleep Disorders Association, 1997). ICSD-R regards insomnia of six-months duration as chronic. Both systems differentiate insomnia from: 1) circadian rhythm disorders, in which timing of the major sleep period is out of alignment with the local clock, 2) from parasomnias, in which behavioural events occur in association with sleep, e.g. sleep walking and night terrors, and 3) from secondary insomnias, in which psychiatric, neurological or medical problems present co-morbidly. Additionally, disorders such as sleep apnoea and disorders of excessive sleepiness, e.g. narcolepsy, are also classified separately.

In a recent epidemiological study (Canals, Domenech, Carbajo & Blade, 1997), insomnia was found to be the most common psychological health problem. The National Sleep Foundation reported that the prevalence of insomnia in the US was approximately thirty three per cent, with one in four of this group reporting occasional insomnia and nine per cent reporting that it occurred on a regular nightly basis (Ancoli-Israel & Roth, 1999). The rates from other European surveys are comparable (Chevalier, Loss, Bianchi, Nutt, et al., 1999; Cirignotta, Mondini, Zucconi, Lenzi & Lugaresi, 1985; Weyer & Dillig, 1991).
Insomnia is often thought of as not only a highly prevalent disorder, but as a chronic and recurring condition (Hohagen, Kappler, Schramm, Rink, Weyerer, et. al., 1994; Vollrath, Wicki & Angst, 1989). More importantly, insomnia is most commonly secondary to diagnoses of mood, anxiety and substance misuse related disorders (Ford and Kamerow, 1989). Harvey (2001) presents a very strong argument for considering insomnia as a disorder in its own right rather than secondary to or as an epiphenomenon of other disorders. It is argued that the idea which implies insomnia is secondary, is unfounded for three reasons: 1) depression is predicted by the presence of prior insomnia, 2) an effective intervention for the primary disorder does not necessarily alleviate the insomnia, and 3) insomnia is a risk factor for the development of psychological disorders.

Insomnia can be subjective. The amount of sleep that an individual feels he or she requires can be quite variable. A short sleeper may feel fine after five hours, whereas a long sleeper may feel unrefreshed after ten hours. Insomnia should, therefore, be defined in relation to the individual's sleep needs. Meddis, Pearson & Langford (1973) reported a case of a seventy-year-old woman who slept for approximately one hour per day, she felt fine and thought that most people “wasted much time” in bed. However, insomnia is typically defined as an extended sleep onset (greater that thirty minutes), or interference with sleep maintenance (extended awakenings) or awakening earlier than usual, combined with one or more of the following: daytime sleepiness, concentration and mood problems. Conservative estimates for chronic insomnia range from nine to twelve per cent in adulthood and up to twenty per cent in later life. Women present about twice as many times as men (Bixler, Kales, Soldatos, Kales & Healy, 1979; Ford & Kamerow, 1989).

An important, yet somewhat ironic, cause of insomnia is sleep-inducing medication (Hypnotics, Anxiolytics and Barbiturates). Patients who receive a sleeping medication develop a tolerance to the drug and suffer rebound effects when it is withdrawn (Weitzman, 1981). The patient then becomes convinced that the insomnia is even worse and seeks out more medication for relief. This common syndrome is called drug
dependency insomnia and Kales, Scharf, Kales & Soldatos (1979) reported that withdrawal from some medications produced rebound insomnia after the drugs were used for as few as three nights.

Disorders of sleep also include sleep apnoea an impairment of breathing rhythm. Problems associated with REM sleep include: narcolepsy (sleep at inappropriate times) characterised by sleep attacks and cataplexy; sleep paralysis (inability to move just before sleep onset or awakening); hypnagogic hallucinations (the person dreams while lying awake, paralysed) and REM without atonia (first described by Schenck, Bundlie, Ettinger, & Mahowald, 1986). Additionally, problems associated with slow-wave sleep are: bedwetting (nocturnal enuresis), sleepwalking (somnambulism) and night terrors (pavor nocturnus).

It has been difficult for health care providers to provide treatment for disorders because of controversies surrounding the addictive nature and distressing side effects of such pharmacological therapies. Evidence against the prolonged use of benzodiazepines and similar drugs is overwhelming: pharmacological treatment is not recommended for the treatment of primary or chronic insomnia, or in the elderly (NIH, 1999 & 1984).

There is a growing literature on the effectiveness of non-pharmacological treatment for insomnia. The treatments include: accurate sleep information, the practice of good sleep hygiene and the use of specific behavioural and cognitive techniques such as relaxation therapy and stimulus control (Morin, Hauri, Espie, Spielman, Buysse & Bootzin, 1999). Cognitive Behaviour Therapy has been regarded as the treatment of choice for chronic insomnia (Espie, 1999); around seventy to eighty per cent of patients benefit from non-pharmacological interventions for sleep disorders (Morin, Hauri, Espie, Spielman, Buysse & Bootzin (1999).

Morin et al, (1999) reported this impressive treatment outcome after conducting two extensive meta-analyses of forty-eight efficacy studies and clinical trials based in Canada, USA and Scotland. The main benefit for patients was a significantly enhanced
sleep quality, the two main targets of treatment being 1) a reduction in time getting to sleep (sleep latency) and 2) a small increase in sleep duration (usually averaged thirty minutes). Following this meta analysis, six of the treatment modalities met the American Psychological Association criteria for clinically validated treatments for insomnia (stimulus control, progressive muscle relaxation, paradoxical intention), or probably efficacious (biofeedback, sleep restriction, multicomponent cognitive behaviour therapy). The report adds, that although there are clinical advantages to a more eclectic approach to treatment of insomnia, there is no such support given to integrated bio behavioural interventions. However, there is no evidence to suppose that such interventions transfer readily to other clinical settings (primary care) and with various co-existing illness (secondary insomnia). This is highly pertinent to this current review, there is a dearth of evidence in this area, as no such treatment trials for the specific group of patients suffering from sleep disorder or insomnia following TBI was found.

1.3.1 Insomnia and Physical Illness

Insomnia is among the most prevalent health problems (Harvey, 2001). Morin (1993) estimated it to be the second most frequent psychological disorder, second only to chronic pain. Sleep disturbances are commonly observed among persons suffering from chronic medical conditions (Shapiro, Devins & Hussainm, 1993). Of the various DSM-IV (APA, 1994) sleep disorders, insomnia occurs most often within the general population and many medical groups (Hyppa & Kronholm, 1989). Studies examining the frequency of insomnia for specific diagnostic groups found higher than normal insomnia rates for asthma (Gislason & Almqvist, 1987; Janson, Gislason, Boman, 1990; van Keipema, Ariaansz, Nauta, 1995), chronic fatigue syndrome (Krupp, Jandorf, Coyle, 1993), chronic pain (Gislason & Almqvist, 1987; Wittig, Zorick, Blumer, 1982), diabetes (Gislason & Almqvist, 1987; Schiava, Stimmel, Mandeli, 1982; Sridhar & Madhu, 1994), renal dialysis patients (Holley, Nespor & Rault, 1992), fibromyalgia (Jennum, Drewes, Andreasen, 1993) and hypertension (Gislason & Almqvist, 1987). In some cases insomnia is not caused by the medical disorder, but rather, is caused by the treatment for the medical disorder. For example, drug treatments for asthma, hypertension and cancer can have stimulant properties.
Insomnia is often caused by disorders such as sleep-related respiratory disturbance, periodic leg movements, restless leg syndrome, obstructive sleep apnoea, narcolepsy, and idiopathic hypersomnia (Spielmann & Glovinsky, 1997; White and Mitler, 1997). Delayed sleep phase syndrome is a particularly common cause of insomnia (Regestein & Monk, 1995). This means difficulty in falling asleep and in waking up in the morning at the desired time, somewhat like “jet lag” where the person’s internal clock is desynchronised to local time as caused, for example, by a trans-Atlantic flight.

In neurological diseases such as epilepsy (Shapiro, Devins & Hussain, 1993), multiple sclerosis (Clark, Fleming & Li, 1992; Ferini-Strambi, Filippi, Martinelli, et al. 1994) and Parkinson’s disease (Factor, McAlarny & Sanchez-Ramos, 1990; Kales, Ansel & Markham, 1971; Lees, Blackburn & Campbell, 1988), sleep disturbance is common. In these cases the sleep disorder might be caused by an underlying disorder involving the central nervous system, the motor system or associated with cognitive and psychological disorders. Insomnia can also be related to the secondary effects of these illnesses (e.g. pain and immobility), or as a side effect of the medication prescribed to treat them.

Katz & McHorney (1998) assessed 3445 patients with hypertension, diabetes, congestive heart failure, myocardial infarction and depression. A follow-up assessment was completed with 1814 patients two years later. Mild insomnia was diagnosed in thirty four per cent of the patients at baseline and fifty nine per cent of these patients still had the diagnosis at follow-up. Severe insomnia was diagnosed in sixteen per cent and this diagnosis persisted in eighty three per cent of that group at two-year follow-up. This study highlights the chronic nature of insomnia, particularly in those with severe insomnia. These results are particularly noteworthy because the primary medical conditions were not TBI, terminal or untreatable conditions.

1.3.2 Insomnia and Psychological Disorders
Insomnia as a symptom of a psychological disorder is ten times more frequent than insomnia related to physical illness (Ford & Kamerow, 1989) and this association
between insomnia and psychological disorders raises important questions relating to cause and effect (Harvey, 2001; Katz & McHorney, 1998).

“Just as night follows day, so does sleep disturbance follow psychological disturbance” (Spielman & Glovinsky, 1997, p133).

Sleep disturbance is a common symptom used in the diagnosis of many psychological disorders. For example, sleep disturbance is listed as a symptom in many DSM-IV disorders (nineteen Axis I disorders and four diagnoses listed in the “Disorders for further study” section list insomnia as a symptom). In addition, there are many other disorders where insomnia is not a listed symptom but is documented as figuring prominently in the clinical presentation of the disorder. For example, individuals diagnosed with panic disorder often report sleep-onset insomnia and avoidance behaviour related to going to sleep because of a fear of having a panic attack through the night (Craske & Rowe, 1997; Lepola, Koponen & Leinonen, 1994). Furthermore, social phobia has been associated with significantly poor sleep quality, longer sleep onset latency, more frequent sleep disturbance and more severe daytime dysfunction compared to matched controls (Stein, Kroft & Walker, 1993).

One of the primary symptoms of depression is disrupted sleep. During depression the patient rarely enters deep sleep. The quality of the sleep is markedly reduced, because the feelings of being refreshed and well rested are associated with this missing sleep phase. However, contrary to the views of Spielman & Glovinsky (1997) that sleep disorders follow periods of psychological distress, the sleep disturbance often precedes the depressive episode. Sufferers of depression may have many brief episodes of REM sleep rather than the less frequent and longer episodes experienced in the healthy individual. This repetitive REM may be more exhausting than restful. Most antidepressants reduce REM sleep rather than improving the overall quality of the sleep.

Generally people who suffer from depression also experience diurnal mood swings, waking up early with a feeling of ominous dread, feeling worst in the morning and a little better as the day goes on. Unfortunately, the number of studies reporting co-
morbidity between insomnia and other disorders is limited, as insomnia is typically not assessed in epidemiological studies (Canals, Domenech, Carbaj, & Blade, 1997). Harvey (2001) suggested three main reasons for this absence: 1) studies may assume insomnia is trivial and secondary to other disorders, 2) there are differing and inconsistent definitions of insomnia, and 3) the lack of psychometrically validated assessment instruments for the diagnosis of insomnia.

There is huge variability in the reported estimated rates of co-morbidity between insomnia and other psychological disorders, which has ranged a great deal (from four to one hundred per cent). There are a number of contributory factors for this variability, mainly relating to methodological differences across studies. Firstly, the criteria used for assessing and diagnoses of insomnia differ vastly across various studies. Most studies employ unstructured clinician interviews or simple rating scales. Secondly, the diagnosis of the psychological disorder also varies a great deal across studies: some used psychometrically validated interviews for assessing the presence of a psychological disorder (e.g. the Structured Clinical Interview for Diagnosis; SCID), while other studies relied on clinician judgement. Thirdly, the range of psychiatric disorders assessed for varied a great deal. For example, Ford & Kamerow (1989) used the Diagnostic Interview Schedule (DIS), which covers Axis I disorders but does not assess for generalised anxiety disorders or Axis II personality disorders. In comparison, Tan, Kales, Soldatos & Bixler (1984) included an assessment for both Axis I and Axis II disorders. It was not surprising, and then, that the Tan et al. (1984) study found a higher estimate of co-morbidity.

However, Harvey (2001) summarises the comorbidity rates succinctly in a tabulated format, which indicate depressive and anxiety disorders to be the most common comorbid disorders. For example, Ford & Kamerow (1989) reported disorders comorbid with insomnia to be anxiety disorders (twenty four per cent), major depression (fourteen per cent), dysthymia (nine per cent), alcohol abuse (seven per cent) and drug abuse (four per cent). Nowell, Buysee, Reynolds, Hauri, Roth & Stepanski, (1997) reported mood disorders (twenty nine per cent), anxiety disorders (five per cent), personality disorders
(five per cent), adjustment disorders (two per cent) and psychotic disorders (two per cent).

1.4 SLEEP DISORDER AND TRAUMATIC BRAIN INJURY

The issue of insomnia is of concern to TBI treatment providers. As Zafonte, Mann & Fichtenberg (1996) have stated:

"Abnormal sleep patterns can exacerbate behavioral disturbances and increase difficulty with new learning. Early identification and evaluation of sleep disorders with the appropriate environmental and pharmacological intervention can limit cognitive and behavioural sequelae following TBI." (Zafonte, Mann & Fichtenberg, 1996, p189)

Sleep disorder following TBI has been investigated at varying times since injury (weeks, months and years), across different TBI severity groups (mild, moderate and severe), in relation to common sequelae of TBI and its effect on the accurate measurement of, for example, poor self-awareness, daytime fatigue and by individual single case study.

1.4.1 Time since injury and sleep disorder

The presence of insomnia and sleep disturbance has been well established in the early stages after TBI. In a study of mild TBI, fifteen per cent of the patients, Rutherford, Merrett & McDonald (1979) interviewed (n=145), reported sleep disturbance, six weeks post injury. Subsequent TBI studies have investigated a wide spectrum of injury severity levels and its effect on sleep disorder. Keshaven, Channabasavanna & Reddy (1981) administered a general symptom checklist to sixty randomly selected TBI cases at one and a half and three months post injury. Sleeplessness was endorsed as a current symptom by seventy per cent initially and by thirty seven per cent six weeks later. Cohen, Oksenberg, & Snir (1992) administered a thirty eight-item sleep questionnaire to twenty two recent onset (less than one year post injury) patients, finding insomnia rates of fifty nine per cent. McLean, Dikman, Temkin, et. al. (1984; 1993) included a general symptom checklist in their longitudinal study of one hundred and two TBI cases at one-month post injury and found that thirty six per cent reported insomnia.
The literature reports huge variations in the rates of sleep disorder between the very early and later stages following TBI. Observed sleep disorder rates vary hugely from two to eighty eight per cent (Rutherford, et al, 1979 and Biswas & Kemp, 2002). The Rutherford, et al study (1979), described above, only found two per cent of the patients followed-up (n=131) at one-year post injury complained of insomnia. Similarly, the sleep questionnaire administered by Cohen et al (1992) to seventy seven longer-term TBI cases (two to three years post injury) only yielded an insomnia rate of four per cent.

However, higher insomnia prevalence has been reported in longer-term time since injury TBI groups. McLean, et al (1984 & 1993) at one year post injury, found twenty seven per cent of their sample reported insomnia as a continuing problem on responses generated by a general symptom checklist. Beetar, Guilmette & Sparadeo (1996) conducted an archival review of records at a neuropsychological outpatient clinic. This review of two hundred and two randomly selected TBI cases identified fifty six per cent of the patients who complained of insomnia when questioned whilst attending the clinic. Clinchot, Bogner, Mysiw, Fugate & Corrigan (1998) inquired about sleep patterns during telephone interviews conducted with eighty six brain-injured patients. The survey revealed that one year after discharge from a rehabilitation program, fifty per cent of their sample reported sleep problems and, of that sample, thirty two per cent described terminal insomnia, twenty three per cent sleep onset insomnia and thirteen per cent hypersomnia. Furthermore, Biswas & Kemp, (2002) designed a recent postal survey to determine the prevalence of sleep disorder in longer term TBI patients living in the community (mean time since injury was 3 years). They reported a massive eighty eight per cent of their fifty randomly selected cases reported a sleep disorder.

In another recent study Fichtenberg, Zafonte, Putnam, Mann & Millard, (2002) investigated the frequency of insomnia within a population of post acute (mean time since injury 3.8 months, range of 2 weeks to 53 months). In this prospective study of fifty consecutive TBI cases they found that almost one third had insomnia, measured by a standardized psychometric tool (The Pittsburgh Sleep quality Index, Buysse et al.
1989) and sleep diaries. Sleep initiation was a problem nearly twice as often as sleep duration. In the study, twelve per cent did not meet DSM-IV criteria for insomnia but, nevertheless, experienced a degradation of sleep quality. However, slightly more than half of the TBI sample reported sleep to be normal and satisfactory. They conclude:

“[that] the occurrence of insomnia among TBI patients is of a magnitude that merits serious attention from TBI healthcare providers”

(Fichtenberg et al., 2002, p203)

It is encouraging to note that of the studies that have addressed the issue of insomnia within the post-acute population, the only large scale full severity range prospective study (McLean, Dikmen, Temkin, 1993), reported results similar to the recent Fichtenberg, et al (2002) study, reporting rates of insomnia of twenty seven and thirty per cent, respectively.

These studies represent the current state of available knowledge regarding the prevalence of insomnia within the TBI population. Observed sleep disorder rates vary widely from two to eighty eight per cent. There are a number of contributors to the large variability observed, mainly relating to methodological differences across studies. Most studies employed non-standardized, researcher designed, questionnaire surveys or general symptom checklists. Very few of the longer-term TBI follow-up studies employ a diagnostic criterion for sleep disorder (e.g. DSM-IV, ICD-10 or ICSD-R) or a psychometrically valid tool for the assessment of insomnia. This lack of standardized definition of the sleep disorder categories employed in past research allow for only a very limited comparison between study findings and might contribute to the large variability of sleep disorder rates.

In addition, the ranges and definition of the time since injury can vary a great deal from study to study, from weeks and months to years, and this appears to be dependent on the source of the study population from acute and primary services (e.g. acute hospital admission ward, rehabilitation unit, outpatient clinic). For example, Fichtenberg et al,
(2002) reported a fairly broad time since injury inclusion range (2 weeks to 53 months) in their rehabilitation outpatient sample. This wide range allows for little comparison between TBI sleep disorder occurrence in the early and later stages of the TBI recovery process and this large inclusion range may further contribute to the observed variability in sleep disorder rates.

However, it is encouraging to see that recent more rigorous studies with post acute TBI groups (e.g. Fichtenberg et al, 2002) have used standardized measures, diagnostic criteria and rehabilitation patient control groups, which have supported earlier, less rigorous study findings (e.g. Rutherford et al., 1979). To investigate whether insomnia really is a problem in the later stages of TBI recovery, following discharge from the acute and rehabilitation services, it would be crucial to also utilize such rigorous methodology in a TBI group at one year or more following injury.

It would be of clinical interest to investigate similar issues in a Scottish population of TBI cases after the acute stage, one year or more following their injury. Many of the previous studies (Keshaven et al., 1981; Cohen et al., 1992) have merely focused on an acute stage (immediately following trauma) or up to one year following the injury time ranges. This emphasis on the early stages of a TBI recovery process may be explained by the difficulties involved with contacting a patient once he or she has been discharged from a service. There are also many ethical implications regarding contacting a patient once they have been discharged from a service. Firstly, they may not wish to be contacted, they may have changed address or their condition may have deteriorated. Additionally, some patients will have died and issues of breaching confidentiality must be considered very carefully. These ethical implications must be considered before embarking on a project but they should not impede the investigation of such an important clinical area.

Any investigation of this kind should aim to describe the frequency and severity of insomnia and poor sleep in the TBI group and identify predictors of poor sleep quality. Past studies have employed various symptom checklists (e.g. Keshaven et al., 1981),
which invariably are not standardized and do not provide a diagnostic criteria for the measurement of insomnia. The Pittsburgh Sleep Quality Index (PSQI) has been validated for persons with post-acute TBI. Fichtenberg et al (2001) reported high sensitivity (93 per cent) and specificity (100 per cent) of a PSQI Global Score within the range diagnostic of insomnia (more than a PSQI global score of 8). In a non TBI clinical sample Buysee et al. (1989) reported satisfactory levels of internal homogeneity, consistency and validity and this measure has been widely used in both clinical practice and research (Espie, 2002).

1.4.2. TBI severity, self-awareness, fatigue and qualitative sleep experience and sleep disorder

An interesting, yet paradoxical, finding has been reported that states the more severe the head injury the less likely the patient is to suffer from sleep disturbance (Beetar, Guilmette, Sparadeo, 1996; Fitchenberg, Mills, Zafonte & Millard, 2000). The relationship between TBI severity and sleep disorder is very important and worthy of further study. Past studies have reported an increased likelihood of developing a sleep disorder if the TBI is of a mild TBI severity (Clinichot, et al., 1998; Beetar et al., 1996; Fitchenberg et al., 2000). These past studies have compared the frequency of sleep disorder between different TBI severity ratings. It would be interesting to replicate this relatively robust finding that indicates that individuals with a severe TBI experience lower frequencies of sleep disorder.

Since self-report measures are most frequently utilized to obtain measures of sleep quality, it would also be very important to investigate the TBI participant’s self-awareness of their sleep quality. Objective measures of sleep quality have been found to identify significantly higher rates of sleep disorder than self report measures, completed by the patient in TBI populations (e.g. Masel, Schiebel, Kimbark, & Kuna, 2001). Few past studies have attempted to validate the TBI patients’ self report responses by including an objective measure, perhaps by means of the assistance of a significant other, in other words a person who knows the participant well (a spouse, parent or carer). The significant other could complete the sleep quality measures as if they were
the participant. This would allow the investigator to examine the validity of the participants’ self-report responses on the sleep quality measure.

Hypersomnia is common in adults with brain injuries (Masel, Schiebel, Kimbark, & Kuna, 2001). The literature has reported that people with TBI’s who suffered from sleep disturbance were more likely to have problems with fatigue (Clinichot et al., 1998). This research suggests that it would be valuable to investigate TBI severity and people’s long-term TBI experience, in the areas of sleep quality, psychological distress and daytime fatigue. This investigation would look at whether TBI severity is related to the experience of daytime fatigue.

Finally, there are few qualitative investigations of sleep disturbance in TBI populations. A few case reports (e.g. Tobe, Schneider, Mrozik & Lidsky, 1999) have reported persistent insomnia following TBI. However, there is a dearth of literature that described the sleep experiences of a sample of TBI patients. It is important to use such studies as an opportunity to explore the individual experiences of such a sample, to distinguish factors that might describe “good” and “bad” sleep patterns in a TBI population.

1.4.3 The present study
When compared to estimates of prevalence of insomnia within the general population, the average, thirty per cent frequency that is reported within this TBI group (Fichtenberg et al., 2002; McLean, et al., 1984; McLean, et al., 1993) is well above population average of around six to ten per cent. The epidemiological reports regarding occurrence of insomnia in the USA and in Europe are comparable. Ford and Kamerow’s epidemiological study of 7954 subjects found a ten per cent prevalence and six per cent incidence of insomnia. Janson, Gislason & Backer (1995) found among 2202 residents of Iceland, Sweden and Belgium, six to nine per cent suffered from sleep onset insomnia and five to six per cent suffered terminal insomnia.

"Sleep disorders are a relatively common occurrence after brain injury. Sleep disturbances often result in a poor daytime performance and a poor individual sense of well-being. Unfortunately, there has been
minimal attention paid to this common and often disabling sequela of brain injury," (Clinchot, Bogner, Mysiw, Fugate, & Corrigan, 1998, p291).

What are the characteristics predictive of sleep disorder in traumatic brain injury patients? Affective status, age, depression, pain, litigation and gender have all been linked to sleep disorder following brain injury (McLean, et al., 1984; McLean, et al., 1993; Fichtenberg, et al, 2002; Beetar, et al., 1996; Fichtenberg et al., 2000, Clinchot et al., 1998).

The relationship between severity of head injury and sleep disturbance needs to be investigated. Previous studies have depended upon self-report measures of sleep difficulties. It might be that persons with more severe injuries reported fewer sleep complaints because of a lack of awareness, or perhaps because of limited memory of difficulties (Clinchot, et al., 1998). It has been recommended that direct observation and verification by others would provide a more objective and reliable evaluation of sleep complaints. To explore this further, this study has included reports of sleep observations by a significant other.

Clinchot, et al. (1998) demonstrated a relationship between sleep disorder and perceptions of fatigue. That is, levels of perceived fatigue correlated with levels of sleep complaints. This study plans to examine the relationship between perceptions of fatigue and sleep disorder further by utilising standardised fatigue measures (Bentall, Wood, Marrinan, Deans & Edwards, 1993; Lee, Hicks & Nino-Murcia, 1991). A further aim was to examine the neglected area of how people cope with their sleep disturbance and explores how that might interact with their experience of sleep. A semi-structured interview was developed to explore these issues.

The study of sleep disturbance in this population is especially important considering that neurobehavioural impairments have been reported to be more common in patients post TBI, with sleep complaints when compared to those who did not report sleep disturbances and also occupational outcome was poorer (Cohen et al., 1992). The implications for studies in this field are immense and crucially indicate that prompt
treatment of the underlying sleep disorder has potential secondary benefits for the neurobehavioural impairments and quality of life of people in this vulnerable group.

1.4.4 Study Aims

- To identify the characteristics predictive of sleep disorder in a sample of traumatic brain injury patients, twelve months or more following trauma.
- To measure the frequency and severity of sleep disturbance in this sample.
- To investigate the relationship between head injury severity and sleep disturbance in this sample.
- To examine the validity of self-report measures of sleep disorder by collecting the objective reports of sleep disorder by a significant other.
- To explore the relationship between subjective perceptions of fatigue and sleep disorder in this sample.
- To use qualitative analysis as a format by which to further investigate sleep disturbance in this sample of TBI study participants. In addition, to investigate how participants described their sleep in the past, current sleep quality and how participants coped with sleep disturbances.

1.4.5 Hypotheses

**Hypothesis 1:** that sleep quality (as assessed by the Pittsburgh Sleep Quality Inventory; PSQI), at one year or more following traumatic brain injury (TBI), will be predicted by: age, TBI severity levels (GCS, PTA), time since injury, length of hospitalisation for rehabilitation, anxiety (HADS anxiety), depression (HADS depression & Hamilton Rating Scale for Depression; HRSD), daytime fatigue (Bentall, VAS-F), and pain.

**Hypothesis 2:** that participants with a mild or moderate severity of traumatic brain injury will report a poorer sleep quality in comparison to those participants with a severe TBI.
**Hypothesis 3:** participants with a mild / moderate severity of TBI will report higher subjective perceptions of fatigue (as assessed by the Bentall Fatigue Inventory and the Visual Analogue Scale-Fatigue; VAS-F) in comparison to those with a severe TBI.

**Hypothesis 4:** participant’s ratings on self-report measures of their sleep quality will be different to the objective reports of a significant other.

**Hypothesis 5:** participants with mild / moderate severity TBI and a bad sleep quality will report higher levels of: depression (HADS depression and HRSD), anxiety (HADS anxiety) and alcohol, nicotine and caffeine use.
2. METHODOLOGY

2.1 DESIGN

This study investigated the variables characteristic of sleep disorder in a Traumatic Brain Injury (TBI) sample, one year after the index injury. A between-subject design explored the relationship between the participant’s sleep disturbance and the severity of TBI. A within-subjects design investigated reliability of the sleep disorder self-report and explored differences between ratings of the participant and a significant other. In addition, qualitative analysis based on content analysis, investigated themes generated by a semi-structured interview.

2.1.1 Participants:

The medical discharge summary of all patients admitted to the Scottish Brain Injury Service (SBIRS) between June 2002 and June 1997, stretching back over the last six years, were reviewed (n=90). Forty four (48 per cent) cases met inclusion criteria for the study and of these twenty one (49 per cent) agreed to participate. The other forty-six cases were excluded or could not be contacted for a variety of reasons (See Appendix D).

Thirteen out of the twenty-one interviews was carried out in the participant’s own home, as the other eight preferred to attend the hospital clinical psychology outpatient service. Due to the SBIRS being a national brain injury service the geographical regions involved in recruiting patients for this study included Lothian, Borders, Central and Fife. Due to travel and time restrictions participants who lived further a field were excluded.

2.1.2 Selection Criteria

Inclusion criteria

These were as follows:

- Patients who were admitted to the Scottish Brain Injury Rehabilitation Service (SBIRS), Astley Ainslie Hospital, Edinburgh, following a traumatic brain injury.
- TBI sustained at least 12 months since commencement of this study.
• Information regarding severity of injury, GCS and/or PTA scores, was available from medical and/or neuropsychology case notes.
• The first language of the participants was English.
• Above eighteen years of age at the time of the injury.
• Not currently, or in the past six months, involved in any other research.
• Literate and scored more than three on the clock drawing test.
• No concurrent psychotic illness, which required psychiatric treatment or medication and differentiated from a posttraumatic confusional state.
• Absence of treatment for alcoholism.
• Within a two hour travelling distance from the research base.

2.2 ASSESSMENT MEASURES

2.2.1 Brain Injury Severity
Patient medical and neuropsychology case notes were reviewed to obtain the following information regarding the severity of TBI: The Glasgow Coma Scale (GCS, Jennet & Teasdale, 1977) and Post Traumatic Amnesia (PTA, Wilson, Shiel Watson & McLellan, 1994), both regarded as robust measures of the severity of brain injury. In addition, the location of injury (CT and/or MRI summary reports) and nature of injury (RTA, assault, fall, sports) were also included in the dataset.

2.2.2 Neuropsychological screening
The Clock Drawing Test (CDT, Freedman, Kaplan, Delis & Morris, 1994) was used as a screening measure for cognitive function. The CDT has achieved extensive clinical use as a cognitive screening instrument and a significant amount of literature has detailed its psychometric properties and clinical utility (Shulman, 2000). In addition, as a quick measure of neuropsychological status, The Mini Mental State Exam (MMSE, Folstein, Folstein & McHugh, 1975) was also administered. An estimate of premorbid intellectual status was obtained using the National Adult Reading Test (NART, Nelson, 1991). This
is a test of accuracy of pronunciation of irregular words. Ability on this test is highly correlated with intelligence and is relatively resistant to the effects of a variety of brain disorders (O’Carroll, 1995).

2.2.3 Sleep Quality
The interview for sleep disorder used The Pittsburgh Sleep Quality Index (PSQI, Buysee, Reynolds, Monk, Berman, & Kupfer, 1989; see Appendix B). The PSQI is a self-report measure of subjective sleep quality over the past month. The measure has been designed to obtain a stable and long-term view of sleep quality, rather than an estimate for one or two nights only. Nineteen individual items generate seven component scores: subjective sleep quality, sleep latency, sleep duration, habitual sleep efficiency, sleep disturbances, use of sleeping medication and daytime dysfunction.

A PSQI Global Score less than or equal to five reflects a good sleep quality and a score of more than five a poor sleep quality (Buysee et al., 1989) The sum of scores for these seven components yields one global score. The PSQI has been validated for persons with post-acute TBI (Fichtenberg, 2001), as sensitivity and specificity to insomnia of a PSQI Global Score more than eight were ninety-three per cent and one hundred per cent, respectively. In a clinical sample Buysee, et al. (1989) reported satisfactory levels of internal homogeneity, consistency and validity and it has been widely used in both clinical practice and research (Espie, 2002).

In the current study a threshold PSQI Global Score more than eight was used to indicate cases of insomnia and poor sleep was defined as a PSQI Global Score of more than five.

2.2.4 Psychological Distress
The Hospital Anxiety and Depression Scale (HADS; Appendix B) is a brief (14-item), self-report measure of anxiety and depression developed by Zigmond & Snaith (1983), see Snaith, & Zigmond, 1994. It was developed for use in the general medical outpatient clinics but is now widely used in clinical practice and research (Hermann, 1997). For the anxiety and depression scales raw scores of around eight to ten identify mild cases,
eleven to fifteen moderate cases and sixteen or above are severe cases (Snaith & Zigmond, 1994). The HADS avoids the inclusion of items such as insomnia or loss of appetite, which are symptoms of a depressive or anxiety disorder, but are also likely to be present in a person suffering from bodily illness. The HADS concentrates on the psychic rather than the somatic manifestations of mood disorder. It is brief and can be completed very quickly, therefore good for patients who may suffer from a poor concentration span.

The Hamilton Rating Scale for Depression (HRSD; Hamilton, 1960, 1967; Appendix B) is a widely used observer scale that includes assessment of cognitive and behavioural components of depression and is particularly thorough in the assessment of somatic aspects (Hamilton, 1967). The HRSD has been used with the TBI population (Rosenthal et al., 1999). The scale is reported to have high concurrent validity with good agreement with other scales, particularly the Beck Depression Inventory (BDI), with correlations reported of over 0.70 (Hamilton, 1976). Its inter-rater reliability is reported to be good (Hamilton, 1976; Knesevich, 1977; Rehm, 1981).

On the HRSD, a score of zero to six is generally interpreted as normal, seven to seventeen as mild, eighteen to twenty-four as moderate and twenty-five and above as severe. The interpretation of categories has been fully described by Hamilton (1967).

2.2.5 Fatigue

Fatigue is a common complaint both for patients with a sleep disorder and those following a TBI. In this study two measures of fatigue, the Bentall Inventory (Bentall, Wood, Marrinan, Deans, & Edwards, 1993, see Appendix B) and The Visual Analogue Scale for Fatigue (VAS-F; Lee, 1991; see Appendix B), were employed.

The Bentall is a brief, nine item, fatigue inventory, which has excellent internal consistency and has been shown to discriminate between physically ill patients, clinically depressed patients and healthy controls (Bentall et. al., 1993). On the Bentall fatigue inventory a score of zero is generally interpreted as not at all fatigued, one to
nine as a little, ten to eighteen as somewhat, nineteen to twenty seven as quite a lot and twenty eight to thirty six as very much.

The VAS-F is a standardised visual analogue scale of fatigue; the scale consists of 18 items related to fatigue and energy. The VAS-F has high internal consistency and reliability, which has been shown to discriminate between healthy and sleep disordered samples (Lee, 1991). The energy component of the VAS-F is calculated by summing items six to 10 and the sum of the remaining thirteen items constitute the fatigue score. In addition an average was calculated and computed the VAS-F overall score.

2.2.6 Semi-structured interview
A semi-structured interview (Appendix C) invited participants to describe their sleep prior to the TBI, their current sleep experience and individual levels of coping with sleep disturbance, including reported quantities of current substance use within the following categories: current medications, recreational drugs, caffeine, alcohol and nicotine. Participants were also asked if they experienced pain (yes/no) and if they answered yes they were invited to rate the pain, at night and during the day, on a scale of one to ten (where one was no pain at all and 10 was excruciating). Part of the interview informed descriptive data and a qualitative investigation, based on content analysis.

2.3 PROCEDURES
2.3.1 Ethical approval
Permission was sought for ethical approval from Lothian Ethics Committee in December 2001. A study proposal was submitted. Several amendments and clarification points were requested following the committees perusal of the proposal, regarding: the researchers position as part of the clinical team, whether patients would be discharged from the service; comments about the wording of particular sections of the patient information sheet and choice of wording on a standardised visual analogue scale (see appendix D1). In the interim, the committee administrator discussed the concerns of the committee regarding the contact of patients who had not been in contact with the service
for some time and who had effectively been discharged to the care of their general practitioner. The Committee administrator made it very clear that it was unlikely that ethical approval would be granted because of this recruitment difficulty. The researcher agreed to contact the patient’s general practitioners, to seek permission to contact their patient, prior to contacting any discharged patients. These clarifications and amendments were completed and resubmitted to the next meeting of the ethics committee, January 2002. At this second meeting a further change to the patient information sheet was requested. The researcher was advised to state that the independent advisors role as someone not directly involved in the study, and to clarify that while the researcher was able to provide information on the study, information from someone not directly involved in the research could also be obtained from the independent advisor (see Appendix D2). This amendment was made and resubmitted to the meeting of the next ethics committee, February 2002 (Appendix D3). The proposal was passed and certificate of ethical review (see Appendix D4) was awarded at the next meeting of the ethics committee, March 2002.

2.3.2. Determining the study size and power
Fichtenberg, et al. (2000) using n=91 found several significant results with an $\alpha <.01$. We used fewer subjects, but by reducing $\alpha$ to $\alpha <.05$ we can retain the same power. Based on the statistic $\delta$ (In Howell, 1997) the n=62 is required for the same power, effect size and $\alpha <.05$. However, this study did not meet the required sample size for predicted power, nevertheless a significant result was obtained.

2.3.3 Study procedures
The permission of the patient’s key clinician was sought prior to making initial contact with each patient. For example, the permission of the current GP was sought where a patient had not attended the SBIRS outpatient department for six months or more. Those patients who met the criteria for participation in the study (n=44) were contacted to take part in the study.
Participants who met the study criteria were invited, by post, to take part. Patient information sheets and consent forms accompanied the initial invitation letters. People who had indicated interest in taking part were contacted and an appointment was agreed upon for the initial interview of the participant and the significant other (see Appendix A).

Following successful performance on the CDT screening measure, the participant was asked to complete the PSQI, a retrospective PSQI and the HRSD, NART, HADS, Bentall, VAS-F, the MMSE and a semi-structured interview (which was audio-taped for transcription). Then the nominated significant other was asked to complete the PSQI “as if they were their relative” (see Appendix B & C).

If the participant reported subjective sleep difficulties they were offered leaflets on good sleep hygiene (see Appendix F) and a comprehensive progressive muscular relaxation tape. They were also given instructions to contact their GP to discuss their difficulties if these problems continued. In addition they were offered a follow-up session with the researcher to discuss any issues they may have. Two of the participants attended the researcher for treatment of sleep difficulties after taking part in this study.

2.3.4 Semi structured interview

Qualitative methodology based on content analysis (Weber, 1995) was drawn upon to analyse the semi-structured interview responses. Taylor and Bogdan (1998) suggest that all qualitative reports should provide enough information about how the research was conducted to allow readers to discount the account or to understand it in the context in which it was produced.

Content analysis is a method that allows the researcher to make inferences from textual material. The central idea is that many words of text can be classified into fewer content categories, usually based on similarity (e.g. in meanings, connotations or topics). Types of data are usually open-ended interview responses, transcripts of verbal interaction or newspaper articles. This method allows data collection in a relatively open-ended and
unconstrained fashion or the analysis of naturally occurring text. It allows the combination of qualitative and quantitative analyses of data and yields results that can be used in some statistical tests (e.g. Chi Square).

Coding is the process of creating categories and assigning them to selected data (Dey, 1993). In qualitative research the process is sometimes referred to as indexing (Mason, 1996). While coding is a term used in both quantitative and qualitative research, very different processes are involved (Darlington & Scott, 2002). In quantitative research, coding is part of data management and involves numerically transforming the data in preparation for analysis. In qualitative research, coding is an integral part of the analysis, involving sifting through the data, making sense of it and categorising it in various ways. The analytic choices made here about what to code and how it will influence every stage of the research from here on. Qualitative analysis is generally concerned with identifying patterns in the data, different ways in which the data relate to each other (Lofland & Lofland, 1995).

The units of analysis were defined as themes identified in the transcripts (see Appendix E). The transcripts were separated into 3 groups: good sleepers, bad sleepers and insomniacs. Two raters coded the data. Themes were searched for under each of the 3 question headings: sleep before the TBI, since the TBI and coping with sleeplessness. When a new theme was identified, previous transcripts were reviewed again to ensure data was not missed. Thematic units were defined according to the question and the emerging themes: sleep experience before the injury (bedtime routine, sleep maintenance, waking from sleep), sleep now (bedtime routine, sleep maintenance, waking from sleep), coping strategies for sleeplessness and miscellaneous categories. Themes were identified and the two coders sorted the transcript themes into the identified categories, independently. Intercoder reliability was assessed by summing the coding agreements (e.g. how many times coder A and coder B put the same unit in the same category) and was divided by the number of units to be coded. This calculation (agreements / total number of units in sample x 100) provided an agreement percentage, which was a reliable category scheme above 80 per cent (Krippendorff, 1980). The
frequencies for each category, for both raters, were entered into the SPSS 10.0 statistical package to enable non-parametric analysis. The maximum frequency on each category, between the raters, was presented in tabulated format and categories were presented and discussed in this format.

Each of the participants was interviewed after completing the initial qualitative screening tests along with the semi-structured interviews about coping since their TBI. During a semi-structured interview the participants were asked about three main areas:

1) To describe a typical night’s sleep before the injury.
2) To describe a typical night’s sleep now.
3) What did they do when they could not sleep? During the interview they were also asked about their alcohol, caffeine and other substance use and asked about lifestyle changes.

A micro cassette recorder was used to audiotape the interviews. The participants were all fully aware that the interviews would be transcribed and presented in a typed, anonymous format and all participants provided written consent prior to taking part. Each recording was approximately five minutes in length, no more than one and a half page of A4 paper. The researcher then transcribed the audio tape recordings in Microsoft Word document format. The transcription followed this process: first the audio recordings were typed as transcripts, then the transcripts were examined while listening to the tapes again and any changes were made.
3 RESULTS

3.1 DESCRIPTIVE STATISTICS

3.1.1 The overall target sample
The medical discharge summary of all patients admitted to the Scottish Brain Injury Service (SBIRS) between June 1997 and June 2002 (n=90). Forty four (48 per cent) cases met inclusion criteria for the study and of these, twenty-one (49 per cent) agreed to participate. Table 3.1 shows the demographic characteristics of the participants and the target sample. There were no significant differences found on comparison of means measures for the variables of age (t=-.814, df=80, p=.418), gender (x²=.030, df = 1, p=.862), time since injury (t=-.420, df = 75, p=.676) and severity of TBI (t=.723, df =61, p=.472; t=-.103, df=38, p = .919, GCS and PTA respectively).

Table 3.1 Demographic characteristics, of the group who participated in the study (participants) and the target sample group (overall target sample), both groups were 1 year or more following TBI and not more than 5 years had passed since their discharge from the rehabilitation unit.

<table>
<thead>
<tr>
<th></th>
<th>Participants (n=21)</th>
<th>Overall target sample (n =69)</th>
<th>Statistical analysis</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mean (s.d) &amp; per cent</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Age (years)</td>
<td>40.4 (18.6)</td>
<td>43.8 (15.1)</td>
<td>t=-.814, df=80, p=.418</td>
</tr>
<tr>
<td>Gender (male)</td>
<td>80%</td>
<td>82.9%</td>
<td>x²=.030, df = 1, p=.862</td>
</tr>
<tr>
<td>Time since injury (years)</td>
<td>3.8 (1.1)</td>
<td>3.9 (1.2)</td>
<td>t=-.420, df = 75, p=.676</td>
</tr>
<tr>
<td>GCS</td>
<td>8.1 (3.6)</td>
<td>8.9 (4.1)</td>
<td>t=.723, df =61, p=.472</td>
</tr>
<tr>
<td>PTA</td>
<td>127.1</td>
<td>129.4</td>
<td>t=-.103, df=38, p = .919</td>
</tr>
</tbody>
</table>

There was no significant differences found on comparison of means measures for the variables of Age, Gender, Time since injury and Severity measures (GCS and PTA).

There was no significant difference found on the categories in the PTA severity rating (x²=7.52, df=5, p<.185). However, when GCS severity categories were considered there was a significant difference (x²=18.12, df=3, P<.001). The frequencies, percentages and statistical analysis are shown in Table 3.2. The GCS severity categories for the participants and the overall group, respectively, were mild 14 per cent (3), 17 per cent (12); moderate 20 per cent (4), 30 percent (20) and severe 66 per cent (14), 20 per
cent(14). That is, the participant group had significantly more participants in the severe GCS range.

**TABLE 3.2** Severity of Traumatic Brain Injury: on the Glasgow Coma Scale and Posttraumatic Amnesia

<table>
<thead>
<tr>
<th>GCS</th>
<th>Participants (n=21)</th>
<th>Overall target sample(n =69)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Frequency</td>
<td>Percent</td>
</tr>
<tr>
<td>Mild</td>
<td>3</td>
<td>14</td>
</tr>
<tr>
<td>Moderate</td>
<td>4</td>
<td>20</td>
</tr>
<tr>
<td>Severe</td>
<td>14</td>
<td>66</td>
</tr>
<tr>
<td>Missing</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>Total</td>
<td>21</td>
<td>100</td>
</tr>
</tbody>
</table>

\[ x^2=18.12 \]
\[ df=2 \]
\[ p<.001 \]

There were no significant difference between nature of the injury, how the TBI was sustained by the participants and the potential sample population. \( (x^2=3.96, df=3, p=.27) \) found on comparison of the categories for nature of injury: road traffic accident (RTA), sports, assault and falls. Table 3.3. shows the percentage and frequency rates for each group according to the method by which they sustained their injury.

**TABLE 3.3** Nature of the injury, how the TBI was sustained, of the group who participated in the study (participants) and the target sample group (overall target sample), both groups were 1 year or more following TBI and not more than 5 years had passed since their discharge from the rehabilitation unit.

<table>
<thead>
<tr>
<th></th>
<th>Participants</th>
<th>Target sample</th>
<th>Statistical analysis</th>
</tr>
</thead>
<tbody>
<tr>
<td>RTA</td>
<td>45% (10)</td>
<td>37.1% (25)</td>
<td>( x^2=3.963 )</td>
</tr>
<tr>
<td>Sports</td>
<td>10% (2)</td>
<td>2.9% (2)</td>
<td>[ df=3 ]</td>
</tr>
<tr>
<td>Assault</td>
<td>5% (1)</td>
<td>18.6% (1)</td>
<td>[ p=.265 ]</td>
</tr>
<tr>
<td>Falls</td>
<td>40% (8)</td>
<td>32.9% (8)</td>
<td></td>
</tr>
<tr>
<td>Missing</td>
<td>8.6% (6)</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

There was no significant differences found on comparison of the categories for nature of injury: Road Traffic Accident (RTA), Sports, Assault and Accidental.
The CT (Computerised axial tomography) scan reports at the time of the injury were examined and there were no significant differences ($x^2=3.46$, df=6, $p=38$, see Table 3.4) were found on comparison of the categories from the CT reports: diffuse/bilateral, left / fronto / temporal / parietal, right fronto / temporal / parietal, basal / occipital, normal, subdural haemorrhage (SDH) or Subarachnoid haemorrhage (SAH).

**TABLE 3.4** The CT reported results of the group who participated in the study (*participants*) and the target sample group (*overall target sample*), both groups were 1 year or more following TBI and not more than 5 years had passed since their discharge from the rehabilitation unit.

<table>
<thead>
<tr>
<th>Category</th>
<th>Participants</th>
<th>Target sample</th>
<th>Statistical analysis</th>
</tr>
</thead>
<tbody>
<tr>
<td>Diffuse/bilateral</td>
<td>47.6% (10)</td>
<td>33.3% (24)</td>
<td></td>
</tr>
<tr>
<td>Left fronto/temporal/parietal</td>
<td>38.1% (8)</td>
<td>24.6% (17)</td>
<td></td>
</tr>
<tr>
<td>Right fronto/temporal/parietal</td>
<td>9.5% (2)</td>
<td>14.5% (10)</td>
<td>$x^2=3.46$ df=6 $p=.383$</td>
</tr>
<tr>
<td>Basal/occipital</td>
<td>4.8 (1)</td>
<td>2.9 % (2)</td>
<td></td>
</tr>
<tr>
<td>Normal</td>
<td>2.9% (2)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>SDH</td>
<td>2.9% (2)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>SAH</td>
<td>2.9% (2)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>missing</td>
<td>16 % (11)</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

In summary, inspection of the data indicated that the study participants were representative of the sample population as a whole. Although the sample size is small (n = 21) the present sample does not differ significantly from the target population on important demographic variables like age, gender, time since injury, nature of injury, and CT report details. However, the present study participants do differ on the categories of GCS severity, which is highest in the severe range in the participant group. This finding shows that the sample has a significantly number of severely head injured participants when compared to the overall target sample.

### 3.1.2 The sample

Out of the total 21 participants there were 3 women (mean age 34 years and s.d.14.7 years) and 18 men (41 years, s.d.18.7 years). All the participants completed each of the measures and 15 significant others completed a sleep quality questionnaire (PSQI). Table 3.5 shows the background details and injury characteristics of this sample. The mean GCS rating for the group was in the moderate to severe range (8.4, s.d 3.6). The
average length of PTA was within the severe category of around 6 days (142.3 hours, s.d 57.4 hours). The average length of time since the injury had been sustained was 3.7 years (s.d 1.1 years). All participants passed the screening measures; scored over 3 on the screening measure the clock drawing test and produced an average score of 27.6 (s.d 4.3) on the Mini-Mental State Exam.

**TABLE 3.5** Background details and injury characteristics of the study participants.

<table>
<thead>
<tr>
<th></th>
<th>mean (s.d.)</th>
<th>percentage (n)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Age</strong></td>
<td>40 (18)</td>
<td></td>
</tr>
<tr>
<td><strong>Gender</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>male</td>
<td></td>
<td>81% (17)</td>
</tr>
<tr>
<td>female</td>
<td></td>
<td>19% (4)</td>
</tr>
<tr>
<td><strong>GCS</strong></td>
<td>8.4 (3.6)</td>
<td></td>
</tr>
<tr>
<td><strong>PTA (days)</strong></td>
<td>142.3 (57.4)</td>
<td></td>
</tr>
<tr>
<td><strong>Time since injury (years)</strong></td>
<td>3.7 (1.1)</td>
<td></td>
</tr>
<tr>
<td><strong>Time in rehab (days)</strong></td>
<td>43 (66.6)</td>
<td></td>
</tr>
<tr>
<td><strong>Skull fracture</strong></td>
<td></td>
<td>33% (7)</td>
</tr>
<tr>
<td><strong>Haematoma</strong></td>
<td></td>
<td>67% (14)</td>
</tr>
<tr>
<td><strong>Contusions</strong></td>
<td></td>
<td>48% (10)</td>
</tr>
<tr>
<td><strong>Diffuse swelling</strong></td>
<td></td>
<td>24% (5)</td>
</tr>
<tr>
<td><strong>NART Premorbid FSIQ</strong></td>
<td></td>
<td>104 (8.8)</td>
</tr>
</tbody>
</table>

The average length of time spent as a patient in the rehabilitation unit following sustaining the TBI was 43 days and this varied widely (s.d 57.4 days). CT scan reports at the time of the injury reported: 33 per cent had sustained a skull fracture (n=7), 67 per cent developed a haematoma, 48 per cent had contusions and 24 per cent were reported as diffuse swelling. The groups National Adult Reading Test (NART) estimated premorbid Full-Scale Intelligence Quotient (FSIQ) was within the average range (mean =104, s.d 8.8). The cause of the TBI in the 21 participants was: 21 per cent were in a RTA (n=9), 38 per cent had a fall (8), 14 per cent were assaulted (3) and 5 per cent sustained their injury while participating in a sporting event (1).
3.1.2 Gender

There were no significant differences between males and females on measures of: age, severity (GCS, PTA), HADS scores, HRSD, VAS-F and the VAS-F sub-scale of energy (see Table 3.6). However, an Independent Samples T Test identified significant differences on the measures: PSQI Global score ($t=2.091, df 19, p=.05$) and the Bentall fatigue score ($t=-2.7, df 17, p=.015$) across gender. The women’s mean scores: PSQI Global (mean = 13, s.d. 2.7); Bentall Fatigue (19.3, 9.7) and VAS-F fatigue (12.7, 11.3), were much higher than the male’s: PSQI Global (6.6, 5.1); Bentall Fatigue (7.9, 6.2) and VAS-F fatigue (34, 31.7). It was felt that because of these differences analysis could not be carried out on this mixed gender sample. Significant differences on key variables and a low female sample size meant that the women’s data was excluded from any further analysis. These gender differences are interesting and are examined in more depth later in the discussion.

Table 3.6 Gender differences between participant characteristics and study measures

<table>
<thead>
<tr>
<th></th>
<th>Male (n=18)</th>
<th>Female (n=3)</th>
<th>Statistical analysis</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age</td>
<td>41 (18.7)</td>
<td>34 (14.7)</td>
<td>$t=.613, df 19, p=.547$</td>
</tr>
<tr>
<td>GCS</td>
<td>8.5 (3.5)</td>
<td>8.3 (5.1)</td>
<td>$t=.054, df 19, p=.958$</td>
</tr>
<tr>
<td>PTA</td>
<td>139.3 (59.9)</td>
<td>169 (0)</td>
<td>$t=.68, df 19, p=.503$</td>
</tr>
<tr>
<td>FSIQ</td>
<td>103.5 (8.7)</td>
<td>109.3 (9)</td>
<td>$t=1.057, df 19, p=.305$</td>
</tr>
<tr>
<td>PSQI Global</td>
<td>6.6 (5.1)</td>
<td>13 (2.7)</td>
<td>$t=2.091, df 19, p=.05^*$</td>
</tr>
<tr>
<td>HADS total</td>
<td>9.5 (6.6)</td>
<td>16.3 (13.58)</td>
<td>$t=1.426, df 18, p=.171$</td>
</tr>
<tr>
<td>HADS anxiety</td>
<td>5.5 (4.3)</td>
<td>9.7 (9)</td>
<td>$t=1.330, df 18, p=.200$</td>
</tr>
<tr>
<td>HADS Depression</td>
<td>4.1 (3.5)</td>
<td>6.7 (4.7)</td>
<td>$t=1.132, df 18, p=.272$</td>
</tr>
<tr>
<td>HRSD</td>
<td>8.7 (8)</td>
<td>16 (9)</td>
<td>$t=1.443, df 18, p=.166$</td>
</tr>
<tr>
<td>Bentall Fatigue</td>
<td>7.9 (6.2)</td>
<td>19.3 (9.7)</td>
<td>$t=2.705, df 17, p=.015^*$</td>
</tr>
<tr>
<td>VAS-F</td>
<td>3.3 (2.3)</td>
<td>6.4 (3)</td>
<td>$z=-1.86, df 18, p=.06$</td>
</tr>
<tr>
<td>VAS-F energy</td>
<td>24.4 (13)</td>
<td>12.7 (11.3)</td>
<td>$z=-1.38, df 18, p=.168$</td>
</tr>
<tr>
<td>VAS-F fatigue</td>
<td>34 (31.7)</td>
<td>79.7 (4.9)</td>
<td>$z=-1.85, df 18, p=.064$</td>
</tr>
</tbody>
</table>
3.1.3 Sample participants
The 18 male participants severity ratings are shown in Table 3.7. The sample is predominantly from the severe TBI category (n = 11; 61 per cent) and the remaining 39 per cent (n=7) are in the mild / moderate range. Ages ranged from 21 to 69 years (mean = 41, s.d 18.7). Time since injury ranged from 2 to 5 years (mean = 3.62 years, s.d 1). Participants had spent between 4 to 285 days in rehabilitation hospital (mean =45.7, s.d 70). Estimated premorbid intelligence (NART FSIQ) ranged from 91 to 122 (mean = 103, s.d 8.5). Scores on the clock drawing test ranged from 5 to 10 (mean =8.8, s.d 1.3). Mini-mental state exam scores ranged from 23 to 30 (mean =28, s.d 4).

Thirty three per cent of the participants took no medications at all, 17 per cent anti-epileptic medication, 17 per cent prescribed (one of which was a controlled drug- MST) or over the counter analgesia regularly, 11 per cent medication for non-insulin dependant diabetes. Eleven per cent were prescribed prophylactic penicillin, 11 per cent took anti-depressant medication (SSRIs) and 11 per cent took herbal tablets for management of urinary frequency. Seventeen per cent admitted to regular recreational drug use (cannabis). Twenty two per cent were smokers, 33 per cent drank more than the recommended daily allowance of caffeine per day (RDA = 500 mg; Julien, 1996) and none of the participants consumed more than 24 units of alcohol per week.

Table 3.7 Severity by Glasgow Coma Scale of the 18 male participants

<table>
<thead>
<tr>
<th>Severity</th>
<th>GCS</th>
<th>Frequency</th>
<th>Per cent</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mild/moderate</td>
<td>&gt;=8</td>
<td>7</td>
<td>39</td>
</tr>
<tr>
<td>Severe</td>
<td>&lt;8</td>
<td>11</td>
<td>61</td>
</tr>
<tr>
<td>Total</td>
<td>18</td>
<td></td>
<td>100.0</td>
</tr>
</tbody>
</table>

In considering psychological distress: 17 per cent met “caseness” criteria with the HADS for clinical depression and 22 per cent for anxiety. On the HRSD 44 per cent met the normal range, 33 per cent mild, 17 per cent moderate and 6 per cent severe. The moderate and severe participants were attending a mental health care professional or
receiving treatment from their GP. In addition fatigue scores on the Bentall ranged from 2 to 31 (mean = 9.2, s.d 8.2), meeting the ranges: 61 per cent were *a little*, 27 per cent *somewhat*, 6 per cent *quite a lot* and 6 per cent *very much* fatigued. On the VAS-F energy scale scores ranged between 2 and 50 (50 indicating the highest score; mean = 23.5, s.d 13) and fatigue ranged from 0 to 123 (the highest rating achievable was 130; mean=39.2, s.d 37.5).
3.1.4 Sleep

The Pittsburgh Sleep Quality Index (PSQI) global, retrospective and significant other ratings are shown in Table 3.8. From the 18 male participants, the mean score on the PSQI was 6.6 (s.d=5.1). The 15 significant other’s ratings of their partners sleep quality mean score was 6.3 (s.d=4.9). The participant’s retrospective score of sleep quality before the injury was 3.2 (s.d=3.2). Poor sleep quality was measured by a score of more than 5 on the PSQI: 50 per cent of the sample (n=9) reported poor sleep quality at this level, 47 per cent (n=7) of the significant others rated the participant as having a poor sleep quality and 22 per cent (n=4) reported a premorbid poor sleep quality. Insomnia was indicated by a score of 8 or more on the PSQI: 22 per cent of the participant’s (n= 4) responded within the insomnia range, 22 per cent of the significant others (n=4) and 6 per cent (n=1) reported premorbid levels within the insomnia range.

Table 3.8

i) Pittsburgh Sleep Quality Index scores: Global, Retrospective and Significant other.

<table>
<thead>
<tr>
<th></th>
<th>Mean (s.d)</th>
</tr>
</thead>
<tbody>
<tr>
<td>PSQI global score</td>
<td>6.6 (5.1)</td>
</tr>
<tr>
<td>PSQI significant other</td>
<td>6.3 (4.9)</td>
</tr>
<tr>
<td>PSQI retrospective global score</td>
<td>3.2 (3.2)</td>
</tr>
</tbody>
</table>

ii) Sleep quality: Poor sleep, retrospective poor sleep and significant others ratings of poor sleep.

<table>
<thead>
<tr>
<th></th>
<th>% (n)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Poor sleep quality</td>
<td>50% (9)</td>
</tr>
<tr>
<td>Significant other ratings of poor sleep quality</td>
<td>47% (7)</td>
</tr>
<tr>
<td>Retrospective poor sleep quality</td>
<td>22% (4)</td>
</tr>
</tbody>
</table>

*Poor sleep indicated by a PSQI global score of greater than 5.*

iii) Diagnosis of Insomnia: Insomnia, retrospective insomnia and Significant other ratings of insomnia.

<table>
<thead>
<tr>
<th></th>
<th>% (n)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Insomnia</td>
<td>22% (4)</td>
</tr>
<tr>
<td>Significant other ratings of insomnia</td>
<td>27% (4)</td>
</tr>
<tr>
<td>Retrospective insomnia</td>
<td>6% (1)</td>
</tr>
</tbody>
</table>

*Insomnia indicated by a PSQI global score of greater than 8.*
More than half of the sample reported having poor sleep and a third reported levels that indicated insomnia. Significant other ratings were lower than the participants and retrospective participant ratings were lower again.

The frequencies for sleep quality and TBI severity are detailed in Table 3.9. It is also important to consider the relationship between severities of TBI and sleep quality responses. The highest ratings of poor sleep quality were in the severe TBI group; 42 per cent (n=9) reported having a poor sleep quality. Furthermore, only 19 per cent (n=4) of the severe group reported a good sleep quality, whereas, 19 per cent (n=4) the mild moderate group reported a good sleep quality and the same percentage reported a bad sleep quality.

Table 3.9 Sleep quality and severity of traumatic brain injury.

<table>
<thead>
<tr>
<th>Sleep quality</th>
<th>TBI: Severity</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>mild/ moderate</td>
</tr>
<tr>
<td></td>
<td>frequency</td>
</tr>
<tr>
<td>Bad</td>
<td>4</td>
</tr>
<tr>
<td>Good</td>
<td>4</td>
</tr>
</tbody>
</table>
3.2 QUANTITATIVE ANALYSIS

The data were analysed using the SPSS 10 statistical software package. First they were checked for normality and variables showing significant skewness or kurtosis were transformed. The following were found to depart significantly from normality and all were positively skewed: length of hospitalisation for rehabilitation (skewness=3.2, standard error of skewness=.5), HADS total score (1.3, .5); HADS Anxiety (1.1, .5); HADS Depression (1.4, .5), HRSD (1.033, .512), Bentall fatigue (1.2, .5), Cigarettes per day (3.4, .5) and Alcohol per week (1.2, .5), and a log transform was carried out. PTA was unable to be transformed sufficiently to perform parametric tests on it, therefore PTA was eliminated from this analysis and GCS was used as the sole measure of severity of TBI.

3.2.1 HYPOTHESIS 1: Characteristics Predictive of Sleep Quality

A backward multiple regression analysis was carried out using age, GCS, time since injury, time in rehabilitation, HADS anxiety, HADS depression, HRSD, VAS-fatigue, Bentall fatigue and pain as the independent variables and PSQI Global score as the dependent variable.

The Adjusted R Square was 0.72, showing that over 70 per cent of the variance in the dependant variable was accounted for by the independent variables. The two depression measures were found to be the best predictors: HRSD and HADS depression (F=22.8, df=2,15,17, p<.001); see table 3.10, which shows ANOVA output from SPSS. The backward multiple regression analysis settled on HRSD and HADS depression as the best predictors of sleep quality. Examinations of the standardised beta coefficients found that HRSD and HADS depression variables were the only variables of those entered into the regression analysis that produced statistically significant beta coefficients. Demonstrating the contribution made by these 2 variables.

Therefore, depression as measured by HRSD and HADS, was the best predictor of sleep quality and adding in any other variables did not add significantly to the variance
accounted for in the dependent variable. Hypothesis 1 stated that these variables would be predictive of sleep quality; while this is true of the two depression measures this was not true of the other variables.

Table 3.10 SPSS output from Multiple Regression showing ANOVA table

<table>
<thead>
<tr>
<th>Model</th>
<th>Sum of Squares</th>
<th>df</th>
<th>Mean Square</th>
<th>F</th>
<th>Sig.</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Regression</td>
<td>365.423</td>
<td>10</td>
<td>36.542</td>
<td>3.328</td>
</tr>
<tr>
<td></td>
<td>Residual</td>
<td>76.855</td>
<td>7</td>
<td>10.979</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Total</td>
<td>442.278</td>
<td>17</td>
<td></td>
<td></td>
</tr>
<tr>
<td>2</td>
<td>Regression</td>
<td>365.118</td>
<td>9</td>
<td>40.569</td>
<td>4.206</td>
</tr>
<tr>
<td></td>
<td>Residual</td>
<td>77.160</td>
<td>8</td>
<td>9.645</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Total</td>
<td>442.278</td>
<td>17</td>
<td></td>
<td></td>
</tr>
<tr>
<td>3</td>
<td>Regression</td>
<td>365.701</td>
<td>8</td>
<td>45.588</td>
<td>5.289</td>
</tr>
<tr>
<td></td>
<td>Residual</td>
<td>77.577</td>
<td>9</td>
<td>8.620</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Total</td>
<td>442.278</td>
<td>17</td>
<td></td>
<td></td>
</tr>
<tr>
<td>4</td>
<td>Regression</td>
<td>363.878</td>
<td>7</td>
<td>51.983</td>
<td>6.630</td>
</tr>
<tr>
<td></td>
<td>Residual</td>
<td>78.399</td>
<td>10</td>
<td>7.840</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Total</td>
<td>442.278</td>
<td>17</td>
<td></td>
<td></td>
</tr>
<tr>
<td>5</td>
<td>Regression</td>
<td>361.633</td>
<td>6</td>
<td>60.272</td>
<td>8.221</td>
</tr>
<tr>
<td></td>
<td>Residual</td>
<td>80.645</td>
<td>11</td>
<td>7.331</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Total</td>
<td>442.278</td>
<td>17</td>
<td></td>
<td></td>
</tr>
<tr>
<td>6</td>
<td>Regression</td>
<td>360.071</td>
<td>5</td>
<td>71.814</td>
<td>10.357</td>
</tr>
<tr>
<td></td>
<td>Residual</td>
<td>83.207</td>
<td>12</td>
<td>6.934</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Total</td>
<td>442.278</td>
<td>17</td>
<td></td>
<td></td>
</tr>
<tr>
<td>7</td>
<td>Regression</td>
<td>354.288</td>
<td>4</td>
<td>88.572</td>
<td>13.086</td>
</tr>
<tr>
<td></td>
<td>Residual</td>
<td>87.990</td>
<td>13</td>
<td>6.768</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Total</td>
<td>442.278</td>
<td>17</td>
<td></td>
<td></td>
</tr>
<tr>
<td>8</td>
<td>Regression</td>
<td>342.192</td>
<td>3</td>
<td>114.064</td>
<td>15.955</td>
</tr>
<tr>
<td></td>
<td>Residual</td>
<td>100.086</td>
<td>14</td>
<td>7.149</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Total</td>
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<td>17</td>
<td></td>
<td></td>
</tr>
<tr>
<td>9</td>
<td>Regression</td>
<td>332.790</td>
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<td>166.395</td>
<td>22.796</td>
</tr>
<tr>
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<td>109.488</td>
<td>15</td>
<td>7.299</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Total</td>
<td>442.278</td>
<td>17</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Predictors:

a Predictors: (Constant), GCS, HRSD, INJAGO, AGE, DIRS, VAS-F, BENT, PAIN, HADSD, HADSA
b Predictors: (Constant), GCS, HRSD, INJAGO, AGE, VAS-F, BENT, PAIN, HADSD, HADSA
c Predictors: (Constant), GCS, HRSD, INJAGO, AGE, VAS-F, BENT, PAIN, HADSD
d Predictors: (Constant), GCS, HRSD, INJAGO, AGE, VAS-F, PAIN, HADSD
e Predictors: (Constant), GCS, HRSD, INJAGO, AGE, PAIN, HADSD
f Predictors: (Constant), GCS, HRSD, INJAGO, AGE, HADSD
g Predictors: (Constant), GCS HRSD, AGE, HADSD
h Predictors: (Constant), GCS, HRSD, HADSD
i Predictors: (Constant), HRSD, HADSD
j Dependent Variable: GPSQI
3.2.2 HYPOTHESIS 2: The Relationship Between Severities of Traumatic Brain Injury and Sleep Quality.

A One Way ANOVA was used to investigate the relationship between severities of traumatic brain injury and sleep quality. Table 3.7 (page 53) shows the severity groups of the 18 male participants: the mild / moderate severity group (n = 7) and the severe group (n=11). PSQI scores were the independent variables and two injury severity groups (mild / moderate and severe GCS ratings) were used as the test factor.

There were no significant differences observed on any of the component or the global PSQI scores between the two severity groups.

There were no significant differences found between the two different injury severity groups (mild / moderate and severe GCS ratings) on the component and global scores of sleep quality. Hypothesis 2 stated that mild/moderate TBI participant ratings of sleep quality would be higher than those with a severe TBI. This distinction was not found and hypothesis 2 was rejected.
3.2.3 HYPOTHESIS 3: The Relationship Between Severity of Traumatic Brain Injury and Daytime Fatigue.

An Independent samples T-test was used to investigate the relationship between severity of traumatic brain injury and the development of daytime fatigue on the Bentall inventory. There were no significant differences found between the severity groups on the Bentall ($t=-.537$, df=17, $p=0.574$). The mild / moderate and severe TBI groups showed no difference on the Bentall measure of fatigue.

Non-parametric tests were used to investigate the Visual Analogue Fatigue Scale measure of fatigue (VAS-F and its two sub components VAS-F energy, and VAS-F fatigue). Mann-Whitney tests were used to investigate the relationship between severity of traumatic brain injury and the development of daytime fatigue. Using a measure of daytime fatigue (the 3 VAS-F scores) as the independent variable and injury severity (mild / moderate and severe GCS ratings) as the grouping variable.

There was no significant differences found between the severity groups on the measures of fatigue: VAS-F ($z =-.41; p>0.05$), VAS-F energy ($z =-.68; p>0.05$) and VAS-F fatigue ($z =-.36; p>0.05$), and Bentall ($z =-.41; p>0.05$). The mild / moderate and severe TBI groups showed no significant difference on the measures of fatigue.

Measures of daytime fatigue were no different, in this sample, across the different TBI severity groups. Therefore, hypothesis 3, which stated that the daytime fatigue of the mild/moderate TBI group would be higher than the severely brain injured group, was not supported.
3.2.4 HYPOTHESIS 4: Self-Report & Significant Other Sleep Quality Ratings

A Paired and Independent Samples T-tests were used to examine the global scores on the PSQI rated as: 1) the participant current rating versus their significant other rating, 2) the participant’s current rating versus their retrospective sleep quality ratings, and 3) the participant retrospectively and the significant other rating of the participant’s sleep quality. There was no significant difference ($t=.36$, df=14, $p=.73$) between, the overall PSQI global scores for the participant and the significant other’s ratings of the participant’s current sleep quality. A significant difference was found between the participant current rating and their retrospective sleep quality rating ($t=2.94$, df=17, $p=.01$) and the participant’s retrospective rating and their significant other rating of sleep quality ($t=2.39$, df=14, $p=.03$).

T-test analysis was also used to investigate the participant and significant other scores on each of the seven component and global scores of the sleep quality measure, in order to further investigate any difference between self-report and observed sleep quality ratings. The seven component scores are made up of nineteen individual items; these items generate the seven component scores: subjective sleep quality, sleep latency, sleep duration, habitual sleep efficiency, sleep disturbances, use of sleeping medication and daytime dysfunction. Table 3.11 shows the results of the analysis between the two groups.

There was no significant difference between the severity groups on the components of subjective sleep quality ($t = 1.38$, df=14, $p = .21$), sleep latency ($t =-.81$, df=14, .14), sleep duration ($t=.45$, df=14, $p=.4$), habitual sleep efficiency ($t=.37$, df=14, $P=.55$), use of sleeping medication ($t=1.0$, df=14, $P=0.70$), daytime dysfunction ($t=1.0$, df=14, $P=.24$) and PSQI global ($t=.36$, df=14, $p=.15$). There was a significant difference on the component score of sleep disturbances ($t=-3.5$, df = 14, $p<0.01$). The mean score for the significant other reports of sleep disturbance was higher (mean = 1.47) than the participant responses (mean =1). Therefore, the significant others rated the sleep disturbance item significantly higher than the participants did.
Table 3.11 The mean scores and Paired Samples T-test analysis for the ratings of the component and global score of the Pittsburgh Sleep Quality Inventory (PSQI) between the significant other and participant groups.

<table>
<thead>
<tr>
<th>Pair</th>
<th>Component</th>
<th>Participant Mean (s.d)</th>
<th>Significant other Mean (s.d)</th>
<th>t</th>
<th>df</th>
<th>Sig.</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>N=15</td>
<td>N=15</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Pair 1</td>
<td>Subjective sleep quality</td>
<td>1.13(1.06)</td>
<td>.93(.96)</td>
<td>1.38</td>
<td>14</td>
<td>&gt;0.05</td>
</tr>
<tr>
<td>Pair 2</td>
<td>Sleep latency</td>
<td>1.20(1.01)</td>
<td>1.33(1.11)</td>
<td>-0.81</td>
<td>14</td>
<td>&gt;0.05</td>
</tr>
<tr>
<td>Pair 3</td>
<td>Sleep duration</td>
<td>0.87(1.19)</td>
<td>0.80(1.08)</td>
<td>0.44</td>
<td>14</td>
<td>&gt;0.05</td>
</tr>
<tr>
<td>Pair 4</td>
<td>Habitual sleep efficiency</td>
<td>0.93(1.22)</td>
<td>0.87(1.25)</td>
<td>0.37</td>
<td>14</td>
<td>&gt;0.05</td>
</tr>
<tr>
<td>Pair 5</td>
<td>Sleep disturbances</td>
<td>1.00(0.53)</td>
<td>1.47(0.64)</td>
<td>-3.5</td>
<td>14</td>
<td>0.004*</td>
</tr>
<tr>
<td>Pair 6</td>
<td>Use of sleeping medication</td>
<td>0.20(0.77)</td>
<td>0.00(0.00)</td>
<td>1.0</td>
<td>14</td>
<td>&gt;0.05</td>
</tr>
<tr>
<td>Pair 7</td>
<td>Daytime dysfunction</td>
<td>0.87(0.99)</td>
<td>0.67(0.90)</td>
<td>1.0</td>
<td>14</td>
<td>&gt;0.05</td>
</tr>
<tr>
<td>Pair 8</td>
<td>PSQI Global</td>
<td>6.13(5.33)</td>
<td>5.93(4.88)</td>
<td>0.36</td>
<td>14</td>
<td>&gt;0.05</td>
</tr>
</tbody>
</table>

* Significant; p less than or equal to 0.01

There was a difference observed on the component for sleep disturbances; the significant others observed significantly greater incidents of sleep disturbance than the participants reported. This difference did not affect the global scores on the PSQI: the participant global responses were no different to their significant other’s. The Global measure was used to denote sleep quality and therefore hypothesis 4, which predicted that the significant other and TBI sufferer’s measures on a sleep quality rating scale would differ, cannot be supported and the hypothesis was rejected.
3.2.5 HYPOTHESIS 5: The Relationship Between Severity of Traumatic Brain Injury and Sleep Quality with Psychological Distress and Alcohol, Caffeine and Nicotine Use.

The four groups were: mild moderate injury and good sleep (n=4), mild moderate injury and bad sleep (n=3), severe and good sleep (n=4) and severe and bad sleep (n=7).

Due to low participant numbers in each of the four groups non-parametric statistics were employed. Good and bad sleep quality & severity (by GCS) frequencies are presented in Table 3.12. A Kruskal Wallis non-parametric test was used to explore the relationship between the independent variables. HRSD was significant (H =12.86, df 3 p=.005). Mean scores on the HRSD were as follows: mild/moderate good sleep 3.6; severe good sleep 3.0; mild/moderate bad sleep 18.5; severe bad sleep 14.1. The significant difference in the HRSD scores would appear to arise from differences between good and bad sleep quality. The other measures, HADS depression (H=4.92, df 3 p>0.05), HADS anxiety (H=5.24, df 3 p>0.05), alcohol (H=2.67, df 3 p>0.05), nicotine (H=3.98, df 3 p>0.05), and caffeine use (H=.59, df 3 p>0.05) were not significant.

Table 3.12 Frequencies of good and bad sleep quality & severity (by GCS) frequency

<table>
<thead>
<tr>
<th>Severity By GCS</th>
<th>mild/mod</th>
<th>severe</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>Good and bad sleep quality</td>
<td>n</td>
<td>n</td>
<td></td>
</tr>
<tr>
<td>good</td>
<td>4</td>
<td>4</td>
<td>8</td>
</tr>
<tr>
<td>bad</td>
<td>3</td>
<td>7</td>
<td>10</td>
</tr>
<tr>
<td>Total</td>
<td>7</td>
<td>11</td>
<td>18</td>
</tr>
</tbody>
</table>

While a difference between the four groups was identified this difference was not as Hypothesis 5 predicted; that the moderate and mild TBI with poor sleep quality would report higher ratings on measures of psychological distress and nicotine, caffeine and alcohol use. In contrast it was revealed that the two groups with poor sleep quality, in both severe and mild/moderate TBI severity groups, had higher ratings on the depression scale (HRSD). Therefore, Hypothesis 5 was rejected.
3.3 INTERVIEW ANALYSIS

The Intercoder reliability was 85 percent. The final label and description of the categories and illustrative examples from the data are presented below. Frequencies and percentages of the occurrence of each category for the four groups: good sleeper, bad sleeper, insomnia (male) and insomnia (female) are presented in tables below (Table 3.13, 3.14 and 3.15). Results are presented under the headings of: experiences of sleep before the TBI, current sleep experience and coping with sleeplessness.

3.1.1 Experiences of sleep before the TBI:

Good sleepers: Many of the participants talked about their sleep before the TBI as being “normal” or “routine”. This usually involved an almost disciplined routine before bedtime, a set time to retire, getting over to sleep quickly and sleeping straight through until the next morning.

HQ03: Hmm well I’d usually—a creature of habit you see—we would watch the telly for a couple of hours and then after the news—I mean between eleven and eleven thirty—I was always in my bed and we used to go to sleep very quickly.

Mostly they reported a deep sleep. However, some reported getting up through the night to go to the toilet or, even, in two cases to go to work. Most of the good sleepers reported that they had no complaints about their sleep before the TBI.
Waking up and getting up was often reported as trouble free and one individual reported that he had never used an alarm clock, he had always awoken naturally with the morning daylight. Although undertaking shift work was discussed as part of that routine, none of the good sleepers commented that work stress affected their sleep.

Individuals frequently stated that they had no complaints about their sleep before the injury. Before going to sleep they might watch TV, have an alcoholic drink or read a book, as the quote above exemplifies, but this behaviour was not a spontaneous activity, whatever it may be. The activity was part of a bedtime routine, which took place over a set time period and preceded sleep.

**Bad sleepers:** Tended to talk about similar routines, but they were more inclined to report further memories of disturbed sleep prior to the injury. They talked about sleep disturbance as if it was to be expected and tolerated; an inconvenience or personal eccentricity within their individual sleep pattern. Individuals might get up through the night. One man awoke between two and three o’clock, every morning, to eat a bowl of cornflakes and continued to do so even after the injury. Lifestyle was also considered to be somewhat more erratic, which led to less routine at bedtime. Two individuals mentioned alcohol as an explanation for their good sleep experience.

**Insomniacs:** This group spoke about their sleep experience in a similar way to the bad sleepers. However, an additional theme emerged, sleep for some of the individuals in this group took on an idealised quality. The reminiscence followed a pattern, which generally stated that sleep was very good back then.

SX02: Hmmm (smiles). Peaceful, quite deep and uninterrupted...Cosy.

Activities prior to bedtime seemed less planned and more spontaneous. Interviewees often said that they might watch TV or read a book, but it depended on how they felt. A major theme with the insomniacs was how they woke up in the morning. Often they
talked about sleeping as late as possible, putting off getting up so they could spend as long as possible in beds.
### 3.3.2 Current sleep experience

#### Table 3.14 Current sleep experience: Frequencies and percentages of units falling into each category

<table>
<thead>
<tr>
<th>Category</th>
<th>Good Sleepers</th>
<th>Bad Sleepers</th>
<th>Insomnia Males</th>
<th>Insomnia Females</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Preparation for sleep</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Go to bed same time</td>
<td>88%(7)</td>
<td>0</td>
<td>0</td>
<td>12%(1)</td>
</tr>
<tr>
<td>Go to bed later</td>
<td>0</td>
<td>40%(2)</td>
<td>40%(2)</td>
<td>20%(1)</td>
</tr>
<tr>
<td>Go to bed earlier</td>
<td>0</td>
<td>50%(1)</td>
<td>50%(1)</td>
<td>0</td>
</tr>
<tr>
<td>Unable to initiate sleep</td>
<td>13%(1)</td>
<td>25%(2)</td>
<td>37%(3)</td>
<td>25%(2)</td>
</tr>
<tr>
<td>Thoughts stop sleep</td>
<td>0</td>
<td>20%(1)</td>
<td>44%(2)</td>
<td>40%(2)</td>
</tr>
<tr>
<td>Anticipatory anxiety about the next day stops sleep</td>
<td>0</td>
<td>50%(1)</td>
<td>50%(1)</td>
<td>0</td>
</tr>
<tr>
<td>Do not get to sleep till early hours</td>
<td>0</td>
<td>25%(1)</td>
<td>50%(2)</td>
<td>25%(1)</td>
</tr>
<tr>
<td><strong>Sleep maintenance</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Prolonged sleep</td>
<td>0</td>
<td>50%(2)</td>
<td>50%(2)</td>
<td>0</td>
</tr>
<tr>
<td>Wake up to go to toilet</td>
<td>50%(1)</td>
<td>0</td>
<td>0</td>
<td>50%(1)</td>
</tr>
<tr>
<td>Wake up to eat</td>
<td>0</td>
<td>50%(1)</td>
<td>50%(1)</td>
<td>0</td>
</tr>
<tr>
<td>Wake up with pain</td>
<td>0</td>
<td>50%(1)</td>
<td>50%(1)</td>
<td>0</td>
</tr>
<tr>
<td>Wake up with cramps</td>
<td>50%(1)</td>
<td>50%(1)</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Wake up snoring</td>
<td>0</td>
<td>100%(1)</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Wake up after a vivid dream</td>
<td>0</td>
<td>50%(2)</td>
<td>25%(1)</td>
<td>25%(1)</td>
</tr>
<tr>
<td>Sleep has improved</td>
<td>67%(2)</td>
<td>33%(1)</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Sleep is longer</td>
<td>50%(1)</td>
<td>50%(1)</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Sleep is more relaxed</td>
<td>50%(1)</td>
<td>50%(1)</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Sleep is the same</td>
<td>88%(7)</td>
<td>12%(1)</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Sleep is worse</td>
<td>11%(1)</td>
<td>11%(1)</td>
<td>44%(4)</td>
<td>34%(3)</td>
</tr>
<tr>
<td>Sleep is deeper</td>
<td>0</td>
<td>100%(2)</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>While asleep is hard to awaken</td>
<td>0</td>
<td>67%(2)</td>
<td>33%(1)</td>
<td>0</td>
</tr>
<tr>
<td>Sleep is lighter</td>
<td>0</td>
<td>0</td>
<td>100%(1)</td>
<td>0</td>
</tr>
<tr>
<td>Wake up naturally</td>
<td>100%(3)</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Have plans next day</td>
<td>50%(2)</td>
<td>0</td>
<td>0</td>
<td>50%(2)</td>
</tr>
<tr>
<td><strong>Waking</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Sleep late if can</td>
<td>0</td>
<td>20%(1)</td>
<td>40%(2)</td>
<td>40%(2)</td>
</tr>
<tr>
<td>Experience angst dreams and wake up</td>
<td>25%(1)</td>
<td>25%(1)</td>
<td>25%(1)</td>
<td>25%(1)</td>
</tr>
<tr>
<td>Dreams become confused with reality unsure whether awake or asleep</td>
<td>25%(1)</td>
<td>25%(1)</td>
<td>25%(1)</td>
<td>25%(1)</td>
</tr>
<tr>
<td>No memory of dreams anymore</td>
<td>0</td>
<td>0</td>
<td>100%(1)</td>
<td>0</td>
</tr>
<tr>
<td>Sleep problems in hospital</td>
<td>0</td>
<td>0</td>
<td>50%(1)</td>
<td>50%(1)</td>
</tr>
<tr>
<td>Sleeping tablets do not help</td>
<td>0</td>
<td>0</td>
<td>100%(2)</td>
<td>0</td>
</tr>
<tr>
<td>Work/shift patterns</td>
<td>67%(2)</td>
<td>0</td>
<td>0</td>
<td>33%(1)</td>
</tr>
<tr>
<td>Improvement is hopeless and sleep unpredictable</td>
<td>0</td>
<td>22%(2)</td>
<td>44%(4)</td>
<td>33%(3)</td>
</tr>
<tr>
<td>Cannot concentrate</td>
<td>0</td>
<td>0</td>
<td>50%(1)</td>
<td>50%(1)</td>
</tr>
<tr>
<td>Make joke about sleep pattern</td>
<td>0</td>
<td>0</td>
<td>50%(1)</td>
<td>50%(1)</td>
</tr>
<tr>
<td><strong>Miscellaneous</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Taking SSRI</td>
<td>0</td>
<td>100%(1)</td>
<td>0</td>
<td>0</td>
</tr>
</tbody>
</table>

**Good sleepers:** Often replied that sleep was almost exactly the same, no change to their sleep pattern or bedtime routine; they went to bed, slept and got up in a similar fashion as before. One reported that he went bed earlier and another had difficulty getting over to sleep.

Sleep maintenance varied: one individual commented that he got up more frequently to go to the toilet, whilst another two complained of awakening through the night due to painful leg cramps. Most reported that sleep was the same as before the TBI, but one individual said it was worse. Sleep had improved for some and was reported to be longer in time length.
A general theme evolved that individuals had something to get up for. Some still rose at the same time even though their daytime activities had changed. This waking, at the same time as before, had an automatic quality about it. One man discussed it as if it was out of his control:

LT11: ...I still wake up about half six in the morning this is the exact time I would wake up for the last job I had, I automatically wake up at the time I would have, I cannae get back to sleep, I just get up, I dinnae have to get up but I do...

Not being sure whether they were asleep or awake, confusing dreams with reality was commented upon and shift working was often discussed in relation to current sleep.

**Bad sleepers:** More commonly reported going to bed later than they did before the accident. In addition, they more frequently reported being unable to get to sleep until later. When asleep they slept for longer than before. One man reported that his sleep had improved since the injury, he slept for longer periods at a time and generally felt more relaxed, but he suffered great anxiety when anticipating having to get up for occasional planned activities. Taking an antidepressant medication had improved one man's sleep quality. Another participant reported that his sleep was worse, he did not get over to sleep until about two or three o'clock in the morning, he was not tired, and he regularly suffered from pain and cramps due to orthopaedic injuries sustained at the time of his TBI.

Once asleep, the sleep was often prolonged, sleeping for longer periods than before. Individuals often woke up through the night to eat, due to pain and cramps or snoring. For some, sleep was the same as before the TBI and for some worse. For one man, sleep had improved and he reported it to be longer and more relaxed. Generally sleep appeared to be variable but mostly deeper and once asleep the individuals were hard to awaken.

Participants often reported sleeping later when they had the opportunity. Dreams might wake people from their sleep, or may become confused with reality when individuals were unsure whether they had thought about something or dreamt about it. A few of the
individuals were quite negative about the possibility of ever being able to improve their sleep. Additionally, it was often reported that sleeplessness had an unpredictable quality; it could go on for night after night.

**Insomniacs:** Most of the insomniac group reported having a worse sleep pattern when compared to their sleep experience before their injury. They tended to have difficulties initiating sleep. They were not tired at bedtime and unable to get to sleep at the times they wanted to. Often they reported that thoughts going through their mind inhibited the initiation of sleep. These thoughts tended to be described as everyday things, not to be confused with worries or ruminations.

HX13: OK say if I go to bed at eleven and can’t get to sleep until three, during that time your thinking about things, something, actually anything about Eh... living its something that I might be thinking about [is it worries?] its not- its different - its no problem if you don’t think about it but if you don’t think about that it would be something else- If you try and stop thinking about it, something else will come in -not that its- its- something else- about anything - its like what will I do tomorrow that sort of thing.

Some participants had more difficulties with sleep maintenance, getting over to sleep quickly but waking a few hours later and unable to get back over to sleep.

Often the individuals reported that they felt quite hopeless about their sleep and that there was little they could do about these sleep problems. Often they thought it was just something they had to deal with. If they had discussed it with their GP, often they would say tablets would not help because they were addictive and few thought there was any other help available. There was a general feeling of hopelessness about their sleep. Another theme encapsulated the unpredictability of the sleeplessness, as it may go on for night after night. Their sleep was generally worse than they had ever experienced it, it was unpredictable and there was nothing they could do about it.

Two individuals reported bad sleep experiences in hospital, one reported being unable to sleep on the ward because of the noise and activity and people waking up so early and he
added that he had found talking to the night staff comforting. Another woman reported experiencing terrible pain after her injury and this had stopped her from sleeping while in hospital.

LN12: at the beginning when I was in hospital and after it was hard to sleep because of my sore head, I used to joke that I needed a really soft pillow when I came home, the pillows in hospital were like bricks and the beds were so uncomfortable, I was just so tired all the time.

They were also more likely to joke about their sleep pattern, “What sleep pattern?”
3.3.3 Coping with sleeplessness:

Table 3.15 Coping with sleeplessness: Frequencies and percentages of units falling into each category

<table>
<thead>
<tr>
<th>Categories</th>
<th>Good Sleepers</th>
<th>Bad Sleepers</th>
<th>Insomnia Males</th>
<th>Insomnia Females</th>
</tr>
</thead>
<tbody>
<tr>
<td>Daytime nap</td>
<td>14% (1)</td>
<td>0</td>
<td>57% (4)</td>
<td>29% (2)</td>
</tr>
<tr>
<td>Read till drowsy</td>
<td>67% (1)</td>
<td>0</td>
<td>0</td>
<td>33% (2)</td>
</tr>
<tr>
<td>Lie in bed and wait for sleep</td>
<td>25% (2)</td>
<td>25% (2)</td>
<td>38% (3)</td>
<td>12% (1)</td>
</tr>
<tr>
<td>Reposition in bed/ get comfy</td>
<td>0 (1)</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Prepare for next day</td>
<td>0</td>
<td>0</td>
<td>100% (1)</td>
<td>0</td>
</tr>
<tr>
<td>Clean car</td>
<td>0</td>
<td>0</td>
<td>100% (1)</td>
<td>0</td>
</tr>
<tr>
<td>Listen to music</td>
<td>25% (1)</td>
<td>50% (2)</td>
<td>0</td>
<td>25% (1)</td>
</tr>
<tr>
<td>Watch TV</td>
<td>17% (1)</td>
<td>17% (1)</td>
<td>66% (4)</td>
<td>0</td>
</tr>
<tr>
<td>Go on Internet</td>
<td>0 (1)</td>
<td>33% (1)</td>
<td>33% (1)</td>
<td>33% (1)</td>
</tr>
<tr>
<td>Smoke a cigarette</td>
<td>0</td>
<td>50% (1)</td>
<td>59% (1)</td>
<td>0</td>
</tr>
<tr>
<td>Pace the house or outside</td>
<td>0</td>
<td>0</td>
<td>33% (1)</td>
<td>67% (2)</td>
</tr>
<tr>
<td>Go for a drive</td>
<td>0</td>
<td>0</td>
<td>100% (1)</td>
<td>0</td>
</tr>
<tr>
<td>Never had any sleep difficulties</td>
<td>100% (5)</td>
<td>0</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Get angry and frustrated</td>
<td>0</td>
<td>50% (1)</td>
<td>50% (1)</td>
<td>0</td>
</tr>
<tr>
<td>Sought HCP advice</td>
<td>0</td>
<td>0</td>
<td>100% (1)</td>
<td>0</td>
</tr>
</tbody>
</table>

The good sleepers: Mostly they would just lie in bed and wait for sleep to come or read till drowsy. One man reported napping through the day more since his injury and individuals might do something else if they could not sleep (listen to music and watch TV).

HC09: If I couldn’t sleep I can’t really say. I’ve never had any trouble after having the bump on the head, I thought I might have problems with headaches and things like that seemingly it was bad when I was in hospital but there is no pain now.

Generally they reported that they had never experienced sleep problems of not being able to sleep and could not imagine it.

The bad sleepers: Tended to lie in bed and wait for sleep to come or reposition themselves, in bed, to become more comfortable. Bad sleepers also reported a variety of solutions to sleeplessness: getting up and watching TV, listen to the radio, smoking and surfing the Internet. Negative automatic thoughts might follow sleeplessness. One man reported that he tended to get very angry and frustrated with himself when he could not sleep.
CS08: Curse Swear get furious tell myself not to be stupid but it doesn’t help.

**Insomniacs:** Often napped through the day or lay in bed longer in the morning to make up for missed sleep the night previously. Most commonly themes developed from beliefs about catching up on sleep later. Often they were resigned to wait for sleep to come and said they just waited until they fell asleep. Others reported watching TV, listening to music, playing on their computer or smoking a cigarette. More often, they would actually report leaving the bedroom to get something to eat or drink or go for a walk outside. Health care professional advice had often been taken about sleep disturbance, but understanding why the advice would help was never discussed.

FT04: ...Through the day is the same, I’ve been told by the physio to rest through the day to pace myself I just rest and relax I don’t sleep...I just stay in my bed because people have said get up and do things but what am I going to get up and do in the middle of the night...sometimes I have a read.

Cognitive deficits increased the likelihood of inappropriate nocturnal behaviour occurring, particularly with one man who was often disorientated to time, his wife often found him at two or three o’clock in the morning outside washing or vacuuming his car.

**3.3.4 Section summary**

The number of units falling into each category was too low to use statistical analysis (e.g. Chi Square) to investigate differences between the frequency of each category in the four groups of good sleepers, bad sleepers, insomnia male and insomnia female. The interview analysis is summarised below in the three areas: experiences of sleep before the TBI, current sleep experience and coping with sleeplessness.

**Experience of sleep before the TBI** was characterised in the good sleepers by a more organised preparation for sleep, most commonly a rigid bedtime routine. They more frequently commented that their premorbid sleep was of a deep quality and were less likely to say that they had no complaints about their premorbid poor sleep quality. The
bad sleepers were more likely to report getting up through the night to eat and reported a greater experience of work stress, which they believed affected their sleep quality.

In the **current sleep experience** the good sleepers report a similar bedtime routine where many talked about going to sleep at the same time. Few issues regarding sleep maintenance were reported. The good sleepers were more likely to report waking up naturally in the morning. The good sleepers did not report high cognitive arousal, such as thoughts racing through their head, as common occurrences that stopped sleep initiation. However, the bad sleepers (20 per cent), and those with insomnia (males, 40 per cent and females, 40 per cent) commonly reported experiencing cognitive hyper arousal, which stopped them from getting to sleep. The bad sleepers were more likely to report snoring being a difficulty with 100 per cent reporting this as a problem for sleep maintenance. The insomniac males were more likely to report lighter sleep (100 per cent) following TBI. Additionally the insomniac males were more likely to report no memory of dreams (100 per cent) since the TBI. Sleep problems in hospital were more likely to be reported by the insomnia group (male=50 per cent, female = 50 per cent).

In the **coping with sleeplessness** section the good sleepers were more likely to comment that they did not know what they would do because they had no experience of the problem. However, the insomnia sufferers (males, 57 per cent and females, 29 per cent) more frequently said that they might cope with sleeplessness by taking a daytime nap. The insomnia patients more frequently reported coping with sleeplessness by taking part in physical or mentally arousing activities, such as: watching TV (66%), surfing the internet (66%), pacing the room (100%), going for a drive (100%). In addition, bad sleepers and insomniac males were most likely to report getting angry and frustrated as a way of coping with their sleeplessness.
4. DISCUSSION

4.1 SUMMARY OF RESULTS

This study investigated the variables characteristic of sleep disorder in eighteen males, one year after a TBI. In this sample, sixty-one per cent had a severe brain injury, as rated by a GCS score of less than eight. Fifty per cent had a poor sleep quality and twenty-two per cent met criteria for insomnia, as indicated by a measure that has been well standardised in TBI populations (Pittsburgh Sleep Quality Inventory; PSQI). These findings are lower than a recent post-acute insomnia study, which reported a thirty per cent frequency of sleep disturbance (Fichtenberg et al. 2002).

Firstly, this study found that among the demographic, affective and injury variables examined, the strongest relationship with sleep quality was found with depression, as measured by the HADS depression scale and the Hamilton Rating Scale for Depression (HRSD). The first hypothesis predicted that, amongst other variables, the depression scales (HADS and HRSD) would be predictive of sleep quality. Among this TBI group sleep quality was linked with the presence of depression. This finding is comparable with similar studies that have reported depression as predictive of sleep disturbance (Fichtenberg et al, 2002; Beetar et al, 1996).

However, at one year or more following injury the other identified variables, age, TBI severity levels (GCS), time since injury, length of hospitalisation for rehabilitation, HADS anxiety, daytime fatigue (Bentall, VAS-F), and pain, were not predictive without a depression measure (HRSD, HADS). This was surprising as Beetar et al, (1996) reported a strong link with pain and sleep disturbance.

Secondly, there were no significant differences found between the participants of different injury severities on the component and global scores of sleep quality. Hypothesis two stated that the less severe group (mild/moderate) sleep quality reports would be significantly worse than those with a severe TBI. The TBI group with good sleep quality reported normal mean ratings on the HRSD and the bad sleep quality group
reported scores within the moderate, mild and severe range on the depression scale. The predicted distinction was not found and hypothesis two was rejected.

Thirdly, measures of daytime fatigue were no different in this sample, across the different severity groups of TBI. Therefore, hypothesis three, which stated that the daytime fatigue of the mild/moderate TBI group would be higher than the severely brain injured group, was rejected.

Fourthly, sleep quality ratings for the participant and significant other did not differ significantly. The PSQI global measure has been used to denote sleep quality. The hypothesis, which predicted the significant other and TBI participant’s measures on a sleep quality rating scale would differ, was rejected.

Finally, the findings of a qualitative analysis, based on content analysis, investigated themes generated by a semi-structured interview, were reported. Analysis of the semi-structured interview identified categories of behaviour, which related to bad sleep quality, in the eighteen males and three females. Bad sleepers more often reported mental alertness, thoughts, inhibiting the initiation of sleep. Disturbed sleep due to snoring was also more problematic with the bad sleepers. The insomnia group were more likely to report having experienced poor sleep while in hospital, following recovery from the TBI. In addition the male insomnia group reported a lighter sleep quality since the TBI and were more likely to report that they had no memory of having had any dreams since their injury.

In summary, fifty per cent of the sample reported poor sleep quality and twenty two per cent of the participants scored within a clinically morbid range diagnostic of insomnia. Among the demographic, affective and injury variables examined the strongest relationship with sleep quality was depression. The significant other ratings were no different to the participant’s self-ratings. Interestingly, sleep quality rather than TBI severity appeared linked to depression. No relationship was found between severity of
TBI and sleep quality or daytime fatigue. Interview analysis revealed a pattern, which related to the good bad and insomniac sleep categories.

Results from this sample found slightly lower rates of sleep disturbance than in a comparable post acute population (Fichtenberg, et al., 2002). However, this finding is still more than double the rate of sleep disturbance reported in the normal population. Perhaps, depression could even be considered as secondary to insomnia, rather than secondary to TBI. The importance of evaluating treatments for insomnia in this group is highly apparent, because of the potential benefits.

4.1.1 Depression as the Most Important Predictor of Sleep Quality
Age, time since injury, time in rehabilitation, psychological distress, anxiety and pain were not significant predictors of sleep quality, without depression. This is surprising, as these have all been associated with sleep disturbance in previous studies (Clinichot et al, 1998; Beetar et al, 1996). Gender differences have been found in many studies (Levin, Mattis, Ruff, Eisenberg, Marshall & Tabaddor, 1987; Hibbard, Uysal, Sliwinski & Gordon, 1998; Clinchot et al. 1998). The females in this sample were excluded from the final analysis because of significant differences on the PSQI scores: all were significantly higher than the men. To allow for such differences in the future, a similar study might have to over sample women to compose a matched sample. Such gender differences might be explained by different health experiences following TBI. Hibbard, et al. (1998) found both age and gender to be significant covariates for many undiagnosed health issues. These health problems were: thyroid conditions, sleep difficulties, urinary incontinence and arthritis, and gender alone was a significant covariate for five health difficulties: weight change, change in texture and growth of hair and skin, body temperature change and headaches and frequent colds.

The HADS depression and the HRSD have been confirmed as sensitive measures in this sample of TBI patients. The number of participants was smaller than other comparable studies and this factor may have contributed. This, however, serves only to emphasize
the significance of the predictive power of these two measures of depression (HADS depression and the HRSD) in this study.

This study replicates the findings of previous research that depression measures are predictive of sleep disorder. In addition, the current study reported a relationship between sleep quality and depression, not with TBI severity. Participants who had a low sleep quality had a scored within clinical levels on the HRDS, good sleepers had normal scores. Fichtenberg et al., (2000) reported a correlation between depression and insomnia (r=0.67, p<.001), when defining depression as a score of fourteen or more on the Beck Depression Inventory (BDI). Their sample had a much greater number, ninety-one subjects, and encompassed a wide spectrum of injury severity, whereas this sample is mostly severe. The fact that an association was found between depression and sleep quality in such diverse samples on two different measures of depression suggests that this finding represents a robust relationship.

The results cannot solely be understood in terms of a depression – mediated association between affective disorder and insomnia. Witol, Kreutzer & Sander (1999), in a review of emotional and behavioural assessment following TBI, acknowledged that the HRSD scale is constituted by: somatic items (forty seven per cent), behavioural factors (twenty-nine percent) and cognitive complaints (twelve per cent). Three quarters of the items may be attributable to neurobehavioural sequelae of TBI rather than depression. This could be viewed as a circular argument, as when we consider TBI and depression it is very difficult to discriminate the differential neurobehavioural and affective symptoms. However, the HAD scale was devised with the hospital population in mind and such factors were controlled for when the measure was designed, therefore, the neurobehavioural sequelae of TBI should considered.

Although a strong relationship was identified between depression and sleep quality following TBI, the regression statistic (R2) suggested that there were additional variables contributing to the insomnia that the researcher failed to consider. It is very difficult to infer what these variables could be; certainly pain and gender have been
identified in past studies. The missing factors may be related to quality of life, physical and mental health, experience of social support networks, life events and loss, or regular involvement in planned daily activities. Further research is required in this area to determine other predictors of sleep quality.

4.1.2 Insomnia and depression in the general population
One of the primary symptoms of depression is disrupted sleep. Insomnia as a symptom of a psychological disorder is ten times more frequent than insomnia related to physical illness (Ford & Kamerow, 1989) and this association between insomnia and psychological disorders raises important questions relating to cause and effect (Harvey, 2001; Katz & McHorney, 1998). Sufferers of depression may have many brief episodes of REM sleep rather than the less frequent and longer episodes experienced in the healthy individual. This repetitive REM may be more exhausting than restful. Most antidepressants reduce REM sleep rather than improving the overall quality of the sleep. Generally people who suffer from depression also experience diurnal mood swings, waking up early with a feeling of ominous dread, feeling worst in the morning and a little better as the day goes on. Wehr (1979) showed that controlled sleep deprivation could have an antidepressant effect. This approach has been advocated as a short-term way of alleviating symptoms and implies that the depressive symptoms may, somehow, have been maintained and intensified during sleep. Similarly, Berger (1997) has advocated and practised “sleep advancement”, in which patients are put to bed at five o’clock in the afternoon and awoken before midnight. This approach has also been shown to have good effects.

During depression the patient rarely enters deep sleep. The quality of the sleep is markedly reduced, because the feelings of being refreshed and well rested are associated with this missing sleep phase. Sleep disturbance often precedes the depressive episode (Solomon, 2001). This runs contrary to the commonly held opinion of Spielman & Glovinsky (1997) that sleep disorders follow periods of psychological distress. However, the idea of sleep disturbance preceding depression would support Harvey’s (2001) suggestion that insomnia should be considered as a diagnosis in its own right, not
solely as a symptom secondary to another condition. If this is the case in the general population, it may also be the case in a TBI population.

4.1.3 Poor Sleep Patterns and Research Findings
Analysis of the semi-structured interview identified categories of behaviour, which related to bad sleep quality, in the eighteen males and three females. Bad sleepers more often reported mental alertness at bedtime; experiencing thoughts constantly running through their mind, which inhibited the initiation of sleep. Sleep was also more likely to be disturbed due to snoring. The insomnia group were more likely to report having experienced poor sleep while in hospital, following recovery from the TBI. In addition the male insomnia group reported a lighter sleep quality since the TBI and were more likely to report that they had no memory of having had any dreams since their injury. These findings support the literature (Espie, 2002; see Appendix F, which shows the factors contributing to good sleep and insomnia within the psychobiological model).

Increased arousal, sleep apnoea and respiratory problems, conditioned association with sleep incompatible activities, reduced REM and deep sleep, have all been associated with the development of insomnia in a normal population. The semi structured interview and qualitative analysis uncovered some interesting patterns, which can readily be compared to difficulties in a non-TBI population.

The most salient problem associated with this group was a sleep initiation difficulty related to cognitive de-arousal. The bad sleepers tended to complain of not being able to empty their mind of a constant stream of thoughts. These are described by Espie (2002) as difficulties with cognitive de-arousal: rehearsing/planning/probлем-solving thoughts in bed, thinking about events the previous or next day, preoccupations with sleep/sleepiness, “stimulant hungry” mind, mind racing, unable to “switch off”.

Nighttime breathing difficulties, most notably snoring were also significant in the bad sleeping patterns of this group. Sleep apnoea has been identified as a problem within the TBI population. Lighter sleep and no memories of dreaming were categories associated with the bad sleep pattern generated by the semi-structure interviews. These reports are
consistent with a decrease of specific sleep rhythm, along with spindles, K complexes and a low level of REM activity.

A larger scale qualitative investigation could explore the changing identity of a person who has sustained a TBI: perhaps a study using discourse analysis (Potter & Wetherell, 1987) could identify further themes and patterns pertaining to an individual’s experience of sleep disturbance and how they talk about it.

4.1.4 Methodological problems
The present study did not find a distinction between the different brain injury severities. It is difficult to explain why this difference was not observed, when it has been such a robust finding in other studies. There were no significant differences found between the sleep quality of the severe and mild / moderate severity groups. This is incongruent with the findings of similar studies, which reported injury severity as a main factor predictive of insomnia, in comparable TBI samples (Beetar et al, 1996; Fichtenberg et al., 2000). That is, they found that injury severity of the milder brain injury group predicted insomnia. Similarly the fatigue groups were not different. This is very interesting because intuitively one might guess that the greater the severity of brain injury the more the fatigue.

The present study sample, all of whom required hospitalisation and rehabilitation following their injury, are an example of a severe TBI sample population. The small sample size no doubt contributed to this non-significant result. The difference would be corrected for if the sample had a broader range of severity. This might be possible by undertaking a multi centre study involving acute and primary care settings, to ensure a broad severity of TBI range. However, a strength of this study is that it has the opportunity to focus on a more severe, long term post injury population. A prospective cohort study assessing, with repeated measures at various time points, the progress of TBI patients would provide a more robust longitudinal picture of changes in sleep quality following TBI. Essentially the subject becomes his or her own control measure
and any improvement or deterioration can be observed, across time. However, such studies also have limitations, such as a high drop out rate due to default rates and mortality and morbidity effects.

It is worth considering the theory, which attempts to explain the findings of other studies, where TBI severity was a significant finding. This suggests that the apparent link between depression and sleep disturbance is masking an underlying organic basis for disturbed sleep. For the mild TBI condition, brain pathology after suffering a TBI such as a diffuse axonal injury (DAI) might cause microscopic damage that was not diagnosed or detected on a CT/MRI scan. More specifically, for the severely brain injured TBI, it might be argued that milder brain injuries are less likely to be marked by agnosia and are therefore more likely to be aware of the impairment and disability. Consequently, milder cases may be more vulnerable to depression and secondarily insomnia (Fichtenberg et al, 2000; Beetar et al 1996). The implication of this is that the capacity to realise and appreciate personal concerns may require the integrity of certain brain functions such as self-awareness (i.e. the absence of an agnosia).

In a future study it would be interesting to further investigate levels of self-awareness. Such a study could compare TBI groups on presence or absence of agnosia and investigate whether the individuals without agnosia suffer from an increased psychological distress, sleep disturbance and daytime fatigue.

Although, in the present study, there were no differences identified between the reports of a significant other and the TBI participant on sleep quality, one of the items that constituted the global score was significant. This item investigated sleep disturbance through the night: “how often do you have trouble sleeping because you…” 1) wake up in the middle of the night or early morning, 2) have to use the bathroom, 3) cannot breathe comfortably, 4) cough or snore loudly, 5) feel too cold, 6) feel too hot, 7) had bad dreams, 8)have pain and 9) other reasons. This significant difference, where the significant other reported higher ratings on this item in comparison with the TBI participant, may be explained by poor self-awareness. Poor self-awareness is a common
finding after TBI (Fleming, Strong & Ashton, 1996; Cicerone, 1996). The causes of this unawareness, or lack of insight, are likely to be complex and multiple. The ability of patients to modify their perceptions and acknowledge deficits following objective feedback may have particular diagnostic and clinical implications in this area.

Such impaired awareness has been hypothesised to limit the patient’s eventual functional outcomes by decreasing motivation for treatment and resulting in the selection of inappropriate long-term goals. The significant other and TBI rating differences were very interesting, but these differences did not reflect on the overall PSQI, which is clearly a more robust measure between observer ratings. However, the difference on item scores may also be explained by the increased opportunity for the observer (significant other) to disturbed by their partner and then to be awake themselves to be then witnessing sleep-disturbed behaviour that the sleeper may not, such as coughing and snoring.

It would be very interesting to look at other measures of affect, general health or quality of life measures with the significant other as an observer. However, there are many measures of self-awareness available. The present study showed that an observer validated the sleep quality measures generated by the TBI participant. This reduces the likelihood that the participant had poor self-awareness. This is, in itself, an interesting result.

4.1.5 Conclusions, Recommendations for Clinical Practice & Future study

The strong relationship between sleep quality and depression, which emerged from this study, is consistent with the research literature pertaining to insomnia within the general population, as well as chronic medical patients. The Ford & Kamerow (1989) epidemiological study revealed that forty per cent of insomniacs suffered from a psychiatric disorder and fifty-six per cent of the psychiatric cases were depressed. According to DSM-IV insomnia is related to another mental disorder in thirty five to
fifty per cent of the people who seek treatment for insomnia at sleep disorder centres. Conversely, insomnia is a common feature of depression and sleep EEG abnormalities may be evident in forty to sixty per cent of outpatients and up to ninety per cent of inpatients suffering from major depression (DSM-IV). Hyppa & Kronholm (1989) investigated the relationship between disordered sleep and a variety of factors with 1325 chronic medical patients. They found that psychosocial factors, particularly depression, were predictive of disturbed sleep, while organic disease by itself did not account for the prevalence of sleep disorders.

Considering the generalized nature of the association between insomnia and depression, the finding that this relationship extends after the post acute TBI stage is not unexpected. It makes intuitive sense based upon existing knowledge about the course of recovery following TBI. During the acute stage, dysregulation of sleep appears to be a function of the diffuse disruption of the cerebral functioning following direct physical damage to the brain and secondary neuropathological events. As brain functioning becomes reorganized and some degree of the neurological stability is re-established, sleep can be expected to normalise in the absence of damage to neural structures (Rosenthal et al, 1999).

Notwithstanding, as a person with a TBI progresses into the later stages of rehabilitation and recovery, psychosocial factors increasingly exert greater control over behaviour. The vulnerability of the brain injured person to social isolation and depression has been well established (Oddy, et al, 1985; Brooks, et al, 1987; Hoofien, 2001). Since depression and insomnia frequently go together the finding that there is a relationship between the two following a TBI is not surprising.

Fichtenberg et al. (2000) suggested that sleep disturbance during acute TBI may often be distinguished from sleep disorder following post acute TBI on the basis of the role of neurological versus psychosocial factors in the determination of the disordered sleep. The results of this study cannot solely be understood in terms of a depression – mediated association between insomnia. An organic basis for the presence of sleep disorder
following TBI should also be discussed. The neuropathology of TBI can be considered as somewhat analogous to depression.

Frieboes, Müller, Murck, von Cramon, Holsboer & Steiger (1999) conducted an intriguing study to test the hypothesis that the changes in brain injured patients several months after injury are similar to those seen in depression. The authors investigated simultaneously the sleep of thirteen young male non-depressed patients after TBI and thirteen age-matched control subjects. Their results showed a pattern of sleep-endocrine changes in the patients after TBI, which had similarities to that of patients with remitted depression. They found a pattern of sleep-EEG (Electro Encephalogram) parameters and nocturnal hormone secretion similar to that seen in patients with remitted depression. They suggest that permanent hypothalamic-pituitary adrenocortical system overdrive and long term modulation of hypothalamic and pituitary receptors may lead to permanent sleep-endocrine alterations. They call this a neurobiological "scar". In addition, they proposed that there might be hypothalamic-pituitary damage due to diffuse thinning of neurons, such as growth hormone-releasing hormone secreting cells. They advocate that further studies into growth hormone peptides may clarify the interactions between sleep and hormone secretion after TBI.

A better understanding of the mechanism of the pathological changes after TBI might help in protecting the patients from subsequent depression. Early augmentation treatment with antidepressive drugs may be useful in the rehabilitation of patients after TBI. This issue is pertinent considering that a high incidence of depression has been reported following TBI (Jorge et al., 1993).

The clinical applications of the study are readily apparent: knowledge of factors associated with the emergence of sleep disorders following a TBI supports the clinical relevance for all treatment providers. Effective long term intervention may require an accurate differential diagnosis. This study makes it clear that cases involving severe brain injury may be at a similar level of risk for the development of insomnia. In addition, depression may actually be secondary to the sleep disorder, rather than sleep
disorder secondary to the depression or adjustment reactions following TBI. It would be useful to carry out comparison studies between depressed and brain injured groups, to identify and further study other similar parallels in the mechanism of the development of depression and brain injury.

It has been proposed that in any treatment plan for mild TBI’s that sleep disorders should usually be treated first for several reasons (Rao & Rollings, 2002), as comparatively significant progress can be achieved quite quickly. In addition to the benefit of having such problems minimised, the individual can actually experience tangible evidence of their improvement. Professionals working in a rehabilitation team should be aware that sleep disorders in TBI could be treated. Treating sleep disorders primarily, is a quick way to reinforce this idea to patients, their family and friends and their support network. It may also help individuals following TBI develop a self-directed sleep maintenance program, which could lead to improved cognitive functioning.

The present study has indicated that treatment for sleep disorder may be as applicable in a long-term severe TBI group as in a mild population. The TBI group appeared to have similar sleep difficulties as normal insomnia samples reported in previous studies; the factors within the psychobiological model of good sleep were identifiable in this predominantly severe injury group. Treatments for sleep disorder should be offered whatever the severity of TBI. Pharmacological treatments in TBI (Rao & Rollings, 2002) and non-pharmacological treatments in other populations have had good outcomes (Rao & Rollings, 2002, Espie, 1999). Future studies could monitor and evaluate the outcome of mixed and non-pharmacological treatments in the severe TBI client group.
References


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Appendix A  Invitation to take part in study

A1 Invitation letter

A2 Patient Information Sheet

A3 Consent form
Dear Postgraduate Doctorate research project: Sleep disorder following traumatic brain injury

I would be grateful if you would consider taking part in this research project. Before you decide it is important for you to understand why the research is being done and what it will involve.

The purpose of this study is to investigate the physical and psychological problems that people may experience related to sleep after a head injury. We are interested in the ways people have managed to cope with any kind of sleep disturbance. We also want to meet people who have had little or no problems with their sleep after recovering from a head injury. Often the long-term costs of suffering from sleep disturbance mean that we are not always fully aware how much impact it has on our behaviour. For this reason we would also like to ask someone who lives with you, or who knows you well, about your sleeping patterns.

I have enclosed a form called Information for participants: this sheet provides you and your family with some background information on the research project. Please read this over carefully as it describes, in more detail, the purpose of the study. Please feel free to contact us if you would like any additional information.

Look forward to hearing from you soon.

Yours faithfully,

[Signature]

Tony Prior
Head of Clinical Neuropsychology

Margaret C. Couston
Postgraduate Doctorate in Clinical Psychology Trainee
Please tick the box below and return this slip in the stamped addressed envelope provided if you would like to talk more about taking part in this project

I would like to talk more about taking part in this project □

Your name (block capitals)

Your address (block capitals)

Your telephone number

Thank you for taking the time to complete this form. A researcher will contact you soon and arrange a convenient time to meet.
You are invited to take part in a research study. Before you decide it is important for you to understand why the research is being done and what will involve. Please take time to read the following information carefully and discuss it with others if you wish. Ask us if there is anything that is not clear or if you would like more information. Take time to decide whether or not you wish to take part. Thank you for reading this.

What is the purpose of the study?
It is unusual for people after a head injury to experience sleep disturbance. Sleep problems can occur even after the initial recovery from a head injury. The purpose of this study is to investigate the physical and psychological problems that people may experience related to sleep after a head injury. We are interested in the ways people have managed to cope with any kind of sleep disturbance. We also want to know whether or not people with little or no problems with sleep after recovering from a head injury.

What has been the long-term costs of suffering from sleep disturbance mean that we are not always fully aware of how much impact it has on our behaviour. For this reason we would also like to ask someone who lives with you, or who knows you well, about your sleeping patterns.

Why have I been chosen?
This study is interested in people who have recovered from an injury to the head, which occurred 1 year or more ago, and we would like to find out about your experience of sleep.

What will happen if I take part?
If you decide you would like to take part in this study, please return the attached form. A researcher will make contact with you and an appointment will be arranged at a place and time suitable for you. The interview will take approximately 1-2 hours. Both you and someone you have identified who knows you well (for example, wife, husband, partner, other family member or friend) will be interviewed. It might be that you would prefer interviews at different times and this can easily be arranged.

What do I have to do?
We would like to ask you some questions about your sleep, both before and after your injury. Also the researcher will ask you and someone close to you (a person who you have identified who knows you well) to complete some questionnaires about your sleep. You will also be asked to complete some questionnaires and measures of how you feel, how tired you are during the day, your attention, concentration and memory.

What are the possible disadvantages or risks of taking part?
There should be no ill effects in taking part in this study; however, some people do experience discomfort when responding to questionnaires about themselves. If you have any questions about the study and wish to discuss this then please do not hesitate to contact us. The interview may take 1-2 hours with each person, so you have to give up some valuable time. We appreciate this and are very grateful that people are willing to donate their valuable time towards helping inform current research, which may benefit others in a similar position to them in the future.

What are the possible benefits of taking part?
We would hope that being able to discuss the way you have coped, even if you have not suffered a sleep disturbance, may help both your own and our understanding. This in turn will provide valuable information, which will go towards informing health care workers. For example by helping to develop future treatments, rehabilitation advice and treatments for people in similar situation as you were. You may also gain some knowledge about your condition and if you wish a summary report of the results, when the study is complete, the researcher would be glad to forward this to you on completion of the study. Your identity will be completely anonymous and you will not be identified in the final report or any future publications.

P.T.O.
Confidentiality

All information, which is collected about you during the course of the research, will be kept strictly confidential. Any information about you, which leaves the hospital, will have your name and address removed so that you cannot be recognised from it.

We would also like to seek your permission to inform your GP that you are taking part in this study. Occasionally information arising from research interviews may lead to the researcher suggesting that you discuss certain issues with your GP, however, no further information will be offered to your GP before it first discussed with you.

Who has reviewed the study?

Approval for this study has been sought from all the consultants concerned, Dr. Brian Pentland, Clinical Director & Consultant Neurologist, and Dr. Stephen Rath, Consultant Neurologist, Charles Bell Pavilion, SBIRS, Astley Ainslie Hospital, Edinburgh. Scottish research ethics committee approval has also been sought and approved.

Contact for further information

You can find out more about the project and get information or advice from someone who is not directly involved by contacting the researcher or the independent adviser.

Please feel free to contact the people below:

The Principal researcher: Margaret C. Couston, Doctorate of Clinical Psychology Trainee, Neuropsychology Department, Astley Ainslie Hospital, EDINBURGH
Tel: 0131 537 9139

Or

If you want information from someone not directly involved in the project, please contact:

The Local Independent Adviser: Dr. Brian Pentland, Clinical Director and Consultant Neurologist, SBIRS, Charles Bell Pavilion, Astley Ainslie Hospital, Edinburgh.
Tel: 0131 537 9394

Thank you very much for taking the time to read this information sheet and for considering taking part in this study. If you decide to take part in this study I would be grateful for your prompt reply.
1. I have read this Consent Form and the Information for Participants sheet and had the opportunity to ask questions about the study.

2. I understand that I do not have to take part in this study and that a decision not to participate will not have any unfavourable consequences for me.

3. I understand that I have at least 24 hours to decide whether to take part in the study or not and I may change my mind and withdraw from the study at any stage.

4. I understand that the discussions in the interviews will be audio-taped and then transcribed for analysis.

5. I understand that the information I give as part of the study will be confidential to the researchers and I will not be identified in any way in the written report of the study.

6. I agree to participate in this study and allow researchers to contact my GP to let them know I am taking part in this study.

Signature of participant ................................................................. date ..................

Signature of researcher ................................................................. date .................
Appendix B: Study measures

B1 Pittsburgh Sleep Quality Index
B2 Hamilton Rating Scale For Depression
B3 Hospital Anxiety and Depression Scale
B4 Visual Analogue Scale-Fatigue
B5 Bentall Fatigue Inventory
B6 National Adult Reading Test
Appendix A

Pittsburgh Sleep Quality Index

The following questions relate to your usual sleep habits during the past month only. Your answers should indicate the most accurate reply for the majority of days and nights in the past month.

Please answer all the questions.

1. During the past month, when have you usually gone to bed at night?
   
   **USUAL BED TIME:** __________________________

2. During the past month, how long (in minutes) has it usually taken you to fall asleep each night?
   
   **NUMBER OF MINUTES:** ______________________

2b. How long have you usually been awake during the night?
   
   **NUMBER OF MINUTES:** ______________________

3. During the past month, when have you usually got up in the morning?
   
   **USUAL GETTING UP TIME:** _________________

4. During the past month, how many hours of actual sleep did you get at night?

   This may be different to the number of hours you spend in bed.

   **HOURS OF SLEEP PER NIGHT:** _______________  

4b. How many nights per week do you usually have difficulties sleeping?
   
   **NUMBER OF NIGHTS PER WEEK:** ____________

5. During the past month, how often have you had trouble sleeping because you:

<table>
<thead>
<tr>
<th>(a) Cannot get to sleep within 30 minutes</th>
<th>Not during the past month</th>
<th>Less than once a week</th>
<th>Once or twice a week</th>
<th>Three or more times a week</th>
</tr>
</thead>
<tbody>
<tr>
<td>(b) Wake up in the middle of the night or early morning</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>(c) Have to get up and use the bathroom</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>(d) Cannot breathe comfortably</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>(e) Cough or snore loudly</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
(f) Feel too cold
(g) Feel too hot
(h) Had bad dreams
(i) Have pain

(j) Other reason(s), please describe

How often during the past month have you had trouble sleeping because of this?

<table>
<thead>
<tr>
<th>Not during the past month</th>
<th>Less than once a week</th>
<th>Once or twice a week</th>
<th>Three or more times a week</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

6. During the past month, how would you rate your sleep quality overall?

<table>
<thead>
<tr>
<th>Very good</th>
<th>Fairly good</th>
<th>Fairly bad</th>
<th>Very Bad</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

7. During the past month, how often have you taken medicine (prescribed or 'over the counter') to help you sleep?

<table>
<thead>
<tr>
<th>Not during the past month</th>
<th>Less than once a week</th>
<th>Once or twice a week</th>
<th>Three or more times a week</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

8. During the past month, how often have you had trouble staying awake while driving, eating meals or engaging in social activity?

<table>
<thead>
<tr>
<th>Not during the past month</th>
<th>Less than once a week</th>
<th>Once or twice a week</th>
<th>Three or more times a week</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

9. During the past month, how much of a problem has it been for you to keep up enough enthusiasm to get things done?

<table>
<thead>
<tr>
<th>No problem at all</th>
<th>Only a very slight problem</th>
<th>Somewhat of a problem</th>
<th>A very big problem</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

10. Do you have a bed partner or room-mate?
<table>
<thead>
<tr>
<th></th>
<th>No bed partner or room-mate</th>
<th>Partner/room-mate in other room</th>
<th>Partner in same room, but not same bed</th>
<th>Partner in same bed</th>
</tr>
</thead>
</table>

If you have a roommate or bed partner, ask him/her how often in the past month you have had:

(a) Loud snoring

Not during the past month

Less than once a week

Once or twice a week

Three or more times a week

(b) Long pauses between breaths while asleep

Not during the past month

Less than once a week

Once or twice a week

Three or more times a week

(c) Legs twitching or jerking while you sleep

Not during the past month

Less than once a week

Once or twice a week

Three or more times a week

(d) Episodes of disorientation or confusion during sleep

Not during the past month

Less than once a week

Once or twice a week

Three or more times a week

(e) Other restless while you sleep; please describe

Not during the past month

Less than once a week

Once or twice a week

Three or more times a week
Hamilton Depression Scale-17 (HAM-D)

Instructions: Circle one number per item. Score all items.

1. Depressed Mood (sad, blue, gloomy, weepy, pessimistic, helpless, hopeless, worthless)
   0 Not depressed
   1 Feeling states elicited only on questioning.
   2 Occasional weeping or spontaneously reports feeling states.
   3 Frequent weeping. Obvious behavioural evidence in facies, posture, voice. Speaks mostly about feeling states.
   4 Exhibits virtually only these feeling states verbally and nonverbally. May have "gone beyond weeping".

2. Guilt feelings and delusions
   0 Absent
   1 Self-reproach, feels he/she has let people down.
   2 Expresses guilt over past errors.
   3 Present illness is deserved punishment. Ruminations over past errors and sins.
   4 Severe self-reproach. Guilty delusions, e.g., is making other people ill. Deserves to die. May have accusatory or denouncing auditory or visual hallucinations.

3. Suicide
   0 Absent.
   1 Feels life is empty, not worth living.
   2 Recurrent thoughts or wishes about death of self.
   3 Active suicidal thoughts, threats, gestures.
   4 Serious suicide attempt

4. Initial Insomnia (as part of present illness)
   0 Absent
   1 Mild, infrequent, less than 1/2 hour
   2 Obvious and severe, more than 1/2 hour usually

5. Middle Insomnia
   0 Absent (Rate 1 if hypnotic is being used)
   1 Complains of feeling restless and disturbed during night.
   2 Wakes during the night; any reading or smoking in bed or getting out of bed except to void.

6. Delayed Insomnia
   0 Absent.
   1 Wakes earlier than usual
   2 Wakes 1-3 hours before usual, unable to sleep again

7. Work and Activities (Apathy: loss of interest in work, hobbies, social life. Anhedonia: unable to feel pleasure)
   0 No disturbance.
   1 Feels incapable, listless, less efficient. (Rate fatigue, loss of energy under item 13)
   2 Has to push self to work or play. No active interests, gets little satisfaction, feels listless, indecisive.
   3 Clearly decreased efficiency. Spends less time at usual work.
   4 Stopped work because of present illness. Doesn't shave, bathe, etc. Works only with urging
8. Retardation (psychomotor slowing of thought, speech, and movement)
   0 Absent
   1 Slight flattened affect, fixed facial expression.
   2 Monotonous voice, delayed answering, sits motionless.
   3 Interview difficult and prolonged. Moves slowly.
   4 Depressive stupor. Interview impossible.

9. Agitation (may co-exist mildly with retardation).
   0 None
   1 Fidgety
   2 Playing with hands or hair, picking at hands or clothes.
   3 Moving about, can't sit still.
   4 Hand wringing, nail biting, hair pulling, biting of lips.

10. Psychic Anxiety (as part of present illness, NOT part of previous disposition. Includes feeling tense, irritable, apprehensive, fearful, phobic or panic attack).
    0 Absent.
    1 Minimal distress, admitted only on direct questioning.
    2 Spontaneously expresses discomfort; worries over trivia.
    3 Obviously apprehensive in face of speech.
    4 Severely anxious, panicky

11. Somatic Anxiety (physiological concomitants of anxiety such as; fainting, tinnitus, blurred vision, headache, tremor, sweating, flushing, hyperventilation, palpitations, indigestion, belching, diarrhoea, urinary frequency)
    0 Absent.
    1 Mild.
    2 Moderate.
    3 Severe.
    4 Incapacitating.

12. Somatic Symptoms Gastrointestinal.
    0 Normal appetite.
    1 Eats spontaneously but without relish.
    2 Marked reduction of appetite and food intake. Eats only with urging. Requests or requires laxatives.

13. Somatic Energy
    0 Normal.
    1 Occasional mild fatigue, easy tiring, aching.
    2 Obviously low in energy, tired all the time; frequent backaches, headaches, heavy feelings in limbs.

14. Genital Symptoms (Rate change in libido, impotence, menstrual disturbances.)
    0 Absent.
    1 Mild.
    2 Severe.

15. Hypochondriasis.
    0 Absent
    1 Self-absorbed about bodily functions and physical symptoms
    2 Preoccupied with health.
    3 Frequent complaints, requests for help, etc.
    4 Muddled convictions of organic disease, e.g., brain tumour, cancer, or delusions e.g., worms eating head, rotting inside, bowels blocked, terrible odour
16. Weight Loss (When rated by history, according to subject)

0  No weight loss
1  Probable weight loss.
2  Definite weight loss.

17. Loss of Insight

0  Acknowledges being depressed.
1  Acknowledges illness but attributes cause : bad food, climate, overwork, virus, need for rest, etc.
2  Denies being ill at all.
Read each item and place a tick in the box opposite the reply which comes closest to how you have been feeling in the past week.

Don't take too long over your replies: your immediate reaction to each item will probably be more accurate than a long thought-out response.

<table>
<thead>
<tr>
<th>Item</th>
<th>Options</th>
</tr>
</thead>
<tbody>
<tr>
<td>I feel tense or 'wound up':</td>
<td>Most of the time, A lot of the time, Time to time, Occasionally, Not at all</td>
</tr>
<tr>
<td>I still enjoy the things I used to enjoy:</td>
<td>Definitely as much, Not quite so much, Only a little, Hardly at all</td>
</tr>
<tr>
<td>I get a sort of frightened feeling as if something awful is about to happen:</td>
<td>Very definitely and quite badly, Yes, but not too badly, A little, but it doesn't worry me, Not at all</td>
</tr>
<tr>
<td>I can laugh and see the funny side of things:</td>
<td>As much as always could, Not quite so much now, Definitely not so much now, Not at all</td>
</tr>
<tr>
<td>Worrying thoughts go through my mind:</td>
<td>A great deal of the time, A lot of the time, From time to time but not too often, Only occasionally</td>
</tr>
<tr>
<td>I feel cheerful:</td>
<td>Not at all, Not often, Sometimes, Most of the time</td>
</tr>
<tr>
<td>I can sit at ease and feel relaxed:</td>
<td>Definitely, Usually, Not often, Not at all</td>
</tr>
<tr>
<td>I feel as if I am slowed down:</td>
<td>Nearly all the time, Very often, Sometimes, Not at all</td>
</tr>
<tr>
<td>I get a sort of frightened feeling like 'butterflies' in the stomach:</td>
<td>Not at all, Occasionally, Quite often, Very often</td>
</tr>
<tr>
<td>I have lost interest in my appearance:</td>
<td>Definitely, I don't take so much care as I should, I may not take quite as much care, I take just as much care as ever</td>
</tr>
<tr>
<td>I feel restless as if I have to be on the move:</td>
<td>Very much indeed, Quite a lot, Not very much, Not at all</td>
</tr>
<tr>
<td>I look forward with enjoyment to things:</td>
<td>As much as ever I did, Rather less than I used to, Definitely less than I used to, Hardly at all</td>
</tr>
<tr>
<td>I get sudden feelings of panic:</td>
<td>Very often indeed, Quite often, Not very often, Not at all</td>
</tr>
<tr>
<td>I can enjoy a good book or radio or TV programme:</td>
<td>Often, Sometimes, Not often, Very seldom</td>
</tr>
</tbody>
</table>
I am trying to find out about your level of energy before and after your night of sleep. There are 18 items I would like you to respond to. This should take less than 1 minute of your time. Thank you.

DIRECTIONS: You are asked to circle a number on each of the following lines to indicate how you are feeling RIGHT NOW.

For example, suppose you have not eaten since yesterday. What number would you circle below?

<table>
<thead>
<tr>
<th>not at all</th>
<th>extremely hungry</th>
</tr>
</thead>
<tbody>
<tr>
<td>hungry</td>
<td>0 1 2 3 4 5 6 7 8 9 10</td>
</tr>
</tbody>
</table>

You would probably circle a number closer to the "extremely hungry" end of the line. This is where I put it:

<table>
<thead>
<tr>
<th>not at all</th>
<th>extremely hungry</th>
</tr>
</thead>
<tbody>
<tr>
<td>hungry</td>
<td>0 1 2 3 4 5 6 7 8 9 10</td>
</tr>
</tbody>
</table>

NOW PLEASE COMPLETE THE FOLLOWING ITEMS:

1. not at all tired 0 1 2 3 4 5 6 7 8 9 10 extremely tired
2. not at all sleepy 0 1 2 3 4 5 6 7 8 9 10 extremely sleepy
3. not at all drowsy 0 1 2 3 4 5 6 7 8 9 10 extremely drowsy
4. not at all fatigued 0 1 2 3 4 5 6 7 8 9 10 extremely fatigued
5. not at all worn out 0 1 2 3 4 5 6 7 8 9 10 extremely worn out
6. not at all energetic 0 1 2 3 4 5 6 7 8 9 10 extremely energetic
7. not at all active 0 1 2 3 4 5 6 7 8 9 10 extremely active
8. not at all vigorous 0 1 2 3 4 5 6 7 8 9 10 extremely vigorous
9. not at all efficient 0 1 2 3 4 5 6 7 8 9 10 extremely efficient

10. not at all lively 0 1 2 3 4 5 6 7 8 9 10 extremely lively

11. not at all bushed 0 1 2 3 4 5 6 7 8 9 10 totally bushed

12. not at all exhausted 0 1 2 3 4 5 6 7 8 9 10 totally exhausted

13. keeping my eyes open is no effort at all 0 1 2 3 4 5 6 7 8 9 10 keeping my eyes open is a tremendous chore

14. moving my body is no effort at all 0 1 2 3 4 5 6 7 8 9 10 moving my body is a tremendous chore

15. concentrating is no effort at all 0 1 2 3 4 5 6 7 8 9 10 concentrating is a tremendous chore

16. carrying on a conversation is no effort at all 0 1 2 3 4 5 6 7 8 9 10 carrying on a conversation is a tremendous chore

17. I have absolutely no desire to close my eyes 0 1 2 3 4 5 6 7 8 9 10 I have a tremendous desire to close my eyes

18. I have absolutely no desire to lie down 0 1 2 3 4 5 6 7 8 9 10 I have a tremendous desire to lie down
IN THE LAST MONTH HOW MUCH HAVE YOU BEEN BOTHERED BY....
(Please circle the response that best applies to you)

<table>
<thead>
<tr>
<th>Spell Of Confusion</th>
<th>not at all</th>
<th>a little</th>
<th>somewhat</th>
<th>quite a lot</th>
<th>very much</th>
</tr>
</thead>
<tbody>
<tr>
<td>Thoughts Getting Mixed Up</td>
<td>not at all</td>
<td>a little</td>
<td>somewhat</td>
<td>quite a lot</td>
<td>very much</td>
</tr>
<tr>
<td>Poor Concentration</td>
<td>not at all</td>
<td>a little</td>
<td>somewhat</td>
<td>quite a lot</td>
<td>very much</td>
</tr>
<tr>
<td>Can't Easily Make Decisions</td>
<td>not at all</td>
<td>a little</td>
<td>somewhat</td>
<td>quite a lot</td>
<td>very much</td>
</tr>
<tr>
<td>Poor Memory For Recent Events</td>
<td>not at all</td>
<td>a little</td>
<td>somewhat</td>
<td>quite a lot</td>
<td>very much</td>
</tr>
<tr>
<td>Can't Take Things In When Speaking To People</td>
<td>not at all</td>
<td>a little</td>
<td>somewhat</td>
<td>quite a lot</td>
<td>very much</td>
</tr>
<tr>
<td>Thoughts Are Slow</td>
<td>not at all</td>
<td>a little</td>
<td>somewhat</td>
<td>quite a lot</td>
<td>very much</td>
</tr>
<tr>
<td>Muzzy Head</td>
<td>not at all</td>
<td>a little</td>
<td>somewhat</td>
<td>quite a lot</td>
<td>very much</td>
</tr>
<tr>
<td>Can't Find The Right Words</td>
<td>not at all</td>
<td>a little</td>
<td>somewhat</td>
<td>quite a lot</td>
<td>very much</td>
</tr>
</tbody>
</table>

**Pronunciation guide**

<table>
<thead>
<tr>
<th>Word</th>
<th>Pronunciation</th>
<th>Pronunciation</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>CHORD</strong></td>
<td>kör'd</td>
<td><strong>SUPERFLIOUS</strong></td>
</tr>
<tr>
<td><strong>ACHE</strong></td>
<td>āk</td>
<td><strong>SIMILE</strong></td>
</tr>
<tr>
<td><strong>DEPOT</strong></td>
<td>dep'ō</td>
<td><strong>BANAL</strong></td>
</tr>
<tr>
<td><strong>AISLE</strong></td>
<td>īl</td>
<td><strong>OUAQRUPED</strong></td>
</tr>
<tr>
<td><strong>BOUQUET</strong></td>
<td>bōōk'ā, bōōkā', bōkā'</td>
<td><strong>CELLIST</strong></td>
</tr>
<tr>
<td><strong>PSALM</strong></td>
<td>sām</td>
<td><strong>FACADE</strong></td>
</tr>
<tr>
<td><strong>CAPON</strong></td>
<td>kā'pn</td>
<td><strong>ZEALOT</strong></td>
</tr>
<tr>
<td><strong>DENY</strong></td>
<td>dī-nī</td>
<td><strong>DRACHM</strong></td>
</tr>
<tr>
<td><strong>NAUSEA</strong></td>
<td>nō'si-a, nō'žha</td>
<td><strong>AEON</strong></td>
</tr>
<tr>
<td><strong>DEBT</strong></td>
<td>det</td>
<td><strong>PLACEBO</strong></td>
</tr>
<tr>
<td><strong>COURTEOUS</strong></td>
<td>kūrt'yas</td>
<td><strong>ABSTEMIOUS</strong></td>
</tr>
<tr>
<td><strong>RAREFY</strong></td>
<td>rā'r'-i-fi</td>
<td><strong>DETENTE</strong></td>
</tr>
<tr>
<td><strong>EQUIVOCAL</strong></td>
<td>i-kwiv'a-kı</td>
<td><strong>IDYLL</strong></td>
</tr>
<tr>
<td><strong>NAIVE</strong></td>
<td>nā-ev</td>
<td><strong>PUERPERAL</strong></td>
</tr>
<tr>
<td><strong>CATACOMB</strong></td>
<td>kat'a-kōōm</td>
<td><strong>AVER</strong></td>
</tr>
<tr>
<td><strong>GAOLED</strong></td>
<td>jāld</td>
<td><strong>GAUCHE</strong></td>
</tr>
<tr>
<td><strong>THYME</strong></td>
<td>tım</td>
<td><strong>TOPIARY</strong></td>
</tr>
<tr>
<td><strong>HEIR</strong></td>
<td>ār</td>
<td><strong>LEVIATHAN</strong></td>
</tr>
<tr>
<td><strong>RADIX</strong></td>
<td>rā'diks</td>
<td><strong>BEATIFY</strong></td>
</tr>
<tr>
<td><strong>ASSIGNGATE</strong></td>
<td>as'-ig-nät</td>
<td><strong>PRELATE</strong></td>
</tr>
<tr>
<td><strong>HIATUS</strong></td>
<td>hī'-ā'tas</td>
<td><strong>SIDERELAL</strong></td>
</tr>
<tr>
<td><strong>SUBTLE</strong></td>
<td>sut'</td>
<td><strong>DEMESNE</strong></td>
</tr>
<tr>
<td><strong>PROCREATE</strong></td>
<td>prō'kri-āt</td>
<td><strong>SYNCOPE</strong></td>
</tr>
<tr>
<td><strong>GIST</strong></td>
<td>jıst</td>
<td><strong>LABILE</strong></td>
</tr>
<tr>
<td><strong>GOUCE</strong></td>
<td>gowj</td>
<td><strong>CAMPANILE</strong></td>
</tr>
</tbody>
</table>
Appendix C: Semi-structured interview
## Interview schedule

### A. Data collection and measures

<table>
<thead>
<tr>
<th>Measure</th>
<th>Participant</th>
<th>Significant other</th>
</tr>
</thead>
<tbody>
<tr>
<td>Glasgow Coma Scale (GCS)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Post traumatic amnesia (PTA)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>CT/MRI reports</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Nature of injury</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Current medication</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Pittsburgh Sleep Quality Inventory (PSQI)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Hamilton Rating Scale</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Hospital Anxiety and Depression Scale (HADS)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Bentall fatigue inventory</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Visual analogue scale for fatigue (VAS-F)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>National Adult Reading Test (NART)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Postcode</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Educational attainment</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mini Mental State Exam (MMSE)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Clock Drawing Test (CDT)</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

### Folstein's Mini-Mental State Examination

<table>
<thead>
<tr>
<th>Max Score</th>
<th><strong>Orientation</strong>: what is the year, season, numerical date, day of the week, month?</th>
</tr>
</thead>
<tbody>
<tr>
<td>5</td>
<td><strong>Where are we now</strong>: region, country, town, place, floor?</td>
</tr>
<tr>
<td>5</td>
<td><strong>Registration</strong>: Name 3 objects (ball, flag, door) patient repeats.</td>
</tr>
<tr>
<td>5</td>
<td><strong>Attn/Calculation</strong>: Spell <em>world</em> backwards OR count backwards from 100 by 7s (stop after 93, 86, 79, 72, 65)</td>
</tr>
<tr>
<td>3</td>
<td><strong>Recall</strong>: ask them to remember the 3 objects from above</td>
</tr>
<tr>
<td>2</td>
<td><strong>Language</strong>: name pencil/watch</td>
</tr>
<tr>
<td>1</td>
<td><strong>Repeat</strong>: “No ifs ands or buts”</td>
</tr>
<tr>
<td>3</td>
<td><strong>Follow 3 step command</strong></td>
</tr>
<tr>
<td>1</td>
<td><strong>Read and obey</strong>: “close your eyes”</td>
</tr>
<tr>
<td>1</td>
<td><strong>Write a sentence</strong></td>
</tr>
<tr>
<td>1</td>
<td><strong>Copy interlocking pentagons</strong></td>
</tr>
</tbody>
</table>

Total score: ___/30

Hearing impaired? Y N
Vision impaired? Y N
Highest educational level
B. Semi structured interview:

1. In your own words describe a typical nights sleep, before your injury:

2. In your own words describe a typical nights sleep now:

3. What do you do when you have trouble sleeping? (if at all)
4. Lifestyle
How many:

Coffee (cups of) per day-----
Tea (cups of) per day-----
Fizzy juice (cups of e.g. Coca cola / Irn Bru?) per day-----
Cigarettes per day-----
Alcoholic drinks (units) per day-----

5. What medications are you on? (including: prescribed, over the counter and social/recreational drugs)

6. Pain? Do you currently suffer from pain?  Yes----- No-----
If yes, please describe the type of pain you suffer from on a regular basis?

Pain (cond)
On a scale of 1 to 10, where 1 is minimal and 10 is excruciating, how would you rate your pain?
Through the day (1-10) -----
Through the night? (1-10) ----
Appendix D Ethics committee communication and certificate of ethical review

Appendix D1: Correspondence from ethics committee following first proposal submission

Appendix D2: Amendment report form

Appendix D3: Correspondence following amendments to initial proposal

Appendix D4: Correspondence following amendments to final proposal

Appendix D5: Correspondence and certificate of ethical review
Dear Miss Coulston,

AN INVESTIGATION OF THE PREDICTORS OF SLEEP DISORDER, DAYTIME FATIGUE, SELF REPORT AND SIGNIFICANT OTHER RATINGS OF SLEEP DISORDER, 12 MONTHS OR MORE, FOLLOWING TRAUMATIC BRAIN INJURY

Thank you for submitting the above protocol for ethical approval. The Psychiatry/Clinical Psychology Research Ethics Sub-Committee has discussed this protocol at its recent meeting and agreed that the Chairman, Dr Alison Richardson, would discuss the following issues directly with you:-

- Clarification was needed as to whether the researcher was part of the clinical team. The first approach to patients regarding research must be made by the healthcare professional or a member of the clinical team responsible for their care.
- Clarification was needed as to whether the patients were still attending the clinic or whether they had been discharged. If they had been discharged this would have implications for recruitment.
- The grammar in the information sheet needs to be tidied up, eg the last sentence under 'What will happen...'.
- Please delete 'definitely' from the statement in the information sheet '....and will definitely aid our understanding.'
- It was felt that the use of the same phrase twice in the visual analogue scale (p2) was confusing.

cont/...
Short Title of Study: Sleep Disorder Following Traumatic Brain Injury

Protocol No.: LREC//2001/7/34

Drug company protocol no. (if applicable):

Amendment Number and Date: Amendment 1 (February 2002)

Principal Investigator: Miss Margaret Couston, Postgraduate Clinical Psychology Trainee

Progress of the Study: -  

1. Is the Amendment considered purely administrative?  
   Yes ☑ No ☐

2. Has the Patient Information Sheet/Consent Form been altered as a result?  
   (If so, please enclose a copy)  
   Yes ☑ No ☐

3. Is this part of a Multi-Centre Study?  
   ☐ ☑

4. Does it have approval from a Multi-Centre Research Ethics Committee?  
   (If yes, please state which MREC and give MREC reference number)  
   ☐ ☑

5. Please give us any further information you feel is necessary.

The protocol has been changed inline with the requested changes of the committee.

1. See protocol, procedure, step 2: GP's will be contacted for all the patients who have not attended the outpatient department at the SBI RS, Astley Ainslie. The GP's will be asked if they know of any reason why the patient should not take part in a study, the patient’s status and current address will be confirmed. Only after this will the patient be contacted by letter and invited to take part in the study.

2. The invitation letter will be signed by the head of the Neuropsychology Department and will invite patients to make contact if they are interested in taking part.

3. The patient information sheet (attached) has been rewritten and proofread (for readability) by health care professionals.

In addition to the changes requested by the committee 1 further amendment to the protocol has been made:

1. To make sure depression is measured sensitively and accurately the whole of the Hamilton-D measure will be administered (not partially as described earlier).

Signature of Principal Researcher: M. C. Couston

Date of Report 18/2/2002
Dear Miss Couston

LREC/2001/7/34 - AN INVESTIGATION OF THE PREDICTORS OF SLEEP DISORDER, DAYTIME FATIGUE, SELF-REPORT AND SIGNIFICANT OTHER RATINGS OF SLEEP DISORDER, 12 MONTHS OR MORE, FOLLOWING TRAUMATIC BRAIN INJURY

Following the discussion of your protocol by the Psychiatry/Clinical Psychology Research Ethics Sub-Committee at its meeting on 19 December 2001 and your telephone discussion with the Chairman of the sub-committee, the Psychiatry/Clinical Psychology Research Ethics Sub-Committee gave further consideration to your study at its meeting on 16 January 2002. The Psychiatry/Clinical Psychology Research Ethics Sub-Committee has agreed that it is prepared to grant ethical approval subject to the following amendment and the amendments detailed in the letter dated 4 January 2002 delegating authority to the Chairman to approve them on receipt:

• Please approach the patients who have not been in touch with the clinic for some time through their GP, who will know whether the patient has moved or is not deceased.

Once this amendment and those previously stated have been received by me and approved by the Chairman a formal Certificate of Approval will be issued. Only then can management approval be given and the research proceed.

The next meeting of the Sub-Committee will be held on 20 February 2002. It would be appreciated if the required amendments could be available prior to that date.
Dear Miss Coulston,

AN INVESTIGATION OF THE PREDICTORS OF SLEEP DISORDER, DAYTIME FATIGUE, SELF REPORT AND SIGNIFICANT OTHER RATINGS OF SLEEP DISORDER, 12 MONTHS OR MORE, FOLLOWING TRAUMATIC BRAIN INJURY

Thank you for submitting the above protocol for ethical approval. The Psychiatry/Clinical Psychology Research Ethics Sub-Committee has discussed this protocol and has agreed that it is prepared to grant ethical approval subject to the following amendments, delegating authority to the Chairman to approve them on receipt:

- Patient information sheet, paragraph ‘Contact for further information’ should be amended to make clear that while the researcher is able to provide information on the study, information from someone not directly involved with the research can only be obtained from the independent advisor.

Once these amendments have been received by me and approved by the Chairman a formal Certificate of Approval will be issued. Only then can management approval be given and the research proceed.

The next meeting of the Sub-Committee will be held on 20 March 2002. It would be appreciated if the required amendments could be available prior to that date.

Chair

[Signature]

Chairman

[Signatures]

[Institutional Logos]
Dear Miss Couston,

AN INVESTIGATION OF THE PREDICTORS OF SLEEP DISORDER, DAYTIME FATIGUE, SELF REPORT AND SIGNIFICANT OTHER RATINGS OF SLEEP DISORDER, 12 MONTHS OR MORE, FOLLOWING TRAUMATIC BRAIN INJURY

Thank you for submitting the amendments or additional information requested by the Sub-Committee for the above protocol. The Chairman of the Psychiatry/Clinical Psychology Research Ethics Sub-Committee has now agreed to confirm the Sub-Committee’s ethical approval under its delegated authority. An official Certificate of Ethical Review is enclosed together with a list of members present at the meeting.

Under the terms of the Scottish Office Home and Health Department Guidelines on Local Research Ethics Committees this decision has been notified to the NHS body under the auspices of which the research is intended to take place. It is that NHS body which has the responsibility of deciding whether or not the research should go ahead taking account of the advice of the Research Ethics Sub-Committee and from whom you must obtain management approval before any work on the study can proceed.

Details of the Lothian Research Ethics Committee and its documentation can be found on http://www.nhslothian.scot.nhs.uk/nhs_lothian/about_lothian_health/lrec/index.html

Yours sincerely

ANNETTE HARRIS
Committee Administrator
LOTHIAN RESEARCH ETHICS COMMITTEE
CERTIFICATE OF ETHICAL REVIEW

LREC Reference Number: LREC/2001/7/34
Title: An investigation of the predictors of sleep disorder, daytime fatigue, self report and significant other ratings of sleep disorder, 12 months or more, following traumatic brain injury
Researcher: Miss Margaret Couston

The Psychiatry/Clinical Psychology Research Ethics Sub-Committee reviewed this proposed study and has agreed that it is ethical and appropriate to be carried out in the Lothian Area. This opinion encompasses all aspects of the application including the Patient/Subject Information Sheet and all other accompanying documentation provided.

The LREC application form, protocol, subject information sheet, information on compensation arrangements, payments to researchers and the provision of expenses to subjects (where appropriate) were reviewed and approved.

The membership of the Psychiatry/Clinical Psychology Research Ethics Sub-Committee is shown on the attached sheet.

It is a condition of this opinion that you must obtain appropriate management approval from the relevant NHS body under the auspices of which the research is intended to take place before starting the study. It is that NHS body which has the responsibility of deciding whether or not the research should go ahead taking account of the advice of the Local Research Ethics Committee. It is also a condition that you are required to notify the Psychiatry/Clinical Psychology Research Ethics Sub-Committee and the relevant NHS body, in advance, of any significant proposed deviation from the original protocol or application form. Reports to the Sub-Committee and the relevant NHS body are also required once the research is underway if there are any unusual or unexpected results which raise questions about the safety of the research.

Researchers are also required to report on success, or difficulties, in recruiting subjects in order to provide useful feedback on perceptions of the project among patients and volunteers.

Peter Reith  Annette Harris
Secretary Administrator
Lothian Research Ethics Committee Psychiatry/Clinical Psychology

06 March 2002  Research Ethics Sub-Committee

The Psychiatry/Clinical Psychology Research Ethics Sub-Committee is fully compliant with the International Committee on Harmonisation/Good Clinical Practice (ICH) Guidelines for the Conduct of Trials Involving the Participation of Human Subjects as they relate to the responsibilities, composition, function, operations and records of an Independent Ethics Committee/Independent Review Board. To this end it undertakes to adhere as far as is consistent with its Standing Orders, to the relevant clauses of the ICH Harmonised Tripartite Guideline for Good Clinical Practice, adopted by the Commission of the European Union on 17 January 1997. The Membership List, Standing Orders and Statement of Compliance were included on the computer disk containing the guidelines and application form and are available on request.
Appendix E

Interview Transcripts
MC: In your own words describe a typical nights sleep before the injury.
MS01: I would go to bed anytime between 9 and 11, at night. Usually take a pint of water to drink and Eh...leave it at my bedside. Probably went to sleep quickly. Had a few. A couple of dreams...and then woke up...at 5 to...10 to...8. Usually my mum woke me...shouting me to get a move on.....or the alarm....I pressed the snooze button sometimes(laughs).

MC: In your own words describe a typical nights sleep now.
MS01: Haven’t got one. (Laughs)... Can’t usually get over to sleep ‘till about 5 or 6 in the morning. Then I sleep till whenever I waken..... its always a hassle to get up.... can sometimes sleep 12-14 hours...like last weekend didn’t sleep at all the night before (Friday) then slept from 5AM on Sunday morning all the way till Monday night at 5 O’clock.... you know I slept right through...I didn’t even hear the alarm...or the door bell. Or the phone...this is fairly typical ‘cos then I dinnae get to sleep the next night and it all seems to start again....

MC: What do you do when you have trouble sleeping?
MS01: I get a nap through the day...and I usually lie and watch TV in my bed...I’ll sometimes get up and have some juice...or something to eat.... I tend to try to catch up on the sleep somehow...
MC: In your own words describe a typical nights sleep before the injury.

RW02: Hmmm..Peaceful, quite deep and uninterrupted...Cosy.

MC: In your own words describe a typical nights sleep now.

RW02: Well. Quite different... Erratic, hot, cold, tossing and turning. I tend to worry a lot at night, I worry if I can’t sleep. Or about other things...hmm.... then I can’t get over.

MC: What do you do when you have trouble sleeping?

RW02: Wander around my flat...check out email on the Internet...listen to music. I tend to get up and leave the bedroom.
MC: In your own words describe a typical nights sleep before the injury.

JP03: Hmm well I'd usually-a creature of habit you see-we would watch the telly for a couple of hours and then after the news-I mean between 11 an 11:30-I was always in my bed and we used to go to sleep very quickly.

MC: And could you describe a typical nights sleep now.

JP03: Ehmm, Yeh it's improved a lot since I took these herbal tablets [for prostate trouble] Ehmm the length seems to be longer.. Maybe 4-5 hours before I waken up. Cos' before it was shorter, generally woke up and had to go to the toilet. The sleep quality seems to be better.

MC: What do you do when you have trouble sleeping?

JP03: Well I've got some books at the side of my bed. But..Eh..I don't really need to pick up a book...sometimes within usually half an hour I can feel myself drowsy and I put the book down and then that's it...
MC: In your own words describe a typical nights sleep before the injury.

ES04: Yes...I might read...a novel or something before I went to sleep and usually I went to sleep reading. Sometimes I would wake up with the book on the floor and the light on. I had gone to sleep reading (laughs). Usually I would set the alarm to get up at a certain time and I would be woken up by the alarm. I had dreams but nothing frightening.. Normally about something that had been on my mind.

MC: In your own words describe a typical nights sleep now.

ES04: Well I don’t go to sleep till... Go to bed till about twelve now and when I go to bed I still read and I find that I read till I do feel tired.. Then I put the light off and settle down.. So I can sleep for a couple of hours and I am really tired after reading sometimes I can wake up and only have slept about half an hour...and then I’m not long in bed and I have to get up to the toilet..Which I never did before. Ehmm. I find that I can be wakened through the night. Not really. It’s not. You think it’s a dream but its things that have happened and things that you are thinking about that are quite real and then you realise that you aren’t sleeping you have been thinking or dreaming. You realise you have not been sleeping. Then if I do have to get up to the toilet...then I can never go back to sleep. That’s me wakened for a good two or three hours afterwards. Its not that I want to. You know its not that I feel so awake I want to get up and do something. I don’t want to get up I want to go to sleep...and so I just lie and try and sleep..Ehmm.. And then especially if something has happened and it is going through your mind. Just like a silly little thing...silly things that wouldn’t have kept me awake before. Or whatever you think
about it...then you try not to think about it. But you just can’t sleep then eventually...I suppose...say I have to get up to go to college... normally I would set the alarm. Say. I have to get up at seven o’clock. I’ll set the alarm because...if I’m not sleeping so well might just go to sleep when I should be getting up. So I set the alarm just in case I sleep in. but its not because...oh goodness me she slept in...And still ‘cannae get up...its because I’ve not slept and I set the alarm just in case. This may go on for night after night. Through the day is the same, I’ve been told by the physio to rest through the day. To pace myself. I just rest and relax. I don’t sleep.

MC: What do you do when you have trouble sleeping?

ES04: I just stay in my bed because people have said get up and do things. But what am I going to get up and do in the middle of the night...sometimes I have a read. But I find. If I am wakened because I am thinking of things I find it difficult to read.. Because I can’t concentrate on what I am reading because of what’s on my mind... You know if I’ve got something on my mind and I go to bed I find it difficult to concentrate...because I can’t just shut it off...because once I’ve read it I can’t remember what I’ve read...
MC: In your own words describe a typical nights sleep before the injury.
JA05: I would go to bed.... go to sleep..And then the next thing I would know the alarm clock would be ringing. Saying get up! (laughs)

MC: In your own words describe a typical nights sleep now.
JA05: It's just about the same
MC: In what way?
JA05: I don’t need to get up when the alarm clock goes off...now we have a son who lives with us who is disabled...is a Para Olympic swimmer.... I coach him.... But the wife gets up to him in the morning

MC: What do you do when you have trouble sleeping?
JA05: I never have any problems or trouble sleeping
MC: In your own words describe a typical nights sleep before the injury.

PB06: I’d make sure my stuffs ready for work. Then I’d go to bed sleep for a while then I’d get up for cornflakes (what time?) about 3 O’clock- 4 O’clock in the morning.. I’d get up. Then I’d go back to bed straight away after that...no trouble.... then that would be me..Next thing it would be morning. Get up about 7. My dad or mum would shout me. I kind’a ignore the alarm! (laughs). Sometimes I would push it and it would be half 7 and I’d have ‘tae burst my trainers! (Laughs)

MC: In your own words describe a typical nights sleep now.

PB06: Roughly the same....eh. I don’t get to sleep quite as quickly really...I still get up through the night to eat my cornflakes or go to the toilet...at about 3-4 o’clock.... takes about 20 minutes to get over. Have to roll about a bit now to get comfortable...I snore now...so my Da says...but it ‘dissnae bother me.... I think its something to do with the medicine....

MC: What do you do when you have trouble sleeping?

PB06: Roll about the bed ‘tae to get comfortable...too hot or too cold...I take the covers off.... usually... that’s the thing that keeps me awake... or that I am in an uncomfortable position.
MC: In your own words describe a typical nights sleep before the injury.

JM07: Just sortae normal. I feel maybe get over quicker to sleep if maybe I’d had a drink and things like that. You know ’cos I’ve never been a tea toaller!(laughs). I’d get up at 6 or seven…my sons girl has got a part time job in Boots. And I’d say I’ll come and pick you up and on a Saturday she’s got to be there for 6 o’clock…I get up and take her down no problems.

MC: In your own words describe a typical nights sleep now.

JM07: There’s no change it’s just the same…as I say I could drive from Edinburgh down to Swindon and I’d be driving all that way down…only time I waken up is if I need the toilet

MC: What do you do when you have trouble sleeping?

JM07: Don’t know. Never had any problems with sleep.
MC: In your own words describe a typical nights sleep before the injury.

BR08: As I said there would be variations. 2 main themes. I would be with the nature of the job we had to do a lot of socialising after socialising I would have trouble with sleep and that was the alcohol. I generally found that after say 3 or 4 nights of socialising I would have great trouble sleeping. I’d cut out the alcohol for several nights and catch up on the sleep. So it was really to do with lifestyle or “jobstyle” and the other pattern would be I have a backache type problem. So getting a decent mattress you could sleep. I have slight aches and pains nothing dramatic but enough to bother you so you could only sleep from twelve thirty to 4 O’clock and I would generally wake early. You would miss out on the last bit of the sleep the alarm awoke me other times quite often I would wake up before it as I was doing a lot of travelling my sleep pattern was all over the place my body clock meant that I would always tend to wake up about ½ an hour an hour before or an hour before the alarm was due to go of. What I would do then was power nap on the planes. Get up at 4-3 after 3-4 hours sleep get an hour or 45 minutes sleep on the plane.

MC: In your own words describe a typical nights sleep now.

BR08: Very much more relaxed... Eh,... I tend to go to bed slightly later actually ...maybe about a quarter past twelve... and I would tend to be asleep within fifteen minutes. My wife will wake up about five or six o’clock to go to work and I tend to wake up just before the alarm goes off. But not much about half an hour. The alarm goes off at 7 but I
tend to have a cup of tea and stay in bed till about half eight. I’ll listen to the radio and I might dose off and get up at 9.

MC: What do you do when you have trouble sleeping?

BR08: The interesting thing actually is that I do have trouble sleeping sometimes. Real trouble and it’s the old mind over matter thing. If there’s an event the following day it could be a social event or a concert whatever something says you’ve gotta get up if there is something on the following day maybe an event something I have got to do I tend to wake up before the alarm a long time before the alarm. Before when I was waking up it might be half an hour this is more like two hours its all very odd the mind is sort of saying you’ve got something to do you have got to go there get there there’s no need but there is no pressure at all it’s a strange thing its similar to before but its worse I think that that has happened since the accident it happens every two to three weeks tends to be when we are travelling somewhere nowhere important (How do you cope with this?) Curse Swear get furious tell myself not to be stupid but it doesn’t help I don’t understand its not important this is not critical you’ve gotta’ try to convince yourself I don’t understand it- its almost as if you are trying to put pressure on yourself because there isn’t any there it’s a strange thing its certainly there’s more anxiety about waking up following my accident this is worse than before but it shouldn’t be there not important events their only social events its not as if I have to my job is not on the line its strange so that is the one thing I would say due to an anxiety effect.
MC: In your own words describe a typical nights sleep before the injury.

GB09: [long pause] its kinda difficult to put into words I was quite happy with my sleeping pattern you went to sleep you went to bed and you had to get up when the alarm went off thats the bit I didn’t like but I have no complaints about my sleeping pattern.

MC: In your own words describe a typical nights sleep now.

GB09: Well the difference now I’d say is that I am inclined to go to bed earlier I am talking about 10 o’clock put my head down then but if we happen to be out for a meal or at the bowling club it might be later than that when I get home I am finished and want to go to my bed. I have quite a busy day I am tired and I want to go to bed its not as if I’ve time on my hands and nothing to do though that might be tiring in itself I am keeping myself busy the sleep is much the same I don’t sleep any longer ‘cos the alarm goes of for my wife and I tend to get up at the same time as I always have and take her to her work just before eight.

MC: What do you do when you have trouble sleeping?

GB09: If I couldn’t sleep I can’t really say. I’ve never had any trouble after having the bump on the head I thought I might have problems with headaches and things like that seemingly it was bad when I was in hospital but there is no pain now.
MC: In your own words describe a typical nights sleep before the injury.
MB10: Then it dynamite lights out then not hear another thing for nine hours need dynamite to get me up (what would wake you up?) she’s sitting over there (motions to his mother and laughs).

MC: In your own words describe a typical nights sleep now.
MB10: The same dynamite sometimes wake up and listen to music but it seems to be as deep its not disturbed probably sleep for shorter periods

MC: What do you do when you have trouble sleeping?
MB10: Depend what time of day it was if at night I would just roll over and close my eyes I would put on my tunes maybe if it was early evening or early morning.
MC: In your own words describe a typical nights sleep before the injury.
KS11: I was good as soon as I fell asleep that was me till the afternoon the next day [he was working shifts as a barman for one and a half years] I would have to set an alarm or I would sleep through

MC: In your own words describe a typical nights sleep now.
KS11: I stay up till about one to two in the morning but I still wake up about half 6 in the morning this is the exact time I would wake up for the last job I had I automatically wake up the time I would have I cannae get back to sleep I just get up I dinnae have to get up but I do it’s a lighter sleep I sometimes wake up through the night to see what the time is and then go back to sleep.

MC: What do you do when you have trouble sleeping?
KS11: I just get up an watch the telly it happens quite a lot but now more so I tend to just wake up really early at 6:30 it will be handy if I get a job (laughs).
MC: In your own words describe a typical nights sleep before the injury.

KM12: Ehmm... 11 to 12 probably keep the light on and read a novel then I start to feel sleepy I maybe read half a page and then put the light off and go to sleep. I would usually sleep right through and keep hitting the snooze button in the morning and not get up until I really had to.

MC: In your own words describe a typical nights sleep now.

KM12: Probably about the same getting off to sleep ... but I tend to wake up in the early hours of the morning and I can’t get back to sleep if I am doing lots of “on call” I don’t usually get to bed ‘till about 2 in the morning. Last night I was awake from 4 in the morning through to six thirty. Then I managed to sleep till seven fifteen when the alarm went off this tends to happen most days for a week then its OK for a week since the accident at the beginning when I was in hospital and after it was hard to sleep because of my sore head I used to joke that I needed a really soft pillow when I came home the pillows in hospital were like bricks and the beds were so uncomfortable I was just so tired all the time. Now because I’m working shifts there is a lot of variation. I might lie there and say I am not going to be able to sleep when I was at university it was different since the accident I have changed my work pattern I am now working fulltime and have changeable working hours (as a house officer) the waking up through the night is particularly bad after I come off nights.
MC: What do you do when you have trouble sleeping?

KM12: I get up look for stuff get my bag packed for next day packed lunch. I might eat something or read a book normally just do something anything tend to catch up on my lost sleep shift patterns mean I work nine to five everyday, with a long day nine to ten every one to two weeks and one in ten weeks I am on nights.
MC: In your own words describe a typical nights sleep before the injury.
JW13: Mostly three in the morning to start and stop whenever you are finished then home go to the shops get something to eat then go to bed about seven and sleep get up about 2 in the morning for the next day [works shifts as a lorry driver]

MC: In your own words describe a typical nights sleep now.
JW13: I’ve ‘gotta…Ehm… when you go to bed you can’t …Ehm.. sleep it might take 3 or 4 years..Eh hours to get to sleep..Ehm I did take some sort of table for that but it ‘didnae help Ehm when I actually do sleep I am up about eight and then during the day I might go back down during the day for an hour. I normally go to bed. Try about eleven most nights I have problems I did take the what do you call them the pills tablets but I don’t use them I would have to have something to stop the drowsiness when I am in the bed you can’t stop thinking nothing it could be anything maybe that’s Ehm things during the day. OK say if I go to bed at eleven and can’t get to sleep until three during that time your thinking about things something actually anything about Ehm living its something that I might be thinking about [is it worries?] it s not different there its no problem if you don’t think about it but if you don’t think about that it would be something else. If you try and stop thinking about it something else will come in not that its its something else anything about its like what will I do tomorrow that sort of thing.

MC: What do you do when you have trouble sleeping?
JW13: Nothing really I just wait until sleep eventually comes it will come eventually.

whats the word not angry [frustrated?] aye I get frustrated sometimes. Aye maybe angry about not being able to sleep sometimes.
MC: In your own words describe a typical nights sleep before the injury.

JG14: I usually like to read three books a week from the library. I would fall asleep and drop the book I’d go to sleep and the light just wakes me never had an alarm just rely on the daylight through the curtains.

MC: In your own words describe a typical nights sleep now.

JG14: Last night we were out at a concert went to bed just turned 12 o’clock not normally so late the last time we got a video from the video shop about a ghost and sat and watched it till 12 o’clock when I get up I have toast and fruit and fibre and coffee and I read the paper right through till 9 o’clock I get over to sleep no bother I am bad for going to sleep I am maybe sitting here in the afternoon and I dose off sometimes about once a week I get cramps in my legs and I have to throw the covers off and do this (rubbing his leg) sometimes the rain keeps us awake you hear it stoating off the slates but its easy to get back to sleep. I get up in the morning and have a good wash before I come down the stairs I have a shower now like the one in hospital..

MC: What do you do when you have trouble sleeping?

JG14: No times I’ve never had it that I cannae sleep I sleep a bit more now I sit her from half six and sometimes the wife gives me a row for falling asleep.
MC: In your own words describe a typical nights sleep before the injury.

SA15: What do you mean [you'd go to sleep when?] I’d usually watch a bit of telly and have a fag before I’d go to bed I’d set the alarm on the telly so it goes off in half an hour a sleep timer thing then I get up with the alarm I usually wake up with or around the alarm time.

MC: In your own words describe a typical nights sleep now.

SA15: I dinnae go to sleep till the back of 12 1 o’clock and I still wake up about seven my alarm goes off and mum makes sure I get up I am never tired I try and do things before bed go out for a walk and watch telly and then I try to get over to sleep I sometimes sleep during the day after work which just knocks my sleep pattern I’ve spoken to the Doctor about it but he dissnae want to give me anything because sleeping tablets are addictive its noticed at work ken I am always yawning if I’ve got a day off I am sleeping till lunch time I wasnae as tired as I was before the accident I’d sleep till maybe 10 things maybe wake me up easier maybe the birds out side wake me up maybe I’m a lighter sleep I just never get tired cannæ remember my dreams have no remembered my dreams for a long time I dinnae really remember any dreams now. I just cannæ get to sleep now when I was in hospital I couldnae sleep either the night staff would come and talk to me if I was awake for hours on end I always woke up with people getting up early.
MC: What do you do when you have trouble sleeping?

SA15: Watch TV have a few cigarettes and drink tea or put the computer on and play a game or if its that bad I will just get in the car and drive and come back about an hour later.
MC: In your own words describe a typical nights sleep before the injury.

DS16: I would go to bed watch a bit of TV that would be me if I was night shift it would be 10 in the morning I might wake up through the night to see the time of go to the toilet

MC: In your own words describe a typical nights sleep now.

DS16: Ehmm well usually try to got op bed for half nine ten fall asleep round about midnight and then I am up for work about 6 o’clock and away to work about half six. am day shift constantly now

MC: What do you do when you have trouble sleeping?

DS16: I don’t really know I’d wait until I had to fall asleep. ’d wait in bed till I fall asleep.
MC: In your own words describe a typical nights sleep before the injury.

SS17: I didn’t have any problems waking up through the night now I wake up with cramps and that I just went straight to bed and and the alarm or someone would wake me up. I go to bed and I am really tired and then I am wide-awake and cannæ sleep

MC: In your own words describe a typical nights sleep now.

SS17: It varies I might go to bed at 12 and lie in bed and have to get up ‘cos I am not tired at all times I cannæ get to sleep till 9 the next day then end up sleeping all day for about 10-12 hours then I would just lie in bed knackered for the next day I think I had something to do through the day I might not be as tired through the day I’ve got pins through my leg and problems with my hip and the cramps sometimes wake me up through the night most nights especially if its cold during the night

MC: What do you do when you have trouble sleeping?

SS17: Just sit and watch the T.V listen to music or go on the Internet sometimes it helps.
MC: In your own words describe a typical nights sleep before the injury.

DS18: I am pretty late to bed over to sleep no problem wake up quick shower and never had to use an alarm clock.

MC: In your own words describe a typical nights sleep now.

DS18: By elevenish I am getting pretty tired then I go to bed I might sleep five to ten hours have a really deep sleep my dreams seem more vivid sometimes I can’t tell whether the dreams are real you know if it really happened or if it was a dream I get up about eleven or twelveish (PM). Now I sleep really soundly and deeply for long periods at a time the door bell the phone people yelling just don’t wake me

MC: What do you do when you have trouble sleeping?

DS18: Before I started my tablets (SSRIs) was tossing and turning would force myself to stay in bed and not be able to get over (to sleep) at all
MC: In your own words describe a typical nights sleep before the injury.
SM19: I would watch some TV go to bed then out like a light when the head hit the pillow and the same most nights then up with the alarm for work the next day

MC: In your own words describe a typical nights sleep now.
SM19: Much the same I maybe get up to the toilet more but mostly the same

MC: What do you do when you have trouble sleeping?
SM19: I never really have any bother I’d just try to get over to sleep lie in bed.
MC: In your own words describe a typical nights sleep before the injury.

AR21: Before the accident I would go to bed about half nine when I got in and then I would get up about 2 or 3 in the morning to do my paperwork I only slept about 2 hours then I was awake and could not get back to sleep and I always got up and did the paperwork and that was me I think all the worries woke me up.

MC: In your own words describe a typical nights sleep now.

AR21: I actually go to bed later but sometimes I go to bed about half past 6 and when I was on that downer I wanted to stay in bed all the time but normally about half eight to nine I go to bed quickly and then that’s me for about an hour and hour and a half and then that’s me I might get up and watch the TV I cat nap sometimes I walk the street or is out hovering out the car and washing [what wakes you up?] my heads not right maybe sometimes dreams and pain I like to move about and keep the muscles going its not a deep sleep maybe it will last about five or twenty minutes when I close my eyes my forehead and nose go numb then I go to sleep it could be 5 minutes it could be half an hour the length is short always up at half four five o’clock.

[Dreams?] They are always getting chased by something or I am chasing something and it always wakes me up and I feel a bit shaky and sweaty.

MC: What do you do when you have trouble sleeping?
AR21: Just lie there with my eyes shut 9 times out of ten I just get up [from above I might get up and watch the TV I cat nap sometimes I walk the street or is out hovering out the car and washing]
Appendix F

F1 Insomnia within the psychobiological inhibition model

F2 The Good Sleep Guide
Table F.1 Insomnia within the psychobiological inhibition model

<table>
<thead>
<tr>
<th>Factors contributing to good sleep</th>
<th>Insomnia: factors inhibitory to sleep homeostasis and the circadian timing and to the protection of good sleep afforded by automatic and plasticity</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Sleep-stimulus control</strong></td>
<td></td>
</tr>
<tr>
<td>• Sleep compatible conditioning</td>
<td>Conditioned association of sleep-incompatible, waking activities (e.g. reading, watching TV, eating, talking, problem-solving) with bed and bedroom environment; keeping the light on.</td>
</tr>
<tr>
<td>• Sleep-wake Sensitivity/specificity</td>
<td>Environmental latitude in sleep and wake behaviours: lying awake in bed either presleep or upon wakening, sleeping in the day, sleeping elsewhere than in bed.</td>
</tr>
<tr>
<td>• Regular sleep habits</td>
<td>Variable and/or reactive patterns: changing times for retiring and rising, extending time in bed to catch up on sleep, sleeping in at weekends, spending longer in bed than current sleep requirements-Reduced sleep efficiency</td>
</tr>
<tr>
<td><strong>Physiological de-arousal</strong></td>
<td></td>
</tr>
<tr>
<td>• Sleep system engagement</td>
<td>Not feeling tired at bedtime, in bed too early, keeping the light on, sleep-incompatible activities, anxiety, trying too hard to sleep, tension, heart rate variability</td>
</tr>
<tr>
<td>• Wake system disengagement</td>
<td>As above; active thinking and problem-solving, self-monitoring of internal (bodily or mental) cues, hypervigilance, poor sleep hygiene</td>
</tr>
<tr>
<td>• Good sleep hygiene</td>
<td>Stimulants (e.g. caffeine, nicotine) in excess/ near bedtime; alcohol withdrawal symptoms during the night.; active exercise late evening; bedroom stuffy, hot or cold; bed uncomfortable.</td>
</tr>
<tr>
<td><strong>Cognitive de-arousal</strong></td>
<td></td>
</tr>
<tr>
<td>• Minimal cognitive drive</td>
<td>Rehearsing/planning/problem-solving thoughts in bed, thinking about events the previous or next day, preoccupations with sleep/sleepiness, “stimulant hungry” mind, mind racing, unable to “switch off”.</td>
</tr>
<tr>
<td>• Accurate sleep-wake attribution</td>
<td>Dysfunctional beliefs and attitudes about sleep and consequences of not sleeping, not expecting to sleep, catastrophic thoughts, concern about next day well-being and coping.</td>
</tr>
<tr>
<td><strong>Affect regulation</strong></td>
<td></td>
</tr>
<tr>
<td>• Minimal affect</td>
<td>Worry, anxiety, frustration, negativity or excitement, intensity in emotional tone associated with above cognitive or physiological processes</td>
</tr>
<tr>
<td>• Minimal effort</td>
<td>Sleeplessness preoccupying: trying to overcome sleep/overcome insomnia, attempts to suppress affect, self-monitoring of alert/sleepiness state, performance effort to fall asleep, performance anxiety.</td>
</tr>
<tr>
<td><strong>Daytime facilitation of night sleep</strong></td>
<td>Attribution of impaired daytime mood, attention, performance to quality of sleep; expectation that sleep should compensate; blaming problems on insomnia; fatigue seen as pathognomonic of insomnia; perception of self as insomniac.</td>
</tr>
<tr>
<td>• Effective coping skills</td>
<td>Experiencing time pressure; problems relaxing; worry frustration, low mood; active late into the evening; poor wind down.</td>
</tr>
</tbody>
</table>

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THE GOOD SLEEP GUIDE

DURING THE EVENING

1. Put the day to rest. Think it through. Tie up "loose ends" in your mind and plan ahead. A notebook may help.

2. Take some light exercise early in the evening. Generally try to keep yourself fit.

3. Wind down during the course of the evening. Do not do anything that is mentally demanding within 90 minutes of bedtime.

4. Do not sleep or doze in the armchair. Keep your sleep for bedtime.

5. Do not drink too much coffee or tea and only have a light snack for supper. Do not drink alcohol to aid your sleep - it usually upsets sleep.

6. Make sure your bed and bedroom are comfortable - not too cold and not too warm.

AT BEDTIME

1. Go to bed when you are "sleepy tired" and not before.

2. Do not read or watch TV in bed. Keep these activities for another room.

3. Set the alarm for the same time every day - 7 days a week, or at least until your sleep pattern settles down.

4. Put the light out when you get into bed.

5. Let yourself relax and tell yourself that "sleep will come when it's ready". Enjoy relaxing even if you don't at first fall asleep.

6. Do not try to fall asleep. Sleep is not something you can switch on deliberately but if you try to switch it on you can switch it off!
THE GOOD SLEEP GUIDE

IF YOU HAVE PROBLEMS GETTING TO SLEEP

1. Remember that sleep problems are quite common and they are not as damaging as you might think. Try not to get upset or frustrated.

2. If you are awake in bed for more than 20 minutes then get up and go into another room.

3. Do something relaxing for a while and don’t worry about tomorrow. People usually cope quite well even after a sleepless night.

4. Go back to bed when you feel “sleepy tired”.

5. Remember the tips from the section above and use them again each time you waken up.

6. A good sleep pattern may take a number of weeks to establish. Be confident that you will achieve this in the end by working through the “GOOD SLEEP GUIDE”!

This guide has been prepared by Professor Colin A. Espie, University of Glasgow, Department of Psychological Medicine, Gartnavel Royal Hospital, 1055 Great Western Road, Glasgow G12 0XH.