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**SEVERE TRAUMATIC BRAIN INJURY:
LONG TERM EFFECTS ON SLEEP, SLEEPINESS AND PERFORMANCE**

by

Claude Manseau

**A thesis submitted to
the Faculty of Graduate Studies and Research
in partial fulfillment of
the requirements for the degree of
Doctor of Philosophy**

Department of Psychology

**Carleton University
Ottawa, Ontario
November, 1994**

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**SEVERE TRAUMATIC BRAIN INJURY:
LONG TERM EFFECTS ON SLEEP, SLEEPINESS AND PERFORMANCE**

submitted by

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in partial fulfillment of the requirements
for the degree of Doctor of Philosophy

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January 1995

ABSTRACT

Traumatic brain injured (TBI) victims often complain of "fatigue" or sleepiness, which is expressed as decreased efficiency in performance. Prior sleep studies done with TBI subjects have mainly concentrated on the coma and early post-coma periods. These studies showed that the reappearance during coma of typical polygraphic sleep patterns (NREM-REM stages) and organized circadian activity (sleep-wake) were associated with a favorable prognosis. On the other hand, in the early post-coma period, the sleep components stabilized and were comparable to those of matched controls except for greater awakenings at night. The aim of the present study was to investigate the nature of sleep, sleepiness and performance in the late stage of recovery.

Sixteen non-medicated TBI subjects (15 males, 1 female; mean age 29) differing in their degree of subjective sleepiness were compared to matched controls (mean age 28). Mean coma duration was 24 days and mean time interval since the accident was 6 years. A screening polysomnography was performed to exclude subjects with significant sleep apnea and periodic leg movements, conditions which could have been present before the accident. Following a second consecutive overnight sleep recording, an objective measure of daytime sleepiness, the Multiple Sleep Latency Test (MSLT), was done. It consisted of five 20-minute nap periods scheduled at 0900, 1100, 1300, 1500 and 1700 hrs. Fifteen minutes before and 5 minutes after each MSLT nap, subjects performed a simple visual reaction time task for 10 minutes. Subsequently, all subjects had neuropsychological testing.

The major finding of this study was the presence of sleep onset and sleep maintenance insomnia in chronic TBI subjects who reported high levels of subjective sleepiness during the day. Difficulty in falling asleep was also found during daytime recording, suggesting the presence of a general disorder of initiating sleep. The insomnia of these subjects was most likely of an organic type since none had a history of sleep problems before their accident and none showed significant affective disorders at the time of testing. Moreover, there was the presence of a marked degree of correlation between subjective sleepiness and neuropsychological results. Thus, the excessive somnolence often reported by TBI subjects may result from chronic insomnia possibly produced by damage to cortical centers, particularly in the basal forebrain region, which are most vulnerable to this type of insult and act to inhibit the arousal system during sleep initiation.

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INTRODUCTION

Traumatic brain injuries (TBI) represent a major health problem, especially in industrialized countries. Over 50% are caused by motor vehicle accidents (Marshall, Becker, Bowers, Cayard, Eisenberg, Gross, Grossman, Jane, Kunitz, Rimel, Tabaddor, & Warren, 1983). Indeed, the more severe the traumatic brain injury, the higher the probability that it has been incurred in a motor vehicle accident (Kalsbeck, McLaurin, Harris, & Miller, 1980). As a result of recent advances in medical life-saving measures, an ever-increasing number of these severe TBI victims survive but are left with a constellation of sequelae affecting their personal, social, and economic lives.

Although ranking fourth as a cause of death behind heart disease, cancer, and stroke (Cartlidge & Shaw, 1981), traumatic brain injuries are considered the most grievous (Ommaya, 1983). Firstly, they represent the leading cause of morbidity (as well as mortality) in the young (those below the age of 45) and thereby have the most significant socio-economic effects (Frankowski, 1986). Secondly, traumatic brain injuries result in various problems of cognition and behavior impairments that are more detrimental, and last much longer, than those of heart disease, cancer or stroke (O'Shanick, 1986). It has been estimated that traumatic brain injuries in Canada cost at least 4 billion dollars per year (Ivan, 1984). These costs can only escalate since the ranks of traumatically brain injured persons grow yearly (Parkinson, Stephensen, & Phillips, 1985).

Given this historically recent and tragic phenomenon of young people having to face

such disability for life, a large number of studies have been undertaken in the last decade which focus on the rehabilitative aspects of TBI. As aptly expressed by Jennett and Bond (1975), success with this population of patients should be measured less by the fact of survival than by the quality of survival. Neurobehavioral changes following TBI have been extensively documented (Levin, Grafman & Eisenberg, 1987; Richardson, 1990). They consist mainly of cognitive (e.g. attention, memory, abstract reasoning) and personality (e.g. irritability, impulsivity, emotional lability) changes.

One of the most troublesome problems affecting many TBI victims is a propensity for a pervasive feeling of fatigue (Lezak, 1978; McMillan & Glucksman, 1987). This tiredness appears to consist both of excessive daytime sleepiness and decreased efficiency in performance capacity. In the sleep research field, a larger literature from sleep deprivation, altered sleep/wake schedules and clinical sleep disorders associates daytime sleepiness with a variety of problems related to the ability to perform and, in particular, to sustain performance (time on task, "fatigue"). This deficit has been discussed for instance in several recent volumes (Monk, 1991; Broughton and Ogilvie, 1992). Notwithstanding, surprisingly few studies, and essentially no well-controlled ones, have been undertaken regarding this disabling aspect of TBI. The present study, therefore, aims at investigating the nature of sleepiness and performance after traumatic brain injury.

A brief review of the neuropathology of traumatic brain injury is first presented. Traumatic brain injuries are usually divided into two categories: missile or penetrating injuries, and nonmissile, blunt or closed head injuries (CHIs). The missile injuries generally

involve a different pathogenesis than the nonmissile ones. The present review focuses on the pathology which results from nonmissile injuries since they currently encompass most traumatic brain injury victims (e.g., road accidents, falls) and were the cause of CHI in the subjects studied in the present research. This review of neuropathology is pertinent because a variety of sleep disorders including insomnia, hypersomnia and disturbances of circadian sleep-wake regulation can be caused by central nervous system (CNS) lesions in various structures involved in sleep initiation, sleep maintenance, wake maintenance and circadian regulation. In that regard, a brief outline of the actual knowledge about sleep mechanisms is presented in the section on sleep and sleepiness after closed head injury. This is then followed by a literature review of the studies done on sleep-wake patterns and performance after closed head injury. Next, the hypotheses of the present research are advanced, followed by a detailed account of the experimental protocol. Finally, the results are presented and discussed.

NEUROPATHOLOGY OF CLOSED HEAD INJURY

There are two main mechanisms operating in closed head injuries, namely contact and acceleration-deceleration forces (Teasdale & Mendelow, 1984). Recent studies with monkeys have demonstrated that all the pathologies, with the exception of skull fracture and epidural hematoma, which occur in man as a result of a nonmissile traumatic brain injury can be produced by inertial loading only, i.e., nonimpact angular acceleration-deceleration forces (Adams, Graham, & Gennarelli, 1982; Gennarelli, Thibault, Adams, Graham, Thompson, & Marcincin, 1982). These pathologies include focal damage such as surface contusion, intracranial hematoma, and generalized damage including diffuse axonal injury, swelling (e.g. edema), and hypoxic change. Depending on the degree, direction, and duration of these acceleration-deceleration forces, different types of traumatic brain injury can occur. For instance, in falls where the acceleration rate is rapid and its duration relatively short, subdural hematoma tends to predominate, whereas in vehicular injuries where the acceleration rate is slower and its duration longer, diffuse axonal injury is more common (Gennarelli, 1983).

Although there is an increasing tendency to describe closed head injuries in terms of focal and diffuse patterns of neuropathology (Adams & Graham, 1984; Gennarelli, Spielman, Langfitt, Gildenberg, Harrington, Jane, Marshall, Miller, & Pitts, 1982), damage following a closed head injury can also be separated into lesions which are primary or directly attributable to impact and consequently not amenable to treatment, and lesions which are secondary to or the result of subsequent pathologic processes and which, if detected early

enough, are potentially preventable (Adams, 1975, 1990; Adams, Graham, Scott, Parker, & Doyle, 1980; Jennett & Teasdale, 1981). Thus, in some cases, the damage sustained at the moment of impact may have been so severe that the individual lost consciousness, whereas in other cases the individual may have been lucid following the impact but subsequently became unconscious, suggesting the presence of complications which may be avoidable.

Primary Damage

Impact causes skull deformation and may produce skull fracture. Though a pathognomonic sign of brain injury, skull fractures are associated with brain damage only about half of the time (Jamieson & Yelland, 1968). They are least common in cases with the greatest amount of diffuse brain injury (Adams, Mitchell, & Graham, Doyle, 1977).

Linear fractures account for approximately 75 % of all skull fractures (Levin, Benton, & Grossman, 1982). However, depressed fractures (25 %) are of greater importance, because they represent a potential source for intracranial infection, especially when such fractures involve the base of the skull (Adams, 1975, 1990). Furthermore, these comminuted fractures are often associated with posttraumatic epilepsy (Jennett, 1972).

Distortion of the skull often results in contusions of two types: coup and contrecoup. Coup contusions occur directly below the site of impact and include lacerations, i.e., local lesions resulting from the detachment of dura and/or breachment of the pia-arachnoid. Contrecoup contusions, on the other hand, occur away from the site of impact and result

from relative inertia of the brain following impact (Gurdjian & Gurdjian, 1976). Regardless of the site of impact, contrecoup lesions following a closed head injury occur most frequently on the undersurfaces of the frontotemporal regions of the brain. This is due to the presence of bony irregularities at the base of the skull, such as the sphenoid ridges, that abrade the cortex (Courville, 1971; Ommaya, Grubb, & Naumann, 1971). Gennarelli and his colleagues (1979) have reported that the frontal lobes are more susceptible to contusions than the temporal lobes, suggesting that temporal contusions rarely occur without frontal pathology. From a contusion index based on depth and extent, Adams, Scott and their associates (1980) also found that contusions were more severe when skull fracture was present and less severe in patients with diffuse damage to white matter.

In their first studies with monkeys using a brain accelerating device, Ommaya and Gennarelli (1974) showed that mild brain injury damage is mainly limited to the surface of the brain but that severe injury extends inward to affect the diencephalic core. They also confirmed the previous findings of Holbourn (1943) and Pudenz and Shelden (1946) that rotational acceleration (i.e., a swirling motion of the brain within the skull) results in shear strain. This mainly occurs at interface between neural structures of different consistency such as at the edge of grey and white matter. Strich (1956) was the first investigator to clearly define this diffuse damage. She reported that the shearing forces induce tearing of small blood vessels and nerve fibers at the time of injury resulting in microscopic changes such as small haemorrhagic and white matter lesions (Strich, 1970). Similar anatomic changes have also been found in cases of mild concussion (Oppenheimer, 1968) and are most probably responsible for the postconcussive syndrome (Binder, 1986).

Although widespread, such diffuse injury is greater in irregular surfaces of the brain (i.e., frontal and temporal poles) and less in relatively smooth surfaces (i.e., occipital lobes). Furthermore, this diffuse damage to white matter is seemingly the pathological basis of so-called 'primary brain-stem injury' in patients who remain in the persistent vegetative state until death and in whom increased intracranial pressure can be ruled out (Adams et al., 1977).

Intracranial hematomas, which are formed by shearing forces tearing blood vessels, represent space-occupying clots and are classified according to their anatomic site (extradural, subdural, intracerebral). They are somewhat similar in their anatomic distribution to contusions being most common around the orbital frontal and temporal areas.

Brain edema, which is an abnormal excess accumulation of fluid, is a commonly occurring and threatening complication of traumatic brain injury (Miller, 1987). It is the most common neurodiagnostic finding in pediatric head trauma. Brain edema which results mainly from a loss of autoregulation of the cerebral vasculature is often the cause of acute cerebral swelling, one of the main forms of secondary damage in closed head injury.

Secondary Damage

Besides causing local damage, space-occupying lesions such as hematomas can also initiate secondary insults by pressure effects. These include diffuse ischemic damage, brain midline shift and herniation. The most common type of herniation is transtentorial or uncal

which compresses brain stem structures harboring the centers responsible for wakefulness (Crompton, 1985). This represents the apparent mechanism by which some patients fall into a coma only after a delay following the impact.

Similarly, if swelling from brain edema reaches a certain threshold, there are a myriad of effects which are created by the increased intracranial pressure (ICP) which has been estimated to occur in more than 75% of patients with severe closed head injury and to account for approximately 50% of the mortality (Miller, Becker, Ward, Sullivan, Adams, & Rosner, 1977). Increased ICP acts by compressing the brainstem resulting in coma and with time (higher pressure) in death. Early monitoring and control of intracranial pressure are the main reasons for the current prognosis for survival being better. Indeed, neurosurgical treatment concentrates on preventing this secondary complication either by surgically evacuating the localized hematoma, or by reducing the edema with agents such as steroids. Hyperventilation with a mechanical respirator is also used to maintain blood pressure within normal limits. Another complication which can necessitate surgical treatment is infection, especially for abscess formation in cases with compound fracture of the skull.

What is most characteristic of TBI following a closed head injury is diffuse axonal injury (DAI) which consists of a triad of signs (focal lesions in the corpus callosum and in the dorsolateral quadrants of the rostral brainstem plus diffuse microscopic damage to axons). This represents a distinct clinicopathological entity resulting most frequently from motor vehicle accidents (Adams, Graham, Murray, & Scott, 1982). As previously stressed, the etiology of DAI is not necessarily related to impact, but to acceleration-deceleration forces

which can at times be significant without actual skull impact.

Diffuse axonal injury can be regarded as one end of a continuum of injury severity resulting from acceleration-deceleration forces. Gennarelli (1993) has described 4 levels of diffuse brain injuries: 1) mild concussion, involving a temporary disturbance of neurological function without loss of consciousness; 2) classical cerebral concussion, consisting of a reversible neurological impairment with temporary loss of consciousness (less than 24 hours); 3) diffuse injury, representing brain trauma with loss of consciousness greater than 24 hours and resulting in residual neurological, psychological, and personality defects; and 4) diffuse white matter pathology involving considerable axonal damage throughout the brain plus brainstem dysfunction and accompanied by prolonged loss of consciousness.

SLEEP AND SLEEPINESS AFTER CLOSED HEAD INJURY

Closed head injury often produces alterations in sleep-wake patterns, especially in severe cases accompanied by a loss of consciousness. This is not surprising considering that the midbrain and brainstem structures vulnerable to traumatic brain injury harbour the complex neurophysiological mechanisms responsible for the regulation of sleep-wake states. Before reviewing the studies on sleep and sleepiness after closed head injury, a brief review of the present state of knowledge about sleep-wake processes will be presented.

Initially, sleep was thought to be a passive unitary process. However, modern research has demonstrated that it is an active dual process comprising two distinct states: slow wave sleep (SWS) or non rapid eye movement (NREM) sleep and paradoxical sleep or rapid eye movement (REM) sleep. Within NREM sleep, four stages are usually recognized (stage 1 to 4). Different neuroanatomical regions and neuronal systems with specific chemical neurotransmitters and widespread projections throughout the brain are involved in the generation and maintenance of wakefulness and sleep.

At the neuroanatomical level, wakefulness results from the arousal mechanism of the ascending reticular activating system which is a neuronal system localized in the brainstem and associated with the intrinsic thalamic nuclei and the posterior hypothalamus (Moruzzi & Magoun, 1949; Mancina & Mariotti, 1985). Recent studies have found that a circadian process of arousal exists which competes with a homeostatic sleep process (Edgar, Dement, & Fuller, 1993). Sleep, on the other hand, consists basically of the inhibition of this central arousal

system. Thus, sleep onset, which leads to SWS, involves the active inhibition of the reticular activating system by different brain regions located in subcortical regions: the basal forebrain, more specifically the preoptic region of the hypothalamus (Serman & Clemente, 1962), the anterior thalamic region (dorsomedial nuclei) (Lugaresi, Medori, Montagna, Baruzzi, Cortelli, Lugaresi, Tinuper, Zuconi, & Gambetti, 1986), and in a lower brainstem medullary region (possibly the tractus solitarius) (Magne, Moruzzi, & Pompeiano, 1961). The other state of sleep, known as paradoxical sleep or rapid eye movement (REM) sleep follows NREM sleep and is dependent upon specific pontine nuclei (Seigel, 1989).

At the neurochemical level, there is a dynamic interaction of several neurotransmitters. Arousal involves both dopaminergic (behavioral arousal) and cholinergic (cortical arousal) mechanisms, whereas sleep induction and maintenance of NREM sleep appear to be governed by serotonin produced in the raphe nuclei of the midbrain, and REM manifestations involve cholinergic and gamma-aminobutyric acid (GABA) mechanisms (Jones, 1989).

Sleep-wake patterns represent a sensitive indicator of the integrity of basic cerebral functions after traumatic brain injury. Indeed, early studies on sleep patterns in TBI patients were primarily concerned with their recovery as a prognostic criterion in posttraumatic coma. Chartrian and his colleagues (1963) were the first to report sleep electroencephalographic (EEG) patterns (i.e., spindles, vertex sharp waves, K-complexes) in comatose patients who had sustained traumatic brain injuries. They also found that the presence of these normal physiological components was associated with a favorable outcome. Subsequently, Passouant

and his colleagues (1965) showed a relationship in TBI patients between the return of the normal cyclic organization of sleep and the improvement of the state of consciousness. These findings were corroborated by more detailed studies (Bergamasco, Bergamini, & Doriguzzi, 1968; Bricolo, Gentilomo, Rosadini, & Rossi, 1968), using repeated follow-up recordings. These authors demonstrated that favorable prognosis was associated with the presence of typical polygraphic sleep patterns of both NREM and REM sleep stages and organized circadian sleep-wake patterns, whereas a fatal prognosis was related to biphasic or monophasic nocturnal sleep activity. A similar study with control subjects came to the same conclusion (Lessard, Sances, & Larson, 1974).

Individual stages of sleep have also been shown to be selectively affected, depending on the site of cerebral damage particularly in the brainstem region (Markand & Dyken, 1976). In some cases, stages 3 and 4 of NREM sleep were abolished while REM sleep was left intact (Jouvet, Pellin, & Mounier, 1962). Other cases presented marked disorganization of NREM sleep as well as a low level or absence of REM activity (Harada, Minani, Haltori, Nakamura, Kabashima, Shikai, & Sakai, 1976).

Thus, the reappearance of normal polygraphic sleep-wake patterns has been found to indicate a good prognosis. The recovery may take weeks or months depending on the severity of the trauma. It is typically gradual and follows a pattern of coma, then sleep disturbances, and finally normal regulation of vigilance functions. Focal EEG abnormalities tend to disappear within 6 months to 2 years in the majority of cases (Courjon, 1972).

Subsequent studies on sleep-wake abnormalities focused mainly on the post-coma phase. A correlation between cognition and sleep patterns, more specifically between REM sleep, has been shown during the recovery process after traumatic brain injury. Ron and colleagues (1980) repeatedly recorded sleep EEG in 9 severe TBI patients (5 of whom had sustained a closed head injury) during the first six months after their accident. In 7 of them, the recovery of REM sleep was correlated with the recovery of intellectual functions as assessed by a neuropsychological test battery. In 2 patients only, a low correlation was found between REM sleep and cognition which was explained by the presence of a brainstem lesion which had probably damaged the pontine neural mechanisms for control of REM sleep.

Using single night sleep recordings done 6 to 59 months post-injury, Prigatano and his colleagues (1982) showed reduced stage 1 sleep and a greater number of awakenings in a group of 10 severe TBI subjects compared to matched controls. These TBI subjects complained of sleep or dream problems, or both. Neuropsychological and psychosocial variables were also assessed. Contrary to the findings of Ron and associates (1980), no correlation was found 6 months or more after the injury between REM measures (percent and latency) and cognitive functioning (tests of attention, memory, and intelligence). The only significantly correlated ($p < .05$) variables were the reduced amount of stage 1 sleep and the increased time to complete the Trail Making Test - part B (TMT-B), a visuo-motor tracking test. The latter suggested an impaired ability for sustained arousal. Many of these severely TBI subjects were reported to yawn frequently during wakefulness.

Only one study has directly assessed posttraumatic sleepiness as its main objective

(Guilleminault, Faull, Miles, & Van Den Hoed, 1983). The subjects were a heterogeneous group of 20 TBI patients (9 severe, 5 moderate, and 6 mild) in whom the unifying dimension was a complaint of excessive daytime sleepiness (EDS). In fact, twelve of the group had been forced to curtail or cease their work because of EDS. All subjects had sustained their injury less than 2 years prior to the study and none were suffering from a seizure disorder. Testing involved nocturnal and diurnal polygraphic recordings and was initiated on average 5 months after the accident with follow-ups on average 2 years thereafter based on the patient subjective reports only. Protocols, especially the daytime evaluation, differed considerably since the patients were referred over a 12 year period and were seen in two different sleep laboratories. Specifically, 5 patients underwent continuous polysomnographic monitoring for 24 to 36 hours while free to occupy their time as they wanted; 6 patients were requested to take 1 monitored nap at 1400 h, lasting for at least 1 hour; 3 patients took 2 30-minute monitored naps at 1030 h and 1400 h; finally, 6 patients were administered the Multiple Sleep Latency Test (MSLT) (Carskadon & Dement, 1982).

The MSLT represents an objective measure of daytime sleepiness which is based upon the premise that those individuals who are sleepy will fall asleep more readily than those who are not. The subject is requested to try and fall asleep as quickly as possible during five 20-minute naps at 2 hour intervals; the dependent measure is the mean latency to sleep onset. It has been estimated following initial studies with narcoleptics that a mean sleep latency of less than 5 minutes is indicative of pathological sleepiness (Richardson, Carskadon, Flagg, Van Den Hoed, Dement, & Mitler, 1978).

Guilleminault and his colleagues (1983) showed that 18 out of 20 patients were objectively sleepy. Sleep apnea, which results in fragmented sleep, was the apparent cause of EDS in 8 patients, 5 of whom improved over time. It is noteworthy that these sleep-related breathing problems were noted primarily in patients with whiplash associated with traumatic brain injury. In those who did not have sleep apnea, EDS was related to a nonspecific brain syndrome. There was a strong correlation between the severity of the initial disturbance of consciousness (duration of coma) and the severity of subjective EDS complaints. However, the objective measure of sleepiness (e.g., MSLT) was unrelated to the severity of subjective sleepiness. The majority of the severe TBI patients (coma lasting several days) did not improve significantly, despite the passage of time and multiple therapeutic trials of medication.

Interestingly, in those objectively sleepy patients who had the chance to take naps as desired, micro-sleeps were detected during continuous ad lib sleep monitoring. The frequency of these micro-sleeps, defined as brief bursts of theta activity, tended to increase until the patient decided to nap.

PERFORMANCE AFTER CLOSED HEAD INJURY

The first experimental performance study on the effects of traumatic brain injury appeared over 50 years ago. Using a battery of 30 different tests, Conkey (1938) provided quantitative evidence to confirm clinical observations that TBIs result in a variety of neurobehavioral deficits. One of the main deficits, she reported, was an inability to maintain a sustained level of performance due to attentional problems. Since then, a number of studies have been undertaken to elucidate the exact nature of this performance decrement in TBI victims, especially as it relates to attention and information processing (Gronwall, 1987).

Although there is as yet no consensus on the definition of attention, 3 components - alertness, selectivity, and conscious information processing - are believed to be involved (Posner & Boies, 1971); each is possibly mediated by a different neural substrate which together would form the attentional complex, a richly interconnected network (Posner & Rafal, 1987). Alertness refers to a generalized state of the central nervous system preparing the organism to respond. Changes in alertness can be tonic or phasic. The former is the result of physiological changes and is best illustrated by relatively slow biorhythmic fluctuations such as the post-lunch dip (Blake, 1967; Broughton, 1975); the latter, on the other hand, involve sudden changes in preparedness such as those following a warning signal. The primary structure mediating arousal is the reticular activating system (RAS) which has fibers broadly distributed to the cortex, the association areas being more densely innervated than the primary sensory areas (Mesulam, 1985). As described earlier, traumatic brain injury which results in brain stem and midbrain insults clearly impair tonic arousal initially, as

evidenced by loss of consciousness.

Selectivity which follows alertness changes in the sequence of attentional processes has to do with the facilitation of the processing of specific information. It enhances responsiveness to selected information. Different anatomical structures appear involved, depending on the type of information being processed (Cohen, 1993). For example, spatial selective attention such as the one involved in hemi-spatial neglect involves the parietal lobes mainly the right one. On the other hand, nonspatial selective attention seems to involve the thalamus. Frontal regions would apparently act as a superordinate control mechanism. Finally, conscious processing is seen as the last stage in the attentional process or complex. Contrary to the first two components which seem to proceed automatically and unconsciously, this last component is under the control of conscious volition and is instrumental for decision-making and response execution. This last aspect of conscious processing refers to vigilance. The amount of information processed by this conscious attention system is limited, as is its ability to be sustained over long periods of time. Conscious processing appears to be a fronto-limbic function. These three components of attention are interrelated; consequently dysfunction of one of them will affect the others. For example, sustained conscious attention (or concentration) is in part dependent upon maintaining adequate levels of alertness.

Few studies have been reported regarding the first postulated component of the attentional complex, i.e., alertness. Deficits in phasic alertness in closed head injured subjects have been most frequently investigated using the evoked potential (EP) technique. Such

studies (McMallum & Cummins, 1973; Rizzo, Amabile, Caporali, Spadaro, Zanasi, & Morocutti, 1978; Curry, 1980; Suffield, 1985) have found evidence for a diminished phasic increase in alertness following a warning signal for preparation for action which was even absent in some severe cases. Interestingly, the last two studies (Curry, 1980; Suffield, 1985) also demonstrated the opposite effect in some of their TBI subjects, that is, an exaggerated increase in phasic alertness. Suffield (1985) used the terms "underprocessing" and "overprocessing" to describe these two types of response. It is noteworthy that results of the RT tasks, though abnormal compared to controls, were nearly identical between the two groups. This pattern of responses is reminiscent of the Yerkes-Dodson effect that performance is optimal at medium levels of arousal and tends to fall off at either high or low levels of arousal (Yerkes & Dodson, 1908).

Concerning the tonic component of alertness, it had been hypothesized that the slowing of information processing found in TBI patients was caused in part by a lowered level of arousal which presumably resulted from a dysfunction of the brainstem reticular activating system (Gronwall & Sampson, 1974). In an attempt to test this under-arousal hypothesis, Van Zomeren (1981) recorded EEG activity and heart rate in control and TBI subjects while they were performing an auditory vigilance task of 30 minute duration. In control subjects, he found performance decrements especially in the second half of the 30 minute sessions with omissions being correlated with drowsy background EEG. The nine severely TBI subjects, on the other hand, showed persistently alert EEGs and higher heart rates throughout the sessions. Their performance, though poorer than for the control group, was reasonably stable and seemingly related to the severity of the traumatic brain injury.

These findings, therefore, do not support the hypothesis that subnormal performance in brain injured subjects is related to lower levels of tonic alertness. Quite the opposite, these findings imply that, when faced with a task, TBI subjects tend to be overaroused.

The only other study to systematically test sustained attention following traumatic brain injuries used a continuous (15 minutes) choice RT task and confirmed poorer performance in TBI subjects than their normal twins who participated as controls (Dencker & Lofving, 1958). More importantly, however, no decline in performance was observed in either group, suggesting that the task used might not have been appropriate for detecting any changes in vigilance. The task stimuli occurred at a predetermined fixed rate, whereas most studies done with normal subjects have shown that the self-paced version - the next stimulus being contingent on the subject's reaction to the preceding one - is the most sensitive to lapses of alertness (Broadbent, 1971; Schacter, 1977).

In the 70's, Van Zomeren (1981) and his colleagues undertook a series of RT experiments to investigate the slowing of information processing following closed head injuries. First, they reviewed the few RT studies which had previously been done with TBI subjects and pointed out their methodological shortcomings. For example, in the studies of Ruesch (1944a, b), the first to use RT tasks with this population of patients, the control subjects used were considerably younger (on average more than 10 years) than the TBI subjects. Also, the mildly concussed subjects of Dencker and Lofving (1958) were evaluated on average 10 years after their injury, suggesting that many of them may have recovered more or less completely by that time.

Despite these methodological differences, consistent findings of a prolonged simple RT confirmed clinical observations of significant slowing of mental processing in the TBI population. Furthermore, subsequent studies (Norrman & Svahn, 1961; Miller, 1970) showed a greater sensitivity for RT tasks which required a choice decision. The magnitude of the difference between control and TBI groups increased with level of task complexity (i.e., number of choices). Contradictory results, however, were obtained by Klensch (1973, cited by Van Zomeran, 1981). This author found that simple auditory and visual RT tasks discriminated between a mild TBI group and a control group, while an intermodal choice RT task did not. As stressed by Van Zomeran (1981), task requirements were quite different between the two tasks (simple and choice) and possibly resulted in different response strategies with the possibility that speed-accuracy tradeoffs (not reported in the study), might have masked group differences. In that regard, Winogron (1986) found that head injured and normal children process information in remarkably similar ways, despite some slowing of responding in the former group.

Given the questionable methodology and ambiguous results of earlier studies, Van Zomeran and Deelman (1976) undertook a series of well-controlled, systematically executed experiments to first address such issues as the differential effects of simple vs choice RT tasks and the effects of level of task complexity. Testing a group of TBI subjects (duration of initial coma at least half an hour; time since injury on average 4 months) and a matched control group, they found a disproportional effect between simple and choice (2 or 4 choices) visual RT tasks. Moreover, an interaction between complexity of the tasks and severity of the injury as indexed by coma duration was demonstrated - the greater the amount of

information processing overload, the slower the performance (severely injured subjects with a coma lasting over a week). This interaction was still observed 22 months after the injury. A subsequent re-analysis of the choice RT task data to control for motor requirements of each task revealed that the slowness exhibited by the TBI subjects was at least partially related to increased time for such central processes as decision-making and response selection.

To study the recovery effects for simple and choice RT tasks as a function of injury severity, Van Zomeren and Deelman (1978) repeatedly tested, over a 2 year period, 3 groups of young brain injured adult subjects classified as mild (initial coma less than 1 hour), moderate (coma of 1 hour to 1 week), and severe (coma over 1 week). Testing was done at 5 weeks, 10 weeks, and 5 months after trauma, and then in the middle of the next 3 successive half-years. Performance on both simple and choice RT tasks improved over time, although differently. Simple RT tasks stabilized within a year showing no significant differences between the 3 TBI groups, all of which remained slightly inferior to a control group, suggesting the presence of residual pathology even in mildly injured patients. Choice RT tasks, on the other hand, continued to improve over the 2 year period and to differentiate between grades of severity, showing a stronger relation than simple RT tasks with clinical variables and social outcome. In the last testing session (i.e., 2 year post-trauma), the mild and moderate groups showed reaction times on choice RT tasks equivalent to those of a control group, whereas the severe group remained significantly slower.

Following their series of RT experiments with closed head injured subjects, Van Zomeren and his colleagues (1984) attempted to explain the slowing of information

processing experienced by this population of patients within the context of information-processing theory, more specifically the two-process model of Shiffrin and Schneider (1977). Based on the dichotomy of pre-attentive and attentive processes originally proposed by Neisser (1967), Shiffrin and Schneider (1977) postulated the existence of two processes: automatic and controlled. The former involves practice or learning and is characterized as pre-attentive (without conscious control), with large capacity and usually simultaneous or parallel modes of processing; the latter, by comparison, requires conscious control (attentive), is limited in capacity and is typically serial in nature.

From this model which emphasizes attentional limitations, Van Zomeren and his colleagues (1984) derived two basic types of attentional deficits: focused attention deficits (FADs) and divided attention deficits (DADs). FADs would normally result from highly practiced, automatic response tendencies conflicting with response requirements of the task at hand. DADs, on the other hand, would result from a slowed rate of consciously controlled processing. This distinction between automatic and controlled processes resulting in focused attention and divided attention deficits is somewhat analogous to the last two attentional components proposed by Posner and Boies (1971) of selectivity and conscious processing.

Van Zomeren and his colleagues (1984) reviewed studies done with TBI subjects in order to provide evidence for these two types of deficits. No firm evidence could be gathered regarding deficits of focused attention (FAD) after brain injury. Earlier studies (Dencker & Lofving, 1958; Gronwall & Sampson, 1974) had used questionable methodologies to assess deficits in selectivity, whereas more recent and better controlled studies revealed opposite

results. Studies using the Stroop Color Word Test (Stroop, 1935) could not find any specific effect of response interference between controls and TBI children (Chadwick, 1976; cited by Van Zomeren, 1981) or adults (Thomas, 1977; cited by Van Zomeren, 1981). Two other studies, on the other hand, using RT tasks (Van Zomeren, 1981) and card sorting tasks (Miller & Cruzat, 1981) showed that distracting stimuli had a disproportionate effect on TBI subjects compared to normal subjects. The fact that both groups of subjects were affected, though to a different extent, suggests that the poorer performance of the TBI subjects was more likely due to slowing of controlled processing (i.e., deficits of divided attention) than to deficits of focused attention.

Mental slowing, as implied by the term divided attention deficits (DADs), has long been recognized as a major deficit following brain injury. Only recently, however, has this performance decrement been described in terms of information processing theory. As shown by Miller (1970), increasing the complexity of RT tasks (i.e., number of choices) results in more marked differences between control and TBI groups; this suggests that traumatic brain injury induces a slowing in both decision-making and response selection. Gronwall and Sampson (1974) obtained similar results using a paradigm in which stimulus and response were incompatible (i.e., subjects had to respond to stimuli numbered from right to left by pressing keys numbered from left to right). Furthermore, these same researchers elaborated a task, the Paced Auditory Serial Addition Task (PASAT), which was clearly aimed at producing DADs. The task consists of tape recorded one-digit numbers presented in series at different rates. Subjects are required to add each pair of successive numbers and answer immediately. Using controls and groups of concussed patients differing in the degree of

severity of their injury as measured by duration of posttraumatic amnesia (PTA), Gronwall and Sampson (1974) found that the TBI subjects were significantly more affected by changes in the rate of presentation of the digit series. Furthermore, an interaction between severity and rate of presentation was reported.

Similar deficits of divided attention were disclosed in a group of TBI subjects presumed to have made a good recovery (Stuss, Ely, Hugenholtz, Richard, LaRochelle, Poirier, & Bell, 1985). In that study two tests were shown to be most important for discriminating TBI and control groups: the Consonant Trigram Test (CCC), a test of memory under interference conditions, and the Wisconsin Card Sorting Test (WCST), a test of conceptualization and mental flexibility. The WCST has been known as a test of frontal lobe functions (Milner, 1963) but its specificity has been questioned recently (Mountain & Snow, 1993).

In sum, performance studies after closed head injury as it relates to attentional processes and information processing have shown deficits which in most cases are correlated with the severity of the insult, as measured by the coma criterion. The preliminary evidence regarding the issue of level of tonic alertness in TBI subjects points in the direction of higher rather than lower levels. No conclusive evidence yet exists regarding the presence of deficits in selectivity (or FADs) in closed head injured subjects. Substantial evidence, on the other hand, exists for the presence of deficits of divided attention resulting in a slowing of information processing. This slowing has been demonstrated particularly for central processes such as decision making and response selection, although more peripheral processes such as

perception and motor response may also be affected.

OBJECTIVES AND RESEARCH HYPOTHESES

Fatiguability is a common and often overlooked sequelae of traumatic brain injury (Lezak, 1978; Van Zomeren & Van Den Burg, 1985). Its nature, however, remains unclear. Studies which have focused on sleep-wake patterns tend to relate sleepiness to a reduced tonic level of arousal (i.e., a subwakefulness syndrome). Performance studies, on the other hand, suggest that TBI subjects tend to be overaroused. This hyperarousal, in turn, could possibly lead to exhaustion and subjective sleepiness. On the other hand, most sleep studies have been done with heterogeneous groups of brain injured people whose accident occurred less than 2 years previously.

The present study, therefore, aims at clarifying the nature of daytime sleepiness in severe TBI (closed head injured) young adults whose injury is over 2 years old. By that interval it may be reasonably expected that the neurological recovery has been completed and that most subjects have adjusted to their new condition. Two groups of TBI subjects, one complaining of daytime sleepiness and the other not, have been studied along with matched controls. Since sleepiness can result from different types of physiological dysfunction such as sleep deprivation and/or sleep satiation, circadian desynchronosis, selective pressure for REM sleep or NREM sleep, impaired arousal, and impaired sleep onset mechanisms (Broughton, 1982, 1992), a variety of measures (subjective and objective) were used. These included overnight sleep EEG recordings, the daytime Multiple Sleep Latency Test (MSLT), a simple visual reaction time (VRT) task, electrocardiogram (EKG), the Stanford Sleepiness Scale (SSS) (Hoddes, Dement, & Zarcone, 1972), and a neuropsychological test battery.

For the neuropsychological battery, the tests used were mainly those that were found sensitive to the pathophysiology of the diffuse impact of closed head injury resulting in frontal lobe and temporal lobe disturbances (Stuss et al., 1985). These consisted of measures of attention, processing of information, problem solving and motor tests. Inventories of personality changes were also completed.

Based upon the above literature review, it was predicted that:

1. Nocturnal sleep measures will significantly differ between the two TBI groups and the control group, mainly in terms of greater awakenings after sleep onset (sleep fragmentation) and more so for the sleepy TBI group.
2. Objective sleepiness as measured by the MSLT will also show a significant difference between the two TBI groups and the control group. Pathological levels (mean latency for sleep onset less than 5 minutes) will be present in the TBI group complaining of sleepiness; less marked (and not in the pathological range) for the TBI group not reporting sleepiness; and absent for the control group. Moreover, time of day effects, specifically maximum sleepiness in mid-afternoon (i.e., post-lunch dip), will be present in all groups.
3. Significant relationships will be present between MSLT measures (mean latencies) and prior nocturnal sleep especially for wake after sleep onset for the TBI groups. Shorter daytime sleep onset would be related to greater nighttime awakening after

sleep onset.

4. Subjective sleepiness as measured by the Stanford Sleepiness Scale will be related to objective sleepiness as measured by the MSLT.
5. On the simple visual reaction time task, both groups of TBI subjects will be significantly slower than the control group. Furthermore, performance over time ("fatiguability") of the group of TBI subjects reporting sleepiness will be worse than in the non-reporting group.
6. MSLT naps will provide recuperative value for performance on the simple VRT task for the TBI subjects reporting sleepiness.
7. A measure of physiological arousal (i.e., heart rate) taken during performance of the simple reaction time task will show significantly higher frequencies for the TBI subjects compared to control subjects.
8. Both groups of TBI subjects will be significantly impaired on neuropsychological testing compared to the control group, especially on the tests of attention, concentration, speed of information processing, and executive tests. Furthermore, neuropsychological data of TBI subjects will show greater inter-subject variability.

METHOD

Subjects

Three groups each of 8 subjects matched for age and educational background were studied. Except for one female and two left handed subjects, all were right handed males. Two groups consisted of severe TBI adults (coma greater than a day and post-traumatic amnesia greater than a week) whose accident occurred at least 2 years before testing. The two groups differed only in regards to their subjective level of daytime sleepiness as assessed by a sleep questionnaire (Appendix A). Three items from that questionnaire were selected to split the group of TBI subjects in two based on their subjective level of daytime sleepiness (Table 1). These questions were straightforward and easy to answer given the judgement

Table 1. Subjective Sleepiness Items

Day time

Do you feel sleepy during the day?

rarely	occasionally	frequently	always
(1)	(2)	(3)	(4)

Do you nap on weekdays?

rarely	occasionally	frequently	always
(2)	(4)	(6)	(8)

Do you nap on weekends (or holidays)?

rarely	occasionally	frequently	always
(1)	(2)	(3)	(4)

problem commonly seen in this population of subjects. The TBI group with a high level of daytime sleepiness (Total of the three items greater than the median 6.5) was called sleepy TBI; and the one with a low level (total of the three items smaller than the median 6.5) was called nonsleepy TBI. The classification results are presented in Table 2.

The TBI subjects were recruited through the Ottawa Head Injury Association and the Société de l'assurance automobile du Québec (provincially owned auto insurance company of Québec). The third group consisted of normal matched controls with no history of neurologic or psychiatric disorders.

Excluded were TBI subjects who were not independent for activities of daily living, had other neurological or psychiatric conditions, or whose current medication was known to affect sleep-wake patterns (e.g., sedative and stimulant medications). Other life habits were also documented, such as smoking and coffee drinking. This information was obtained from an interview with both the subjects and their family as well as from medical records. A screening polysomnographic recording was also performed to exclude subjects showing significant sleep apnea (apnea index over 5 per hour or apnea plus hypopnea index over 10 per hour) or periodic leg movements (over 10% of total sleep time). Because of their prevalence such sleep disorders could have been present before the accident and actually been responsible for the accident (Aldrich, 1989). All subjects were informed about the nature and purpose of the experiment (Appendix B) and were paid for their participation. Signed informed consent was obtained.

Table 2. Subjective Sleepiness Scores

Subject	Sleepy	Weekdays	Weekends	Total
Sleepy TBI				
CC	2	4	2	8
KD	3	4	2	9
LD	4	6	4	14
JJ	1	4	2	7
GL	3	6	2	11
JM	4	2	1	7
PM	2	4	2	8
SP	4	2	1	7
Nonsleepy TBI				
MA	2	2	1	5
JC	3	2	1	6
KC	2	2	1	5
RL	1	2	1	4
GM	1	4	1	6
SM	1	2	2	5
MH	1	2	1	4
MT	1	2	1	4
Controls				
AB	1	2	2	5
KB	2	2	2	6
MD	1	2	1	4
RH	2	2	2	6
JL	1	2	1	4
PL	2	2	2	6
DS	2	2	2	6
MO	1	2	1	4

Procedure

Subjects were studied individually by laboratory evaluations involving electrophysiological recordings and performance measurements (Figure 1). They subsequently completed the neuropsychological test battery.

1st STAGE

2nd STAGE

NEUROPSYCHOLOGICAL EVALUATION

Figure 1. Physiological and Performance Protocol

Nocturnal and daytime recording

On day 1 of the experiment, subjects came to the sleep laboratory in the evening for the screening polysomnographic recording. This first night was also an adaptation night (Agnew, Webb, & Williams, 1966; Browman & Cartwright, 1980). Since they were all awakened at 0700 hour (hr), subjects came early enough so they could benefit from their

usual amount of sleep. Following this first night, subjects returned home for the day and came back to the sleep laboratory the next evening.

On day 2, all subjects underwent a second nocturnal polygraphic recording. Following their awakening at 0700 hr, subjects remained in the sleep laboratory and daytime polygraphic recording was performed at regular intervals during the MSLT. Starting at 0900 hr and every 2 hours thereafter until 1700 hr, subjects were asked to fall asleep as quickly as possible. Nap time was restricted within a 20 minute recording period.

Fifteen minutes before and 5 minutes after each scheduled nap period, subjects performed a simple visual reaction time task (Glenville, Broughton, Wing, & Wilkinson, 1978) for 10 minutes. After the last nap period ending at 1720 h, subjects also performed the simple visual reaction time task 35 and 65 minutes post-nap to assess the duration of any recuperative period.

Heart rate was recorded to determine the level and temporal organization of physiological arousal during the VRT task performance.

Throughout the day, subjects also completed the Stanford Sleepiness Scale (SSS) (Hoddes, Zarcone, Smythe, Phillips, & Dement, 1973) every 1/2 hour, as well as before and after each nap period. The SSS consists of a 7-point scale varying from 1 (maximum alertness) through 7 (maximum sleepiness).

During internap periods, subjects were free to occupy themselves as they wanted (e.g., reading) but were prohibited from napping and were continuously observed to ensure that this did not occur. They could move around in the sleep laboratory but could not leave the facilities. Breakfast and a light lunch were provided.

Neuropsychological assessment

Subsequent to the sleep laboratory studies, each subject was administered the neuropsychological test battery to measure particular variables known to affect reaction time performance and sleep (e.g., attentional deficits, depression). Testing lasted half a day and was uniformly distributed among the three groups in terms of morning and afternoon sessions.

Apparati

Electrophysiological recordings

Polygraphic recordings were made at a paper speed of 15 mm/sec, and calibrated at 100 μ V/cm for electroencephalograms (EEG) and electromyograms (EMG), and at 500 μ V/cm for electrooculograms (EOG) using a Nihon Kohden Model apparatus for the first night and a Grass Model apparatus for the second night. Gold cup 8 mm electrodes were used. Electrode impedance was maintained below 5 KOhms. The following variables were monitored using the 10-20 international placement system (Jasper, 1958):

EEG - C4-A1 and O1-A2

EOG - Right eye-A2 and Left eye-A2

EMG - submental

ECG - precordial

Respiration - nasal and oral airflow; abdominal and thoracic movements

EMG - anterior tibialis (both legs)

The last two variables (respiration and leg movements) were monitored only for the first night to rule out sleep apnea and periodic leg movements.

The second night was followed by a Multiple Sleep Latency Test which comprised the same electrophysiological variables as per the previous night. If sleep occurred during one of the five 20-minute scheduled naps, subjects were awakened 10 minutes following sleep onset, as defined by 2 consecutive 40 second epochs of stage 1 or REM (Carskadon, Dement, Mitter, Roth, Westbrook, & Keenan, 1986).

Performance tests

A simple visual reaction time task was performed for which subjects sat in a straight back chair at a desk located in the room where they slept. The task consisted of a compact digital millisecond clock connected to a laptop computer. The stimulus was the onset of the clock and the response was a key press stopping the clock. Stimulus onset of the next series of numbers occurred randomly between 2 and 10 seconds after the extinction of the previous

displayed response. The dependent measure was the time (ms) between the onset of the stimulus and the response. This visual task has been shown to be useful for assessing vigilance fluctuations because of its sensitivity and its relative immunity to practice effects (Herscovitch & Broughton, 1981).

For the neuropsychological assessment, the following tests, which are commonly used clinically, were administered:

I. Cognitive domain

A. Intelligence

- Wechsler Adult Intelligence Scale - Revised (WAIS-R) (Wechsler, 1944, 1981)

This is a test of intelligence based on 11 subtests, 6 verbal and 5 nonverbal. The dependent measures are scaled scores for the subtests from which are calculated 3 intellectual quotients (Verbal, Performance, and Full Scale).

For the nonverbal subtests, timed and untimed scores were obtained.

B. Attention, Information processing

- **Digit Span (DS) (Wechsler, 1981)**

This is one of the subtests of the WAIS-R which consists in repeating a sequence of single-digit numbers read aloud by the examiner. There are two conditions. In the first one, digits must be repeated in the same order (digits forward); and in the second one, they are repeated in the reverse order (digits backward). For both conditions, the length of the sequences increases progressively and two trials are presented at each sequence length. The dependent measure is the length of the longest sequence completed successfully for each condition.

- **Mental Control (MC) (from Wechsler Memory Scale-Revised; WMS-R) (Wechsler, 1945, 1987)**

This subtest from the WMS-R consists of automatisms (e.g., counting backward and reciting the alphabet) and simple mental calculation. The dependent measure is the sum of the 3 items.

- **Trail Making Tests (TMT) A & B (Army Individual Test Battery, 1944; Reitan, 1958)**

These are visuomotor tracking tasks measuring attention, cognitive sequencing, and psychomotor speed. The first test (A) consists in connecting as quickly as possible, in numerical order, a series of randomly arranged circles each containing a different number from 1 to 25; and the second one (B) consists in connecting as quickly as possible, a series of randomly arranged circles, alternating between sequential numbers (1 to 13) and letters (A to L). The dependent measure is the time required for completion of each test.

- Paced Auditory Serial Addition Task (PASAT) (Gronwall & Wrightson, 1974; Gronwall, 1977)

This is a test of attention and speed of information processing. It consists of the serial presentation (via a tape recorder) of four sets of randomized digits between 1 and 9. For each set, sixty numbers are presented at a constant presentation rate which increases with each set (one per 2.4, 2.0, 1.6 and 1.2 second). Subjects must add the number they currently hear to the number which preceded and respond with the sum. Therefore, for each number presented, a new sum is given. The dependent measure is the number of correct sums for each set.

- Consonant Trigram Test (CCC) (Brown, 1958; Peterson & Peterson, 1959)

This test measures auditory short term memory for three consonants under interference conditions (counting backward from a given number for a set period of time). The delay between the stimulus presentation and recall varies between 3, 9 and 18 seconds. The dependent measure is the number of correctly recalled consonants for each interval of time (3, 9 and 18 seconds).

- Stroop Color Word test (Stroop, 1935; Nehemkis & Lewinsohn, 1972)

This test consists of three subtests (words, colors, and color-word interference): words - reading printed color names (green, red, and blue); colors - naming colored blocks (using the same three colors); and color-word interference - naming the colors of words that are themselves printed in a different color (again using the same three colors). The dependent measure for all three tasks is the time (seconds) taken for completion.

C. Executive functions

- Wisconsin Card Sorting Test (WCST) (Berg, 1948; Robinson, Heaton, Lehman, & Stilson, 1980)

This test measures the ability to deduct, sort according to the category deducted and change the sorting categories using the verbal feedback (right

or wrong) provided by the examiner. The dependent measures include the number of correct categories, of correct choices and of errors (perseverative and nonperseverative).

D. Motor functions

- **Grooved Pegboard (GP) (Klove, 1963; Matthews & Klove, 1964)**

This is a test of psychomotor coordination and speed, requiring the subject to place pegs into holes. The 25 small metal pegs have a ridge along their side and therefore must be rotated to match the randomly positioned slot of the holes. The pegs must be placed in the holes as quickly as possible, first with the right hand, then the left hand. The dependent measure is the time (seconds) required for completion using each hand.

- **Finger Tapping (FT) (Halstead, 1947; Reitan & Davison, 1974)**

This is a test of simple motor speed which requires one to tap on a key counter for 10 seconds using the index finger of the right hand and then the left hand. The dependent measure is the average of the five trials for each hand.

- **Grip Strength (GS) (Halstead, 1947; Reitan & Davison, 1974)**

This is a test of motor strength using a hand dynamometer. Three trials are administered alternately to each hand. The dependent measure is the average strength (in kilograms) of the three trials for each hand.

II. Non-cognitive domain

- IPAT anxiety scale questionnaire (Cattell, Scheier, & Madge, 1968)

This test consists of a 40 item instrument designed to assess the level of anxiety in adolescents and adults. Each item is scored either 1 or 2. The dependent measure is a single total anxiety score based on all 40 items.

- Beck depression inventory (Beck, Ward, Mendelson, Mock, & Erbaugh, 1961; Beck, Rush, Shaw, & Emery, 1979).

This is a measure of the severity of depression in adolescents and adults. It consists of 21 items on symptoms and attitudes which are rated on a 4-point scale (range 0 to 3) of severity. The dependent measure is the sum of the ratings given for each of the items.

- Katz Adjustment Scale (KAR) (Katz & Lyerly, 1963)

The Katz Adjustment Scale - Relative's form (KAR-R) is a measure

of psychosocial adjustment that consists of different forms: R1 has 55 items and provides for 13 dimensions relevant to symptom discomfort (somatic, mood, and psychoneurotic symptoms), R2 measures general activities, and R4 measures free-time activities. A close relative of the subject was requested to complete the scale.

The KAS-R has been used in brain injured populations and has been found sensitive to adjustment difficulties (Klonoff, Costa, & Snow, 1986; Goodman, Ball, & Peck, 1988).

Data Analyses

Sleep Variables

Night time

Sleep staging was done visually based on the Rechtschaffen and Kales (1968) criteria. Sleep variables included: time in bed, total sleep period, total sleep time, sleep efficiency index (percentage of time spent actually sleeping); latencies for sleep onset, stage 2, SWS (stages 3 & 4), and REM; duration of wakefulness after sleep onset, stages 1 and 2, SWS and REM, and movement time; and number of stage shifts per hour. Wakefulness was scored as active or quiet following criteria of Volk and his colleagues (1984) and sleep stage 1 was

subdivided into subtypes 1A and 1B according to Valley and Broughton (1983). Substage 1A consists of diffusion and slowing of alpha rhythm, and substage 1B consists of mixed frequency medium voltage patterns identical to stage 1 of Rechtschaffen and Kales (1968).

Each dependent measure was treated separately and tests for significance were performed with one-way analyses of variance (ANOVAs). Post-hoc comparisons were made using Tukey's honestly significant difference (HSD) Test (Kirk, 1982). All analyses were performed using SPSS (1990).

Multiple Sleep Latency Test

For each nap, the variables included: sleep latencies to stage 1 and to stage 2 or RFM.

Two factors (groups and MSLT sessions) formed a 3 X 5 matrix with repeated measures taken on the MSLT nap sessions. ANOVA was applied to the two-way mixed model design. Post-hoc comparisons were performed; and for main effects and interactions involving repeated measures factors, significance levels were determined using the more conservative, adjusted Geisser-Greenhouse method (Kirk, 1982).

Arousal Measures

Visual Reaction Time task

For each 10 minute trial, the mean and standard deviation were calculated.

The ANOVA consisted of a factorial model (3 X 5 X 2) with repeated measures on the pre- and post-MSLT trials for each MSLT nap sessions. As for the MSLT, post-hoc comparisons were performed.

Heart rate

Heart rate data consisted of the mean and standard deviation for each pre-trial (eye closed condition for 2 minutes) and pre- and post-MSLT trials (first and last 2 minutes of eyes opened condition for each trial) for each MSLT nap sessions.

The ANOVA model was similar to the one for the VRT task, but with one more measure for each MSLT nap session, the pre-trial (eyes closed condition). It consisted of a 3 X 5 X 3 level factorial. Post-hoc comparisons were also performed.

Stanford Sleepiness Scale

Results across the day were averaged for members of each group and these data were subjected to the Kruskal-Wallis nonparametric analysis of variance in a similar fashion to that of sleep variables. Post-hoc comparisons for group differences were made using Tukey's method adapted for the ranks (Marascuilo & McSweeney, 1977).

Neuropsychological assessment

For each test, means and standard deviations were calculated. As for sleep variables, each dependent measure was treated independently and post-hoc omnibus tests for significance were performed with one-way ANOVAs.

Finally, relationships between the different data sets (clinical, sleep, arousal, and neuropsychological variables) were examined with simple bivariate regression. Individual differences were stressed.

RESULTS

The major demographic characteristics of each group, and clinical characteristics of the TBI groups, are outlined in Table 3. Except for one TBI subject (GM), all were male. The mean (M) age of TBI subjects was 29.6 and standard deviation (SD) 6.6 and of controls M 27.5 and SD 6.2. The mean number of years of education was 12.4 (SD 2.7) and 13.4 (SD 1.4), respectively. Neither of these variables were significantly different among the three groups. All but one TBI subject (SP) had done high school studies. At the time of their accident, more than half (10) of the TBI subjects were working and the remaining (6) were studying. Following their accident, less than half (6) had returned to a productive life but at a lower level of functioning (e.g., unskilled or part-time jobs); one resumed his studies but at a slower pace; four were unemployed and five were considered disabled. Most were thus unable to resume their previous activities.

For the TBI subjects, the interval between injury and testing ranged from 3 to 16 years (M 6.3; SD 3.3). The severity of the TBI is normally characterized by the following clinical criteria: coma depth (Glasgow Coma Scale (GCS), which measures visual, verbal, and motor responsiveness (Teasdale & Jennett, 1974)) and duration, and posttraumatic amnesia period (PTA; Russell, 1971). All had significant periods of coma lasting from 1 to 84 days (M 23.7; SD 24). The duration of coma, however, was recorded in an unsystematic manner. Except for the diffuseness of their brain injury (which for all subjects resulted from a motor vehicle accident), the type of CNS injury varied, some having sustained hematoma, others contusions or both. The common neurodiagnostic tool was computed tomography (CT) which was used

Table 3. Demographic and Clinical Data

Subject	Sex	Age	Education	Chronicity (years)	Coma (days)	Hand (dominant)	Occupation	
							Before	After
Sleepy TBI								
CC	M	43.8	18	7.5	14	R	Officer	Disabled
KD	M	24.9	15	6.1	2	R	Student	Student
LD	M	27.3	14	7.8	7	R	Student	Clerk
JJ	M	38.4	14	16.3	56	R	Teacher	Clerk
GL	M	21.7	11	4.1	1	R	Student	Unemployed
JM	M	24.2	12	5.2	14	R	Student	Labourer
PM	M	25.3	11	3.0	10	L	Manager	Disabled
SP	M	33.4	5	7.2	35	R	Labourer	Disabled
$\bar{X}$		29.9	12.5	7.2	17.4			
Nonsleepy TBI								
MA	M	21.1	12	2.8	6	R	Student	Unemployed
JC	M	32.0	12	4.8	1	R	Technician	Labourer
KC	M	39.0	12	3.0	49	R	Manager	Unemployed
RL	M	24.8	12	4.9	21	R	Labourer	Clerk
GM	F	28.8	14	7.3	35	R	Supervisor	Counselor
SM	M	28.9	12	6.3	84	R	Student	Disabled
MH	M	27.3	12	8.5	21	R	Technician	Unemployed
MT	M	33.3	12	10.9	3	R	Labourer	Disabled
$\bar{X}$		29.4	12.3	6.1	27.5			
Controls								
AB	M	22.7	14			R	Student	
KB	M	31.6	14			L	Technician	
MD	M	25.9	12			R	Manager	
RH	M	22.9	14			R	Student	
JL	M	28.0	13			R	Secretary	
PL	M	40.3	12			R	Clerk	
DS	M	27.2	16			R	Student	
MO	M	21.3	12			R	Baker	
$\bar{X}$		27.5	13.4					

Note: no significant difference between the groups for all variables

only in the acute phase for most of the TBI subjects. No systematic pattern of brain injuries was found comparing sleepy and nonsleepy TBI subjects. Furthermore, the incidence of neurosurgical procedures, neuroradiological investigations and pharmacological treatments was comparable between the two TBI groups. Overall, these two groups of TBI subjects were homogeneous in most respects other than in their difference on subjective daytime sleepiness.

In summary, the majority of TBI subjects in this study were single, young adult males with a high-school education. Motor vehicle accidents were the cause of the injury for all of them. This profile is typical of the population of TBI victims normally encountered, which are predominantly young men involved in motor vehicle accidents.

Qualitative and quantitative information was also gathered regarding their sleeping habits throughout the 24 hour day (Table 4). Although all TBI subjects reported changes in their sleep habits (e.g., in bed earlier, not enough sleep, sleep more), most (except for two) were satisfied with the quality of their sleep. Except for the sleepiness factor during the day (i.e., high vs low napping), the two TBI groups were comparable as for their present sleeping habits. When compared with the controls, many TBI subjects reported difficulties falling asleep at night.

Table 4. Sleep Questionnaire Data

Subject	Hours of sleep per night	Falling asleep Problems	Avg. time	Refreshed upon awakening	Quality of sleep satisfying	Changes in sleep habits	Comments
Sleepy TBI							
CC	8-9	rarely	15 min.	always	yes	yes	tires easily, in bed earlier
KD	7-8	rarely	1-3 min.	occasionally	yes	yes	sleep less
LD	8	frequently	15-20 min.	occasionally	yes	yes	changed sleep need
JJ	9.5	rarely	15 min.	always	yes	yes	in bed earlier
GL	9	occasionally	10 min.	occasionally	yes	yes	more regular sleep
JM	6	always		occasionally	yes	yes	
PM	7-8	occasionally	30 min.	frequently	yes	yes	in bed earlier
SP	6	always	30-45 min.	frequently	yes	yes	sleep less
Nonsleepy TBI							
MA	7-8	occasionally	1-30 min.	rarely	no	yes	feel like not enough sleep
JC	9	occasionally	60 min.	occasionally	yes/no	yes	sleep more
KC	8-9	rarely	15 min.	always	yes	yes	urinate more
RL	7-8	rarely	15 min.	always	yes	yes	need more sleep
GM	7-8	rarely	15-20 min.	frequently	yes	yes	need more sleep
SM	7-8	always	60 min.	always	yes	yes	
MH	7-8	frequently	120 min.	occasionally	yes	yes	more difficult to sleep
MT	7-8	frequently	120 min.	occasionally	yes	yes	more anxious
Controls							
AB	7	rarely	20-30 min.	frequently	yes		
KB	5-6	rarely	15 min.	frequently	yes		
MD	7	rarely	15 min.	occasionally	yes		
RH	7-8	occasionally	15 min.	occasionally	yes		
JL	7-8	rarely	5 min.	frequently	yes		
PL	8	rarely	15-20 min.	always	yes		
DS	7-8	rarely	5 min.	frequently	yes		
MO	9-10	rarely	10-15 min.	occasionally	yes		

Nocturnal Sleep Structure

The records of the first night were visually analyzed by a qualified polysomnographic technician to rule out sleep apnea and periodic leg movements. Two subjects, one TBI and one control, were excluded because of sleep apnea and leg myoclonus, respectively. Both were referred to the sleep clinic and replaced by other subjects. Therefore, all subjects included in the study showed normal breathing and leg movements patterns during sleep.

The records of the second night and of the MSLT were scored by three experienced raters. Interjudge reliability was 84.4% for overnight recordings and 90.7% for MSLT. The normal criteria for analyzing sleep stages (e.g. eye movements, sleep spindles, K-complexes) were easy to apply to TBI records, suggesting normalcy of sleep components.

Examination of the night recordings showed that few subjects had to be awakened in the morning. Sleepy TBI subjects tended to awaken earlier than nonsleepy TBI and control subjects. Thus, 6 out of 8 sleepy TBI awakened spontaneously before the planned awakening time at 0700 hr compared to 2 out of 8 for the nonsleepy TBI group. All the controls were still sleeping at 0700 hr. Many of the night sleep architectural measures showed greater variability for the sleepy TBI subjects compared to the nonsleepy TBI and control subjects (Table 5).

Table 5. Sleep Variables

	Sleepy TBI		Nonsleepy TBI		Controls		F(2,21)	P
	M	SD	M	SD	M	SD		
Night sleep								
TIBED (min.)	426.3	63.8	437.0	42.7	469.1	27.1	1.79	ns
TSP	382.3 ^a	80.4	421.5	38.4	458.1 ^a	29.2	3.93	< .05
TST	336.3 ^a	107.5	382.6	46.4	443.5 ^a	39.2	4.55	< .05
SEFF (%)	87.5	17.4	90.7	6.7	96.7	4.2	1.44	ns
S1 lat. (min.)	32.0 ^a	24.9	14.0	13.5	8.0 ^a	4.3	4.59	< .05
S2 lat.	3.0	2.7	5.1	4.8	3.2	3.3	0.76	ns
SWS lat.	11.0	10.1	24.0	20.1	15.4	8.2	1.83	ns
REM lat	72.6	37.8	98.4	36.7	77.8	39.0	1.03	ns
WASO (min.)	46.0	64.7	38.9	27.4	14.6	17.2	1.25	ns
S1	40.2	34.3	43.4	36.0	27.0	10.1	0.71	ns
S2	137.8 ^{ab}	55.1	196.8 ^b	52.7	229.6 ^a	23.9	8.12	< .01
SWS	78.5	30.6	74.4	24.5	95.3	33.0	1.12	ns
REM	79.8	40.2	68.0	26.2	91.6	27.5	1.09	ns
MT	1.8	2.2	2.5	2.9	1.0	0.8	0.84	ns
AWAKENINGS	6.0	5.3	5.1	3.6	2.1	3.6	1.74	ns
SH/HR	25.9	7.3	19.8	5.5	23.2	8.7	1.42	ns
MSLT								
S1 lat. (min.)	15.1	4.0	12.8	4.2	10.6	4.4	2.23	ns
S2/REM lat.	17.3	3.4	17.8	2.5	15.1	2.5	2.12	ns

TIBED, time in bed; TSP, total sleep period; TST, total sleep time; SEFF, sleep efficiency; S1 lat., sleep onset latency; S2 lat., stage 2 latency; SWS lat., slow wave sleep latency; REM lat, rapid eye movement latency; WASO, wake after sleep onset; MT, movement time; AWAKENINGS, no of awakenings after sleep onset longer than 1 min.; SH/HR, stage shifts per hour.

Note: The superscripts indicate groups which differ significantly by the Tukey HSD multiple comparison procedure ($p < .05$). For example, S2 for sleepy TBI differed significantly from S2 for both nonsleepy TBI and controls.

For the statistical analyses, the Bonferroni *t* procedure (Miller, 1966) was applied to control for type I errors given the large number of variables analyzed. There were over 50 ANOVAs performed on the data set and thus the adjusted and more conservative level of significance for each test was decreased from 0.05 to 0.001. The marginal level was set at $p < .05$ for interesting differences. The relatively small number of subjects per group ($N=8$), which is not uncommon for sleep studies, and the marked variability of the data mainly for TBI subjects, compromised the power of the statistical tests and thus made the statistical comparisons even more conservative.

In night sleep, there was a marked difference in total sleep time (TST) between sleepy TBI subjects and controls ($F(2,21) = 4.55, p < .05$) (Figure 2). The controls slept on average 7.6 hours (458 min.), whereas nonsleepy TBI slept 7 hours (422 min.) and sleepy TBI subjects, 6.4 hours (382 min.). This shorter sleep time was mainly related to a shorter duration of stage 2 (S2) ($F(2,21) = 8.12, p < .01$). Sleepy TBI subjects had less S2 than both nonsleepy TBI and control subjects. The duration of stage 2 represented 50% (230 min.) of total sleep period (TSP) for controls, 47% (197 minutes) for nonsleepy TBI, and only 36% (138 minutes) for sleepy TBI subjects. Wake after sleep onset (WASO) was also more pronounced, in both TBI groups (sleepy, M 46 min. and nonsleepy, M 39 min.) compared to controls (M 15 min.) as well as the number of awakenings (sleepy TBI, M 6.0, nonsleepy TBI, M 5.1, and controls, M 2.1). Thus, both TBI groups showed greater arousal at night. This was reflected in the sleep efficiency index which was lower in TBI subjects (sleepy 87.5%; nonsleepy 90.7%) compared to controls (96.7%), suggesting somewhat less sleep

time for the TBI subjects. The other sleep stages (S1, SWS and REM) were comparable among the three groups.

Figure 2. Night Sleep Variables

Sleep onset latency (SOL) revealed an interesting difference between sleepy TBI subjects and controls ($F(2,21) = 4.59, p < .05$) (Figure 3). On average, controls took less than 10 minutes to fall asleep, whereas sleepy TBI subjects took more than 30 minutes. Moreover, there was greater variability in the TBI groups as compared to the controls, and this was more evident for the sleepy TBI subjects.

In young adults, SOL is in the region of 0-15 minutes and constant on a night by night basis (Carskadon, Mitler, Billiard, Phillips & Dement, 1975; Birrell, 1983). In the present study, both nonsleepy TBI and control subjects showed on average a normal SOL.

Figure 3. Night Sleep Latency (stage 1)

In short, night sleep studies revealed that sleepy TBI subjects had longer sleep latencies and shorter sleep times.

The sleep architecture during the second night for a control subject is shown in Figure 4. The histogram shows regular sleep cycles with smooth transition from SWS to REM sleep.

Figure 4. Polysomnogram of a Control Subject

In Figure 5, a typical polysomnogram of one of the nonsleepy TBI subjects is presented. Although sleep onset remains relatively the same as controls, sleep is more fragmented and divided into several shorter sleep periods with awakening periods.

Figure 5. Polysomnogram of a Nonsleepy TBI Subject

Figure 6 shows a typical sleep histogram for a sleepy TBI subject. There is little resemblance to the regular sleep cycles which exist in a normal subject. The figure illustrates the longer sleep onset, the greater fragmentation of the sleep content with much less stage 2 and more frequent and longer awakening periods. Thus, the polysomnogram reflects these subjects' trouble both in falling asleep (sleep onset insomnia) and in staying asleep (sleep maintenance insomnia).

Figure 6. Polysomnogram of a Sleepy TBI Subject

Multiple Sleep Latency Test

Mean latency to stage 1 on MSLT revealed no significant difference among the three groups, and more specifically no pathological sleepiness (SOL shorter than 5 minutes) for the TBI groups (Figure 7). On the contrary, TBI subjects experienced problems falling asleep when compared to controls. The average latency on the MSLT for the sleepy TBI group was 15 minutes, nonsleepy TBI 13 minutes, and controls 11 minutes. None of the subjects presented sleep onset REM periods during MSLT naps.

Figure 7. MSLT - Mean Latency (stage 1)

The temporal pattern of sleep latencies on individual naps was rather different

between the sleepy TBI group and the other groups (nonsleepy TBI and controls). Interestingly, the mid-afternoon dip effect, which reflects a facilitation of sleep onset mechanisms (Broughton, 1975), was more prominent in controls than in TBI subjects.

Similar results were obtained for mean MSLT latency to stage 2 (Figure 8). No significant difference was found between the groups and on average TBI subjects took somewhat longer to reach deeper levels of sleep, 17 minutes for sleepy TBI group, 18 min. for nonsleepy TBI and 15 min. for controls. Again, the mid-afternoon dip effect was most evident in controls and absent in TBI groups.

Figure 8. MSLT - Mean Latency (stage 2)

Arousal Measures

Three different arousal measures were taken (one behavioral, one autonomic and one subjective) to evaluate the issue of sleepiness (Table 6). Whereas the RT measure showed a group effect (both TBI groups being slower than the controls), no such difference was found for either the autonomic (HR) or subjective sleepiness (SSS) measures.

Table 6. Arousal Measures

	Sleepy TBI		Nonsleepy TBI		Controls		F(2,21)	P
	M	SD	M	SD	M	SD		
<u>VRT</u>								
Pre-MSLT (ms)	346.8 ^a	91.9	341.4 ^b	47.4	238.9 ^{ab}	15.7	8.11	< .01
Post-MSLT	363.4 ^a	99.0	356.2 ^b	59.7	243.9 ^{ab}	18.2	7.87	< .01
<u>HR</u>								
EC (beats/m)	69.1	3.7	70.1	8.6	64.3	9.1	1.36	ns
Pre-MSLT	68.3	3.9	70.6	8.0	65.7	8.3	1.00	ns
Post-MSLT	66.8	4.0	69.0	7.4	65.1	8.5	0.64	ns
<u>SSS</u>								
Daytime	2.3	1.0	2.7	1.0	1.8	0.6	1.15	ns

VRT, visual reaction time; HR, heart rate; SSS, Stanford sleepiness scale

Note: The superscripts indicate groups which differ significantly from the control condition by the Tukey HSD multiple comparison procedure ($p < .05$). For example, Pre-MSLT VRT for controls differed significantly from Pre-MSLT VRT for both sleepy and nonsleepy TBI.

Visual Reaction Time

Figure 9 illustrates mean RT in 10 min blocks before each MSLT and an hour after the last MSLT to measure for the recuperative effect.

As expected, TBI groups had significantly longer RT compared to controls ($F(2,21) = 8.11, p < .01$). No significant difference, however, was found between sleepy and nonsleepy TBI groups. Moreover, the performance of both groups seems to deteriorate similarly by the end of the day compared to controls, as shown by a slightly upward direction in the slopes.

Figure 9. Visual Reaction Time

Sleep Inertia on VRT

Sleep inertia consists of a post-sleep decrement in performance observed upon awakening from night sleep (Lubin, Hord, Tracy, & Johnson, 1976) and daytime naps (Dinges, Orne, Evans, & Orne, 1981). It typically lasts from 1 to 15 minutes (Wilkinson & Stretton, 1971; Webb & Agnew, 1974), but may persist much longer.

All groups showed sleep inertia as evidenced by longer RT shortly after awakening (pre/post ratio below 1) (Figure 10). Sleep inertia was more pronounced in the afternoon for all groups. In that regard, it is interesting to note that the time of day effect was more obvious for sleep inertia than for sleep latency on MSLT, especially for TBI groups.

Figure 10. Sleep inertia on VRT (pre/post MSLT ratios)

Heart Rate

The HR measures taken before (eyes closed condition) and during (eyes open) the VRT task were compared. No group and time effects were found, suggesting no significant difference in the level of autonomic arousal among the three groups (Figure 11). Compared to the Van Zomeren study (1981) which had found higher heart rates during the last part of a 30 minute task in his TBI population, the present task lasted only 10 minutes. Interestingly, sleepy TBI showed less variability on HR measures than nonsleepy TBI and controls.

Figure 11. Heart Rate

Stanford Sleepiness Scale

Similarly, no significant difference was found between the groups as to their subjective level of sleepiness across the day (Figure 12). All felt alert which was in parallel with their higher sleep latency on the MSLT. Greater variability was again found in the data of the TBI groups.

Figure 12. Stanford Sleepiness Scale

Neuropsychological Results

For the neuropsychological testing, the data of one nonsleepy TBI subject are missing. He was unavailable for the neuropsychological evaluation since he had left the region.

The neuropsychological measures revealed more striking differences between the groups than the sleep measures (Table 7). For most tests, the greatest differences were between the sleepy TBI and control groups. Four measures, however, distinguished the controls from both TBI groups. These were the Digit Span, Digit Symbol, Consonant Trigrams (18 sec.), and Finger Tapping (right hand). On the other hand, sleepy TBI showed greater deficits than the nonsleepy TBI and control groups on three tests (PIQ, TMTA, PASAT).

Although TBI subjects showed significant cognitive deficits, they denied experiencing anxiety or depression.

Overall, these results suggest that sleepy TBI seem to have sustained greater brain impairment than nonsleepy TBI. Furthermore, as for the sleep variables, there was a gradient effect present for many measures with the sleepy TBI and controls at both extremities and the nonsleepy TBI in between.

Table 7. Neuropsychological Results

	Sleepy TBI		Nonsleepy TBI		Controls		F(2,20)	P
	M	SD	M	SD	M	SD		
<u>Intelligence</u>								
FSIQ	86.9 ^a	10.8	97.9	11.2	105.1 ^a	5.2	7.71	< .01
VIQ	88.3 ^a	11.2	94.1	9.9	101.6 ^a	5.1	4.39	< .05
PIQ	87.3 ^{ab}	11.5	103.7 ^b	11.5	109.6 ^a	7.3	10.18	< .001
Verbal subtests								
Information	8.3	2.7	9.1	3.1	11.0	1.7	2.48	ns
Digit span	7.1 ^a	1.6	8.7 ^b	2.5	11.5 ^{ab}	1.1	12.57	< .001
Arithmetic	8.6	2.7	11.0	2.6	10.5	1.6	2.20	ns
Comprehension	8.0	2.7	8.1	1.2	9.3	1.8	0.90	ns
Similarities	8.4	2.6	9.4	2.1	10.0	1.3	1.27	ns
Nonverbal subtests¹								
P. completion	7.6 ^{ab}	2.3	10.9 ^b	1.6	9.9 ^a	0.8	7.53	< .01
P. arrangement	10.5	3.3	11.0	2.8	12.6	2.3	1.24	ns
Block design	8.8 ^{ab}	2.4	12.4 ^b	1.8	12.5 ^a	2.9	5.95	< .01
Object assembly	7.1 ^{ab}	2.7	10.6 ^b	1.1	10.9 ^a	2.0	7.63	< .01
Digit symbol	5.8 ^a	1.4	7.7 ^b	3.3	10.9 ^{ab}	1.0	12.45	< .001
<u>Attention and information processing</u>								
DSf	6.1 ^a	1.6	7.4	1.9	9.8 ^a	1.9	8.40	< .01
DSb	5.3 ^a	1.7	6.3	2.7	8.4 ^a	2.4	3.93	< .05
MC	6.6	1.8	7.6	1.6	8.4	0.5	2.96	ns
TMTA	53.9 ^a	16.3	42.1 ^b	10.5	26.3 ^{ab}	5.0	11.40	< .001
TMTB	123.1 ^a	53.0	79.9	17.2	56.8 ^a	16.5	7.77	< .01
PASAT A	17.4 ^{ab}	20.3	39.1 ^b	10.1	49.5 ^a	7.2	11.11	< .001
PASAT B	11.6 ^{ab}	17.1	34.0 ^b	9.4	43.8 ^a	4.5	15.86	< .0001
PASAT C	9.1 ^{ab}	13.8	30.4 ^b	9.9	34.0 ^a	6.7	12.84	< .001
PASAT D	7.6 ^{ab}	11.3	21.9 ^b	10.2	23.5 ^a	7.0	6.48	< .01

Table 7. Neuropsychological Results (cont'n)

	Sleepy TBI		Nonsleepy TBI		Controls		F(2,20)	P
	M	SD	M	SD	M	SD		
CCC 3"	8.9 ^a	3.1	10.7	3.4	13.4 ^a	1.8	5.17	< .05
CCC 9"	6.0	2.2	6.6	3.9	9.5	3.2	2.85	ns
CCC 18"	5.0 ^a	3.2	5.4 ^b	2.9	10.1 ^{ab}	2.2	8.23	< .01
Stroop A	65.3 ^a	16.3	55.0	11.9	44.9 ^a	5.8	5.62	< .01
Stroop B	87.3 ^a	21.9	74.7	16.0	58.5 ^a	6.3	6.43	< .01
Stroop C	140.3	53.1	128.4	32.6	92.6	25.4	3.19	ns
<u>Executive Test</u>								
WCST cat	4.4	2.1	6.0	0.0	5.6	0.7	3.29	ns
WCST pers	10.5	8.1	4.4	4.2	5.1	7.4	1.80	ns
<u>Motor Tests²</u>								
GP R	98.3 ^a	21.8	79.5	20.6	68.3 ^a	9.2	5.77	< .01
GP L	101.4	68.9	98.7	16.5	69.9	12.9	1.25	ns
FT R	39.5 ^a	6.0	45.8 ^b	6.4	55.5 ^{ab}	7.9	11.05	< .001
FT L	36.2 ^a	11.8	42.2	4.8	51.0 ^a	4.3	6.90	< .01
GS R	34.4	6.5	38.2	10.5	39.6	6.1	0.96	ns
GS L	34.4	9.5	35.4	8.0	36.4	5.4	0.13	ns
<u>Affective scales</u>								
Anxiety	30.0	11.8	22.7	11.3	22.4	14.7	0.90	ns
Depression	12.0	9.3	10.0	5.6	5.0	2.5	2.46	ns

¹ Timed and untimed trials showed the same significant trends.

² (R) Right, dominant hand for all except two (one sleepy TBI and one control subjects); (L) Left

Note: The superscripts indicate groups which differ significantly by the Tukey HSD multiple comparison procedure ($p < .05$). For example, CCC 18" for controls differed significantly from CCC 18" for both sleepy and nonsleepy TBI.

The test of intelligence, WAIS-R, showed marked difference between controls and sleepy TBI subjects for all scales, full scale ($F(2,20) = 7.71, p < .01$), verbal ($F(2,20) = 4.39, p < .05$), and nonverbal ($F(2,20) = 10.18, p < .001$) (Figure 13). Moreover, results on the nonverbal scale also revealed a marked difference between the two TBI groups, sleepy TBI showing significantly poorer scores than nonsleepy TBI.

Figure 13. Intelligence Quotients (WAIS-R)

The WAIS-R subtest means for the three groups are presented in Figure 14. Subtest profiles revealed that two tasks, one verbal, Digit Span ($F(2,20) = 12.57, p < .001$) and one visuo-spatial, Digit Symbol ($F(2,20) = 12.45, p < .001$), differentiated controls from both TBI groups.

On the other hand, three visuo-spatial tasks distinguished sleepy TBI from both nonsleepy TBI and controls (Picture Completion ($F(2,20) = 7.53, p < .01$), Block Design ($F(2,20) = 5.95, p < .01$) and Object Assembly ($F(2,20) = 7.63, p < .01$)). These differences were not related to time but rather to cognitive difficulties in executing the tasks since both the timed and untimed conditions showed similar trends.

Figure 14. Subtest Profiles (WAIS-R)

No significant difference was found on the Mental Control subtest of the Wechsler Memory Scale, although a trend was present (Figure 15). Sleepy TBI subjects seemed to experience more difficulties in executing automatisms.

Figure 15. Mental Control (WMS-R)

On both Trail Making Tests, there was a marked difference between sleepy TBI and controls (Figure 16). Furthermore, TMT part A also distinguished both TBI groups. Thus for part A (the simpler task), sleepy TBI subjects were slower than nonsleepy TBI and control ones ($F(2,20) = 11.40, p < .001$), and on part B (the more complex task) nonsleepy TBI were midway in terms of speed of execution, with the sleepy TBI slowest ($F(2,20) = 7.77, p < .01$).

Thus, a gradient effect was present on both tasks. This gradient was less uniform on part B which is cognitively more demanding and consequently more taxing for both TBI groups.

Figure 16. Trail Making Tests

The PASAT results, like the performance subtests, differentiated the most between the sleepy TBI subjects and both, nonsleepy TBI and control subjects, for all presentation rates (2.4", $F(2,20) = 11.11, p < .001$; 2.0", $F(2,20) = 15.86, p < .0001$; 1.6", $F(2,20) = 12.84, p < .001$; 1.2", $F(2,20) = 6.48, p < .01$) (Figure 17). For all groups, performance decreased as rate of presentation increased.

Moreover, there was a disproportionate deterioration in performance between sleepy TBI and both nonsleepy TBI and control subjects. It appeared as if the task was too demanding at all levels (presentation rates) for these former, thereby suggesting more severely affected information processing capabilities.

Figure 17. Paced Auditory Serial Addition Task

The CCC showed marked differences between sleepy TBI and controls for the 3" ($F(2,20) = 5.17, p < .05$) and 18" ($F(2,20) = 8.23, p < .01$) delay (Figure 18). For the longer delay (18"), both TBI recalled fewer letters than the control subjects. Thus, the greater the delay, the worse the performance for both TBI groups. The interference effect occurred at the shortest delay and reached somewhat of a plateau with increasing delay intervals.

Figure 18. Consonant Trigram Test

On the Stroop test (Figure 19), there were marked differences between the sleepy TBI subjects and controls for the reading (A) ($F(2,20) = 5.62, p < .01$) and the naming (B) ($F(2,20) = 6.43, p < .01$) subtasks, but not for the interference subtask (C), which was likely due to the greater variability of the data ultimately compromising the sensitivity of the test. A disproportionate deterioration was evident on the interference subtask in disfavor of the sleepy TBI, suggesting again more severely impaired information processing abilities. Again, a clear gradient effect was revealed for the sleepiness factor.

Figure 19. Stroop Test

Although on the Wisconsin card sorting test the sleepy TBI group deducted on average fewer categories and produced more perseverations than the other two groups, no significant differences were found (Figure 20). Moreover, these results were within the norms.

Figure 20. Wisconsin Card Sorting Test

Manual dexterity as measured by the Grooved Pegboard was markedly different between sleepy TBI subjects and controls for the right hand ($F(2,20) = 5.77, p < .01$) but not for the left hand (Figure 21). Greater variability in the results of the left hand for the sleepy TBI subjects was present (SD 68.9 VS 16.5 for nonsleepy TBI and 12.9 for controls), attenuating the power of this statistical tests.

Figure 21. Grooved Pegboard

The speed of finger tapping was inferior in both hands for the sleepy TBI group compared to controls, and more so for the right hand ($F(2,20) = 11.05$, $p < .001$) compared to the left hand ($F(2,20) = 6.90$, $p < .01$) (Figure 22). For the nonsleepy TBI subjects, only the results for the right hand showed significantly slower speed for finger tapping when compared with controls. Again, greater variability in the results of the left hand for the sleepy TBI subjects was present (SD 11.8 VS 4.8, nonsleepy TBI and 4.3, controls).

Figure 22. Finger Tapping

Grip strength measures revealed no significant difference among the different groups for either hand (Figure 23).

Figure 23. Grip Strength

Affective scales measuring levels of anxiety (IPAT) and depression (Beck) showed no significant difference among the different groups, suggesting relatively normal emotional status for both TBI groups (Figure 24). The results for both scales were within the norms. Moreover, these normal findings were corroborated by the results on the Katz adjustment scale (Table 8), suggesting that TBI subjects were well adjusted overall.

Figure 24. Affective Scales

Interestingly, the Katz questionnaire for TBI subjects was mostly completed by their parents, especially their mother (9 out of 15) or wife (2 out of 15), whereas for controls it was most usually a close friend. This reflects the different living arrangements between TBI

Table 8. Katz Adjustment Scale Scores (Relatives's Form)

	Sleepy TBI		Nonsleepy TBI		Controls		F(2,20)	P
	M	SD	M	SD	M	SD		
<u>R1</u>								
Belligerence	6.5	2.3	5.0	1.2	5.6	1.5	1.38	ns
Verbal expans.	9.5	3.5	6.4	1.3	8.1	2.6	2.47	ns
Negativism	16.4 ^a	3.9	12.4 ^a	2.0	13.8	2.1	3.91	<.05
Helplessness	6.5	1.4	5.3	1.5	6.0	1.9	1.07	ns
Suspiciousness	5.1	1.7	4.7	0.8	5.5	2.8	0.29	ns
Anxiety	7.1	1.8	6.6	1.6	7.3	1.6	0.34	ns
Withdrawal/ret.	12.6	3.5	9.0	2.0	10.4	3.4	2.59	ns
General psycho.	11.7	4.1	5.6	2.1	9.5	3.4	1.97	ns
Nervousness	7.9	2.4	6.7	2.1	7.5	1.7	0.60	ns
Confusion	4.3	2.1	3.6	0.5	3.1	0.4	1.60	ns
Bizarreness	6.1	1.7	5.3	0.5	6.6	1.7	1.61	ns
Hyperactivity	5.9	2.7	5.3	1.1	4.6	1.8	0.74	ns
Stability	30.6	3.9	32.7	2.4	32.5	2.3	1.14	ns
<u>R2</u>	32.3	4.3	35.0	6.2	33.1	3.9	0.63	ns
<u>R3</u>	31.6	6.9	36.0	6.0	29.9	13.3	0.71	ns
<u>R4</u>	39.0	11.3	40.1	8.0	36.3	5.3	0.41	ns
<u>R5</u>	22.8	6.7	21.8	4.0	21.7	4.4	0.08	ns

R1, symptom discomfort; R2, R3 general activities; R4, R5 free-time activities

Note: The superscripts indicate groups which differ significantly by the Tukey HSD multiple comparison procedure ($p < .05$). For example, negativism for sleepy TBI differed significantly from negativism for nonsleepy TBI.

and control subjects.

Correlation Measures

Correlations analyses between major variables were performed in TBI subjects. From the clinical data, sleep variables and arousal (especially VRT) measures (Table 9), a positive correlation was found between nighttime sleep latency and daytime sleep latency (MSLT) ($r = .69$, $p < .01$), suggesting a generalized disorder for initiating sleep around the 24 hours. Also, visual reaction time, which is recognized as a good indicator of the severity of the brain injury, was negatively correlated with TST ($r = -.62$, $p < .01$) and SEFF ($r = -.65$, $p < .01$) and positively correlated with sleep latency ($r = .61$, $p < .01$). In other words, the

Table 9. Correlations with Clinical Data, Sleep Variables and Arousal Measures

<u>Night Sleep</u>				
	TST	SEFF	S 1.	S2
<u>Clinical</u>				
Coma	-.10	-.10	.37	.11
<u>MSLT</u>				
S1 1.	-.48	-.23	.69*	-.45
<u>Arousal</u>				
VRT	-.62*	-.65*	.61*	-.46

* $p < .01$

greater the visual reaction time (and in turn the severity of the brain injury), the longer it took the TBI subjects to fall asleep at night, the shorter their sleep time and the poorer their sleep efficiency.

Correlations from all the data set (clinical data, sleep variables, arousal measures, and neuropsychological results) revealed some interesting findings (Table 10). Three tasks (TMT-B, WCST cat., GP L, and FT L) were markedly correlated ($p < .001$) to the sleep variables. TMT-B was negatively correlated with stage 2 ($r = -.78$, $p < .001$). Other less significant correlations involving TMT-B and sleep variables were also present following the same trend than with VRT.

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The most significant correlation was between Finger Tapping for the left hand and nighttime sleep latency ($r = -.91, p < .001$). As illustrated in Figure 25, the slower the speed of the left hand, the longer the latency to fall asleep at night. Finger tapping is known to be a good measure of the integrity of the cerebral hemispheres. In the present case, this marked correlation suggests that those sleepier TBI had greatest cerebral impairment, especially the right hemisphere.

Figure 25. Finger Tapping Left Hand vs Sleep Latency

Finally, individual differences about subjective and objective sleep data are presented in Table 11.

Table 11. Individual Differences: Subjective and Objective Sleep Data

Subject	Nighttime Sleep			Daytime Sleep	
	Subjective Initiation difficulty	Latency (min.)	Objective Latency	Subjective Sleepy	Objective Latency
Sleepy TBI					
CC	rarely	15	66	occasionally	15
KD	rarely	1-3	1	frequently	11
LD	frequently	15-20	9	always	7
JJ	rarely	15	54	rarely	20
GL	occasionally	10	30	frequently	15
JM	always		60	always	20
PM	occasionally	30	24	occasionally	17
SP	always	30-45	13	always	18
Nonsleepy TBI					
MA	occasionally	1-30	2	occasionally	10
JC	occasionally	60	3	frequently	11
KC	rarely	15	22	occasionally	13
RL	rarely	15	6	rarely	7
GM	rarely	15-20	19	rarely	16
SM	always	60	38	rarely	20
MH	frequently	120	22	rarely	15
MT	frequently	120	1	rarely	10
Controls					
AB	rarely	20-30	9	rarely	18
KB	rarely	15	4	occasionally	12
MD	rarely	15	7	rarely	12
RH	occasionally	15	12	occasionally	12
JL	rarely	5	14	rarely	6
PL	rarely	15-20	1	occasionally	4
DS	rarely	5	9	occasionally	16
MO	rarely	10-15	8	rarely	16

Summary

In summary, nocturnal sleep measures differed between the TBI groups and the control group (prediction 1). This difference was mainly seen in sleepy TBI subjects. They showed a form of hyposomnia (rather than hypersomnia) which consisted of less total sleep with a particular reduction in stage 2 sleep. This loss of sleep was seemingly due to a difficulty in initiating rather than maintaining sleep. This was evidenced by a longer sleep onset latency and a relatively normal sleep efficiency. There was also evidence of more arousal after sleep onset and early morning awakenings.

The MSLT results revealed no gradient effect among the three groups contrary to prediction 2. In fact, TBI groups, though not statistically significant, took longer to fall asleep than controls. Furthermore, the time-of-day effect (midday shortening of sleep latency) was mainly present in the controls.

A positive relationship was found between MSLT and night sleep latency measures for TBI groups, suggesting a similar difficulty falling asleep during the night and the daytime. This finding is contrary to prediction 3 which stated that greater nighttime awakenings after sleep onset would be related to shorter daytime sleep onsets as determined by the MSLT.

A relationship was found between subjective (as measured by SSS) and objective

sleepiness (MSLT) but not in the direction expected in prediction 4. TBI groups reported feeling alert throughout the day and showed high alertness levels inferred from the extended latency to sleep on the MSLT.

RT was slower for both TBI groups compared to controls (prediction 5). The sleepiness factor, however, did not influence the performance of either TBI group.

No recuperative effect following naps was seen for the sleepy TBI group (prediction 6). On the contrary, performance tended to deteriorate over time.

No significant difference was found between groups for heart rate during the performance of the simple VRT task (prediction 7). All groups had similar levels of autonomic arousal.

Finally, of all the measures taken, neuropsychological tests showed the greatest gradient effect between the three groups; sleepy TBI subjects performing worse than controls with nonsleepy TBI subjects in between (prediction 8). This held true except for a few tests on which both TBI groups did poorer than controls. Inter-subject variability was greater in both TBI groups more so for the sleepy one.

DISCUSSION

Post-traumatic dysomnia

The major finding in this study is the presence both of sleep onset and, to a lesser extent, of sleep maintenance difficulties in chronic TBI subjects who report high levels of daytime subjective sleepiness. The longer sleep onset latency was also found during daytime (i.e., MSLT) suggesting the presence of a generalized disorder of initiating and maintaining sleep (DIMS). This finding is reminiscent of that of Stepanski and colleagues (1988) who, in a group of subjectively sleepy insomniacs, found prolonged, rather than shortened, sleep latencies at night and on MSLT.

In the TBI subjects, this insomnia is most likely of an organic type. This is for several reasons:

- 1) the absence of a previous history of any form of sleep disorders before their accident,
- 2) the absence of significant affective disorders known to be a major factor in some sleep disorders and, more importantly,
- 3) the marked correlations between the neuropsychological (including the VRT) results and sleep variables, indicating greater brain impairment in those subjects with higher

subjective (sleep questionnaire) and objective (night sleep and MSLT) sleep problems.

As mentioned earlier, no obvious pattern differences of cerebral injuries could be found to distinguish sleepy from nonsleepy TBI subjects. This can partially be explained by the limited resolution of the neurodiagnostic tool and further by its use only in the acute phase. Again, for most of these TBI subjects, this was the computed tomography (CT) (Snow, Zimmerman, Gandy & Deck, 1986). On the other hand, the presence of a gradient effect between subjective (i.e., sleepy vs nonsleepy) and objective (i.e., nighttime and daytime SOL) sleepiness and most neuropsychological results suggests greater brain impairment in sleepy TBI subjects.

The longer SOL seen in the sleepier and, at the same time, the more severely brain injured subjects (as measured by neuropsychological testing) could be due to impairment of basal forebrain cortical centers known to be the most vulnerable cerebral region to a closed head injury (Courville, 1947) and also to act as inhibitors of the arousal system at sleep onset (Serman & Clemente, 1962). Experimental studies with animals suggest that the orbitofrontal cortex is important in the generation of synchronized slow wave activity and behavioral sleep (Jouvet, 1962); and bilateral lesions of this region result in a permanent insomnia (Villablanca, Marcus & Olmstead, 1976). The orbitofrontal cortex, together with the basal forebrain, thus appears to constitute a forebrain synchronizing system (Jones, 1989). Interestingly, the first documented cases of organic insomnia also had their lesions predominantly in the basal forebrain (Von Economo, 1930).

On the other hand, the relatively normal sleep efficiency index found in both TBI groups once sleep is initiated appears to indicate a subsequent normal course of sleep with the exception for some individuals of greater susceptibility to awakening after sleep onset. Sleep fragmentation has been shown to diminish the restorative value of sleep (Levine, Roehrs, Stepanski, Zorick, & Roth, 1987). Once sleep begins, both slow wave and REM sleep appear preserved in chronic TBI at least in terms of quantity.

After the acute injury phase or in the first two years following the accident, sleep patterns in TBI patients tend to reappear and stabilize over time. Initially, the victim fluctuates between light sleep (stage 2) and the waking stage. Then over time, stages 3, 4 and REM show improvement. As suggested by the present findings, there seems to be a long-term homeostatic process in which the two main sleep stages (NREM and REM) eventually return to normal but at the expense of stage 2. This stage is a relatively light sleep which represents 50% of the night in normal subjects (Hauri, 1982) and seems to act as a transitional stage. Furthermore, sleepier subjects typically have decreased total sleep time and frequent arousals and awakenings. Therefore, sleepiness after TBI appears to result from hyposomnia with chronic sleep deprivation rather than hypersomnia. A frequent sequence of sleep recuperation following severe TBI would therefore be from coma to hypersomnia and subsequently chronic insomnia.

As for the lack of longer sleep onset in some previous sleep studies with TBI subjects (Ron et al., 1980; Prigatano et al., 1982; Guilleminault et al., 1983), various reasons can be

advanced such as group heterogeneity (including stroke, whiplash and TBI victims) or a shorter time interval between the accident and the sleep study. The main reason most probably relates to the absence in previous studies of group classification on the basis of subjective sleepiness. For example, in Prigatano's study (1982), which is the only prior study done with a homogeneous but single group of TBI subjects, all were complaining of sleep or dream problems or both. However no rating of subjective sleepiness was reported, which could mask important individual differences revealed in the present study.

In a recent study documenting sleep complaints from acute and chronic TBI victims (Cohen, Oksenberg, Snir, Stern & Groswasser, 1992), disorders in initiating and maintaining sleep (DIMS) were found more prevalent in acute hospitalized subjects, whereas disorders of excessive somnolence (DOES) were mainly reported by discharged patients whose accident had occurred two to three years previously. No polysomnographic studies, however, were done to document the true nature of their sleep. Also, as pointed out by the researchers, the sleep problems persisting for a long time may have provoked chronic insomnia which in turn may have lead to excessive somnolence from sleep deprivation. This could well explain the findings of the present study, since the TBI subjects with subjective sleepiness during the day turned out to be insomniacs.

Although TBI subjects felt subjectively sleepier according to the sleep questionnaire, no such feelings of sleepiness was recorded with the Stanford Sleepiness Scale. This is possibly due to the fact that the SSS tends to measure feelings of sleepiness at a particular

time which as a method has proven unreliable for chronic sleepy subjects (Herscovitch & Broughton, 1981b). As suggested by Johns (1991), the measurement of general level of daytime sleepiness as measured by the present sleep questionnaire (Appendix A) is possibly more appropriate. Johns (1991) devised a similar scale to measure sleep propensity, the Epworth Sleepiness Scale, and showed better correlations with sleep components such as sleep latency measured by nighttime and daytime (MSLT) polysomnography than that with the SSS. In the case of insomnia, however, feelings of tiredness and fatigue are not related to sleep propensity as manifested by the tendency to fall asleep.

Only one other study, and seemingly the first one to link a specific sleep disorder to head injury, documented a case of insomnia following a traumatic brain injury (Guilleminault, Cathala & Castaigne, 1973). It was a single case report of a man who following a car accident had survived for a long period of time (6 years) with a brainstem lesion and who had developed a chronic hyposomnia (sleeping fewer hours than before his accident) without any subjective complaints. The absence of significant functional problems in this man was somewhat atypical of present traumatic brain injuries. It should be pointed out that CT and other structural scanning techniques did not exist at the time and that no neuropsychological evaluation was performed.

A circadian sleep-wake schedule disorder arising after a closed head injury has also been reported recently (Patten & Lauderdale, 1992). A TBI adolescent had delayed nocturnal sleep onset followed by a normal sleep. Although no objective sleep measures were taken,

the authors concluded that this disorder was possibly the cause of some of the insomnia that frequently follows closed head injury. On the other hand, this TBI adolescent showed normal neuropsychological functions, in contrast to the present subjects, suggesting a more functional cause for his sleep problem. The authors did in fact suggest such a possible explanation referring to a syndrome of motivated sleep phase delay in adolescents (Ferber & Boyle, 1983). In the present study, TBI subjects, especially the sleepy ones, had difficulty falling asleep but also slept on average less than the normal amount of sleep and tended to awaken earlier.

Brain injuries can also lead to other sleep disorders. Posttraumatic narcolepsy, a rare entity, was found to be the result of an interaction between the brain injury and a genetic predisposition (Good, Barry, & Fishman, 1989). Jactatio nocturna (nocturnal rhythmic movements of the head and limbs) can occur after head injury and may indicate disordered arousal from stage 2 sleep controllable by medication (Drake, 1986). Kleine-Levin syndrome, a rare condition typified by periodic hypersomnolence, behavioural disturbance and hyperphagia seemingly related to hypothalamic dysfunction has also been reported following head trauma (Will, Young, & Thomas, 1988). Most of these studies, however, consisted of single individuals who did not necessarily sustain the type of traumatic brain injury typical of car accidents. Other conditions that may occur following brain injury, documented by polysomnography, include sleep apneas, both central and peripheral, nocturnal myoclonus, restless leg syndrome, and post-traumatic nocturnal seizures.

Neuropsychological impact

As previously mentioned, statistical analyses of the neuropsychological results revealed a marked relationship between the groups and the severity of the deficits. For most variables, there was almost a linear relationship among the three groups with the sleepy TBI subjects showing the worst scores, the controls the best and nonsleepy TBI subjects in between. Previous long term studies had also shown that severe TBI results in diverse cognitive deficits (Dikmen, Machamer, Temkin, & McLean, 1990; Drudge, Williams, Kessler, & Gomes, 1984; Levin, Grossman, Rose, & Teasdale, 1979; Tabaddor, Mattis, & Zazula, 1984). The present study, however, is the first one to distinguish group differences based on subjective sleepiness.

The intelligence test (WAIS-R) showed greater group differences on performance than on verbal variables. Similar findings showing a differential recovery pattern as a function of type of ability have been reported (Dikmen et al., 1990; Mandleberg & Brooks, 1975; Tabbador et al, 1984). Nonverbal tasks tap into more fluid cognitive abilities which are possibly more vulnerable to the diffuse neurologic damage resulting from TBI (Lezak, 1987). Indeed, one of the performance variables, Symbol Digit, has been shown to be correlated with white matter atrophy (Johnson, Bigler, Burr, & Blatter, 1994) and also to be a good indicator of vocational success (Prigatano, 1986) in TBI patients. Also, its equivalent form, the Symbol Digit Modalities Test (Smith, 1973) has been found to be the best single measure of the deficit in speed of information processing following a closed head injury (Ponsford &

Kinsella, 1992).

Tests of attention such as TMT and PASAT were impaired for both TBI groups, although more so for the sleepy one. TMT-B was one of the neuropsychological variables that correlated significantly with sleep parameters, in particular sleep latency and stage 2. It is noteworthy that Prigatano et al. (1982), in one of the few previous sleep studies with TBI subjects, reported a modest positive correlation between TMT-B and stage 1. That the performance on these attentional tests (TMT and PASAT) is due to head injury effects per se and not to post-traumatic sleepiness is supported by the finding that untreated pathologically sleepy narcoleptics with very short sleep latencies perform the PASAT at normal levels (Valley & Broughton, 1981). Compared to most of the other tasks, the PASAT makes greater cognitive demands by not being paced by the subject (Ponsford & Kinsella, 1992).

The CCC was the only test on which both TBI groups did poorly compared to controls. Stuss and his colleagues (1985) demonstrated that this test was the most sensitive in discriminating between a group of subjects with an apparently good recovery after a closed head injury and a group of matched controls. They related this limitation in information processing capacity to a divided attention deficit, either in terms of speed of processing or in terms of the amount of information that can be handled simultaneously. Furthermore, this discrimination power between TBI and control subjects was shown to be independent of the time of recovery, suggesting the stability of this deficit following a closed head injury.

As for the Stroop test, recent studies (McLean, Temkin, Dikmen & Wyler, 1983; Stuss et al., 1985; Ponsford & Kinsella, 1992) have obtained similar findings with TBI subjects (i.e., being slower than controls on all subtests but not disproportionately slower on the interference task).

Analyses of the neuropsychological test performance in chronic severe traumatic brain injury have underlined two factors, the selective impairments of specific cognitive processes and the impairments in the capacity to allocate resources effectively (Peach, 1992). As suggested by previous research (Ponsford & Kinsella, 1992; Stuss et al., 1985, Van Zomeran, 1981), the impaired performance of TBI subjects on tests of attention seems to relate more to reduced speed of information processing than to accuracy. Where possible, TBI subjects tend to sacrifice speed in an effort to maintain accuracy. On the other hand, these impairments in information processing could be related to the sleep problems, both resulting from a structural disruption in the normal frontal-limbic-reticular activating system connections (Herscovitch, Stuss, & Broughton, 1980). One possible explanation would be that the impaired cortical mechanisms necessary for the deactivation of the arousal system and initiation of sleep would also be involved in the regulation of information processing (Doghramji & Zinsenheim, 1993). Furthermore, these impaired gating systems could also be partly responsible for the greater variability and inconsistency of performance found in TBI subjects (Stuss, Pogue, Buckle, & Bondar, 1994).

The findings on the WCST are comparable to those of previous studies (Teuber,

Battersby, & Bender, 1951; Rosvold & Mirsky, 1964; Heck & Byer, 1986, and Anderson & Damasio, 1991) which showed that the performance of subjects with frontal lobe damage who are studied in the chronic stages tend to be within the normal range. Therefore, the correlations found between WCST and different sleep components (TST, S2) are somewhat spurious and must be replicated with a larger population of TBI subjects, given that most TBI subjects had deduced all the card sort categories. No explanation has been given for this normalization of performance on the WCST in severe TBI subjects. One possible reason from clinical experience is a practice effect especially in severe TBI subjects who have been tested on numerous occasions. They tend to remember response routines instead of deducing the sorting principles.

Interestingly, the most significant correlation was between sleep onset latency (SOL) and finger tapping by the left hand. This suggests greater right hemisphere impairment in those TBI subjects with greater difficulties falling asleep. It is noteworthy that in closed head injury the main effect is the shearing of axon tissue (Strich, 1961) which may affect more the right hemisphere than the left (Tromp & Mulder, 1991). This is based on the fact that the right hemisphere contains more fibers (white matter) than cells (grey matter) than the left hemisphere (Goldberg & Costa, 1981), particularly in the frontal and precentral regions (Gur, Packer, Hungerbuhler, Reivich, Obrist, Amanek & Sackeim, 1980). Interestingly, Prigatano (1989), using finger tapping speed, had also found a greater right-hemisphere impairment in TBI patients who showed several months after injury unawareness of deficits, which is the most deleterious effect of such a cerebral insult. Greater right brain impairment indexed as

an abnormal right frontal visual P300 evoked potential brain map has also been reported in a group of closed head injury patients, some of whom complained of excessive daytime sleepiness (Sangal & Sangal, 1994).

As suggested by the Ommaya and Gennarelli study (1974) which found a centripetal spread of the injury in TBI, it would appear that the more severe the impact, the greater the damage, especially in the orbitofrontal and basal forebrain regions (Courville, 1942; Malloy, Bihrlé, Duffy, & Cimino, 1993). These tissues harbour the deactivating mechanisms for sleep, resulting in a greater sleep dysfunction (i.e., longer SOL). Thus, individuals with deeper lesions would present more profound impairments in sleep architecture than those with only cortical injuries. This would also suggest that different types of sleepiness can be found in closed head injured subjects. In milder head injured subjects in whom the damage is mainly limited to the surface of the brain, the fatigability often reported (Lishman, 1988) might be related to a decreased cognitive endurance. In the more severe cases of deeper damage both cerebral fatigue and impaired sleep mechanisms would be present.

From recent studies on the neurobiology of sleep which support a thermoregulatory model of sleep (McGinty & Szymusiak, 1994), cerebral fatigue is possibly related to an increased metabolic rate. Thus, it is known from clinical experience that following a TBI, individuals tend to expend greater energy in performing tasks. Such cerebral fatigue or decreased cognitive endurance often observed in mild TBI could be the result of an increased metabolic rate. As pointed out by McGinty & Szymusiak (1994), further evidence is needed

to demonstrate a role for cerebral fatigue in regulating arousal (homeostatic sleep-promoting process). Sleep studies have also demonstrated that the hypothalamic mechanisms regulating temperature and metabolism and in turn sleep are also closely linked to the adjacent suprachiasmatic nucleus which is responsible for the circadian regulation of sleep. This biorhythmicity as shown by the data on MSLT and VRT tasks would seem to be somewhat affected (i.e., phase advanced) in sleepy TBI as compared to controls.

The type of sleepiness which was found in severe TBI subjects raises the issue of the use of the most appropriate tests for its evaluation. As discussed by Stepanski et al. (1998), the use of the MSLT in evaluating sleep tendency in insomniac subjects may not be appropriate. Both sleep need and sleep ability, also termed "sleepability" (Lavie & Scherson, 1981), are confounded (Broughton, 1992). Furthermore, as pointed out by Wilkinson (1992), the MSLT does not provide adequate control for the mental activity in some subjects (i.e., insomniacs). It may be quite high, thus affecting the arousal level and in turn sleep propensity. Performance tasks sensitive to fluctuations in arousal, on the other hand, provide for adequate control of subject activity at both behavioral and physiological levels. This is corroborated by the greater sensitivity of RT to detect the midday effect as compared to MSLT. Furthermore, in contrast to the MSLT, these tasks can correlate well with subjective levels of sleepiness (Johnson, Spinweber, Gomez, & Matteson, 1990). In the present study, the correlations mainly involved the objective sleep components.

Use of RT tasks may also be more appropriate for detecting deficits in mild as

opposed to severe TBI subjects. It has been found that the assessment of mild TBI subjects must utilize sensitive tasks such as complex attentional tasks. These tests are long lasting and truly tax the fatigability of these subjects (Binder & Rattok, 1989). Thus, the subtle deficits in attention in these individuals can be best detected by the use of tasks which create enhanced attentional demand over longer periods of time.

The present neuropsychological results underline the importance of investigating sleep problems in TBI subjects. They represent a very significant and confounding source of variability. In terms of overall sleep evaluation for these subjects, it may be more appropriate to use ambulatory recording (Broughton, 1989). The use of 24 hour sleep-wake monitoring would permit a better understanding of sleep-wake patterns by documenting variables of clinical interest such as the amount, type, and time of occurrence of different sleep components. In mild TBI subjects in whom mental fatigue is often the major obstacle to resuming their previous occupation, ambulatory sleep-wake recordings would provide useful representative electrophysiological measures. Subjects would be free to go about their normal activities and engage in tasks that tax their impaired cognitive functions. Correlations of behavioral events with polygraphic recordings could be obtained by the maintenance of a sleep wake log during the recording and also the use of the event marker built into the recorder. More advanced quantitative EEG technology, such as spectral analysis, could also be applied in an effort to better characterize brain state (Tebano, Cameroni, Gallozzi, Loizzo, Palazzino, Pezzini, & Ricci, 1989; Thatcher, Walker, Gerson, & Geisler, 1989; Gevins, Bressler, Cutillo, Illes, Miller, Stern, & Rex, 1990).

Also, use of more powerful neurodiagnostic tools to image the brain after trauma such as magnetic resonance imaging (MRI) for structural damage (Levin, Amparo, Eisenberg, William, High, McArdle, & Weiner, 1987) and single photon emission computed tomography (SPECT) for functional pathology (Ichise, Chung, Wang, Wortzman, Gray, & Franks, 1994) would provide more detailed evidence of structural damage to correlate with impaired neuropsychological performance (Wilson & Wyper, 1992). In addition to the stronger correlations expected with more clearly defined TBI groups, one would expect to reduce the extraneous variability evident in many of the sleep and neuropsychological measures. This line of reasoning can be somewhat supported by the marked correlations reported in the present study (e.g., finger tapping left hand vs sleep latency). Therefore, improved neurodiagnostics could serve to better control for this problem.

A second aspect related to the issue of variability involved the choice of statistical test employed in the study, specifically the ANOVA. When the underlying assumptions of normality and homogeneity of variance were suspect, the non-parametric Kruskal-Wallis test was performed and showed similar trends.

Finally, as for remedial solutions to the sleep problems of TBI victims, no specific or proven ones exist for the moment. Although medication such as hypnotics has been suggested by some clinical practitioners (Kowatch, 1989; Cassidy, 1991), no systematic study has been done to evaluate the use of drugs to alleviate sleep disorders after TBI. Drug trials would need to be performed using double-blind protocols in conjunction with systematic

objective polygraphic measurement. Cerebrospinal fluid analyses would be useful for possible involvement of specific neurotransmitter metabolites. On the other hand, non-pharmacological strategies, such as basic sleep hygiene, relaxation training, and cognitive treatments, could potentially assist in the management of these sleep problems (Hauri, 1989).

REFERENCES

- Adams, J.H. (1975). The neuropathology of head injuries. In P.J. Vinken & G.W. Bruyn (Eds.), Handbook of clinical neurology: Injuries of the brain and skull, (Vol. 23) Part I (pp. 35-65). Amsterdam: Elsevier Publishing Co.
- Adams, J.H. (1990). Brain damage in fatal non-missile head injury in man. In P.J. Vinken, G.W. Bruyn, H.L. Klawans, & R. Baakman (Eds.), Handbook of clinical neurology: Head Injury, (Vol. 57) (pp. 43-63). Amsterdam: Elsevier Publishing Co.
- Adams, J.H. & Graham, D.I. (1984). Diffuse brain damage in non-missile head injury. In P.P. Anthony & R.N.M. MacSween (Eds.), Recent advances in histopathology (Ch. 12). Edingburgh: Churchill Livingstone.
- Adams, J.H., Graham, D.I., & Gennarelli, T.A. (1982). Neuropathology of acceleration-induced head injury in the subhuman primate. In R.G. Grossman & P.L. Gildenberg (Eds.), Head injury: Basic and clinical aspects (pp. 141-150). New York: Raven Press.
- Adams, J.H., Graham, D.I., Murray, L.S., & Scott, G. (1982). Diffuse axonal injury due to nonmissile head injury in humans: An analysis of 45 cases. Annals of Neurology, 12, 557-563.
- Adams, J.H., Graham, D.I., Scott, G., Parker, L.S., & Doyle, D. (1980). Brain damage in fatal non-missile head injury. Journal of Clinical Pathology, 33, 1132-1145.
- Adams, J.H., Mitchell, D.E., & Graham, D.I., & Doyle, D. (1977). Diffuse brain damage of immediate impact type: Its relationship to "primary brain-stem damage" in head injury. Brain, 100, 489-502.
- Adams, J.H., Scott, G., Parker, L.S., Graham, D.I., & Doyle, D. (1980). The contusion index: A quantitative approach to cerebral contusions in head injury. Neuropathology and Applied Neurobiology, 6, 319-324.
- Agnew, H.W., Jr., Webb, W.B., & Williams, R.L. (1966). The first-night effect: An study of sleep. Psychophysiology, 2, 263-266.
- Aldrich, M.S. (1989). Automobile accidents in patients with sleep disorders. Sleep, 12, 487-494.
- Anderson, S.W., Damasio, H., Jones, R.D., & Tranel, D. (1991). Wisconsin Card Sorting Test performance as a measure of frontal lobe damage. Journal of Clinical and Experimental Neuropsychology, 13, 909-922.

- Army Individual Test Battery: Manual of directions and scoring (1944). Washington: U.S. Army, War Department, Adjutant General's Office.
- Askenasy, J.J.M. & Rahmani, L. (1988). Neuropsychosocial rehabilitation of head injury. American Journal of Physical Medicine, 66, 315-327.
- Beck, A.T., Rush, A.J., Shaw, B.F., & Emery, G. (1979). Cognitive therapy of depression. New York: Guilford Press.
- Beck, A.T., Ward, C.H., Mendelson, M., Mock, J., & Erbaugh, J. (1961). An inventory for measuring depression. Archives of General Psychiatry, 4, 561-571.
- Berg, E.A. (1948). A simple objective technique for measuring flexibility in thinking. Journal of General Psychology, 39, 15-22.
- Bergamasco, B., Bergamini, L., & Doriguzzi, T. (1968). Clinical value of the sleep electroencephalographic patterns in posttraumatic coma. Acta Neurologica Scandinavica, 44, 495-511.
- Binder, L.M. (1986). Persisting symptoms after mild head injury: A review of the postconcussive syndrome. Journal of Clinical and Experimental Neuropsychology, 8, 323-346.
- Binder, L.M. & Rattok, J. (1989). Assessment of the postconcussive syndrome after mild head trauma. In M.D. Lezak (Ed.), Assessment of the behavioral consequences of head trauma (pp. 37-48). New York: Alan R. Liss, Inc.
- Birrell, P.C. (1983). Behavioural, subjective and electroencephalographic indices of sleep-onset latency and sleep duration. Journal of Behavioural Assessment, 5, 179-190.
- Blake, M.J.F. (1967). Time of day effects on performance in a range of tasks. Psychonomic Science, 9, 349-350.
- Bohnen, N., Jolles, J., & Twijnstra, A. (1992). Modification of the Stroop Color Word Test improves differentiation between patients with mild head injury and matched controls. The Clinical Neuropsychologist, 6, 178-184.
- Bricolo, A., Gentilomo, A., Rosadini, G., & Rossi, G.F. (1968). Long-lasting post-traumatic unconsciousness. A study based on nocturnal and polygraphic recording. Acta Neurologica Scandinavica, 44, 512-532.
- Broadbent, D.E. (1971). Decision and stress. London: Academic Press.
- Brooks, N. (1984). Cognitive deficits after head injury. In N. Brooks (Ed.), Closed head

- injury: Psychological, social, and family consequences (pp. 44-73). New York: Oxford University Press.
- Broughton, R.J. (1975). Biorhythmic fluctuations in consciousness and psychological functions. Canadian Psychological Review, 16, 217-239.
- Broughton, R.J. (1982). Human consciousness and sleep/waking rhythms: A review and some neuropsychological considerations. Journal of Clinical Neuropsychology, 4, 193-218.
- Broughton, R. J. (1989). Ambulant home monitoring of sleep and its disorders. In M. H. Kryger, T. Roth, & W.C. Dement (Eds.), Principles and practice of sleep medicine (pp. 696-701). Philadelphia: W.B. Saunders Co.
- Broughton, R.J. (1992). Qualitatively different states of sleepiness. In R.J. Broughton & R.D. Ogilvie (Eds.), Sleep, Arousal, and Performance (pp. 45-59). Boston: Birkhauser.
- Broughton, R.J. & Ogilvie R.D. (1992) (Eds.). Sleep, Arousal, and Performance. Boston: Birkhauser.
- Browman, C.P. & Cartwright, R.D. (1980). The first-night effect on sleep and dreams. Biological Psychiatry, 15, 809-812.
- Brown, J. (1958). Some tests of the decay theory of immediate memory. Quarterly Journal of Experimental Psychology, 10, 12-21.
- Carskadon, M.A. & Dement, W.C. (1982). The Multiple Sleep Latency Test: What does it measure? Sleep, 5, S67-S72.
- Carskadon, M.A., Dement, W.C., Mitler, M.M., Roth, T., Westbrook, P.R., & Keenan, S. (1986). Guidelines for the Multiple Sleep Latency Test (MSLT): A standard measure of sleepiness. Sleep, 9, 519-524.
- Carskadon, M.A., Mitler, M.M., Billiard, M., Phillips, R., & Dement, W.C. (1975). A comparison of insomniacs and normals: Total sleep time and sleep latency. Sleep Research, 4, 211.
- Cartlidge, N.E.F. & Shaw, D.A. (1981). Head Injuries. Philadelphia: Saunders.
- Cassidy, J.W. (1991). Insomnia. Journal of Head Trauma Rehabilitation, 6, 81-83.
- Cattell, R.B., Scheier, I.H., & Madge, E.M. (1968). Manual for the IPAT Anxiety Scale Questionnaire. Pretoria: National Bureau of Educational and Social Research.

- Chadwick, O. (1976). Conferences notes of a meeting of the joint British-Dutch head injury workshop. Oxford. (Unpublished observations)
- Chartrian, G.E., White, L.E., & Daly, D. (1963). Electroencephalographic patterns resembling those of sleep in certain comatose states after injuries to the head. Electroencephalography and Clinical Neurophysiology, 15, 272-280.
- Cohen, R.A. (1993). The neuropsychology of attention. New York: Plenum Press.
- Cohen, M., Oksenberg, A., Snir, D., Stern, M.J., & Groswasser, Z. (1992). Temporally related changes of sleep complaints in traumatic brain injured patients. Journal of Neurology, Neurosurgery, and Psychiatry, 55, 313-315.
- Conkey, R.C. (1938). Psychological changes associated with head injuries. Archives of Psychology, 33, 1-62.
- Courjon, J. (1972). Traumatic disorders. In A. Remond (Ed.), Handbook of electroencephalography and clinical neurophysiology: Clinical, (Vol. 14) IV Part B. Amsterdam: Elsevier Publishing Co.
- Courville, C.B. (1942). Coup-contrecoup mechanism of cranio-cerebral injuries: Some observations. Archives of Surgery, 45, 19-43.
- Crompton, R. (1985). Closed head injury. Its pathology and legal medicine. London: Edward Arnold.
- Curry, S.H. (1980). Event-related potentials as indicants of structural and functional damage in closed head injury. Progress in Brain Research, 54, 507-515.
- Dencker, S.J. & Lofving, B. (1958). A psychometric study of identical twins discordant for closed head injury. Acta Psychiatrica et Neurologica Scandinavica, 33, supplementum 122, 1-50.
- Dikmen, S., Machamer, J., Temkin, N. & McLean, A. (1990). Neuropsychological recovery in patients with moderate to severe head injury: 2 year follow-up. Journal of Clinical and Experimental Neuropsychology, 12, 507-519.
- Dinges, D.F., Orne, F.C., Evans, F.J., Orne, M.T. (1981). Performance after naps in sleep-conducive and alerting environments. In L.C. Johnson, D.I. Tepas, W.P. Colquhoun, & M.J. Colligan (Eds.), Advances in sleep research: Biological rhythms, sleep, and shift work (Vol. 7) (pp. 539-552). New York: Spectrum Publishers.
- Doghramji, K. & Zinsenheim, J. (1993). Sleep abnormalities following head trauma. In S. Mandel, R.T. Sataloff, & S.R. Schapiro (Eds.), Minor head trauma: Assessment.

management, and rehabilitation (pp. 123-141). New York: Springer-Verlag.

- Drudge, O.W., Williams, J.M., Kessler, M. & Gomes, F.B. (1984). Recovery from severe closed head injuries: Repeat testings with the Halstead-Reitan Neuropsychological Battery. Journal of Clinical Psychology, **40**, 259-265.
- Edgar, D.M., Dement, W.C., & Fuller, C.A. (1993). Effect of SCN lesions on sleep in squirrel Monkeys: Evidence for opponent processes in sleep-wake regulation. The Journal of Neuroscience, **13**, 1065-1079.
- Ferber, R. & Boyle, M.P. (1983). Delayed sleep phase syndrome versus motivated sleep phase delay in adolescents. Sleep Research, **12**, 239.
- Frankowski, R.F. (1986). Descriptive epidemiologic studies of head injury in the United States: 1974-1984. Advances in Psychosomatic Medicine, **16**, 153-172.
- Fuster, J.M. (1989). The prefrontal cortex: anatomy, physiology and neuropsychology of the frontal lobe (2nd ed.). New York: Raven Press.
- Gennarelli, T. (1993). Cerebral concussion and diffuse brain injuries. In P.R. Cooper (Ed.), Head injury (3rd ed.) (pp. 137-158). Baltimore: Williams & Wilkins.
- Gennarelli, T. (1983). Head injury in man and experimental animals: Clinical aspects. Acta Neurochirurgica, **S32**, 1-13.
- Gennarelli, T., Abel, J.M., Adams, J.H., & Graham, D. (1979). Differential tolerances of frontal and temporal lobes to contusion induced by angular acceleration. Proceedings of the 23rd Stapp Car Crash Conference. New York: Society of Automotive Engineers.
- Gennarelli, T.A., Spielman, G.M., Langfitt, T.W., Gildenberg, P.L., Harrington, T., Jane, J.A., Marshall, L.F., Miller, J.D., & Pitts, L.H. (1982). Influence of the type of intracranial lesion on outcome from severe head injury: A multicenter study using a new classification system. Journal of Neurosurgery, **56**, 26-36.
- Gennarelli, T.A., Thibault, L.E., Adams, J.H., Graham, D.I., Thompson, C.J., & Marcincin, R.P. (1982). Diffuse axonal injury and traumatic coma in the primate. Annals of Neurology, **12**, 564-574.
- Gevins, A.S., Bressler, S.L., Cutillo, B.A., Illes, J., Miller, J.C., Stern, J., & Rex, H.R. (1990). Effects of prolonged mental work on functional brain topography. Electroencephalography and Clinical Neurophysiology, **76**, 339-350.
- Glenville, M., Broughton, R.J., Wing, A.M., & Wilkinson, R.T. (1978). Effects of sleep

- deprivation on short duration performance measures compared to the Wilkinson auditory vigilance task. Sleep, **1**, 169-176.
- Goldberg, E. & Costa, L.C. (1981). Hemisphere differences in the acquisition and use of descriptive systems. Brain and language, **14**, 144-173.
- Good, J.L., Barry, E., & Fishman, P.S. (1989). Posttraumatic narcolepsy: The complete syndrome with tissue typing. Journal of Neurosurgery, **71**, 765-767.
- Goodman, W.A., Ball, S.D., & Peck, G. (1988). Psychosocial characteristics of head-injured patients. A comparison of factor structures of the Katz Adjustment Scales. Journal of Clinical and Experimental neuropsychology, **10**, 42.
- Gronwall, D.M. (1977). Paced Auditory Serial-Addition Task: A measure of recovery from concussion. Perceptual and Motor Skills, **44**, 367-373.
- Gronwall, D. (1987). Advances in the assessment of attention and information processing after head injury. In H.S. Levin, J. Grafman, & H.M. Eisenberg (Eds.), Neurobehavioral recovery from head injury (pp. 355-371). New York: Oxford university Press.
- Gronwall, D.M. & Sampson, H. (1974). The psychological effects of concussion. Auckland: Auckland University Press.
- Gronwall, D.M. & Wrightson, P. (1974). Recovery after minor head injury. Lancet, **2**, 1452.
- Guilleminault, C. Cathala, J.P. & Castaigne, P. (1973). Effects of 5-hydro-tryptophan on sleep of a patient with a brainstem lesion, Electroencephalography and Clinical Neurophysiology, **34**, 177-184.
- Guilleminault, C., Faull, K.F., Miles, L., & Van Den Hoed, J. (1983). Posttraumatic excessive daytime sleepiness: A review of 20 patients. Neurology, **33**, 1584-1589.
- Gur, R.C., Packer, I.K., Hungerbuhler, J.P., Reivich, M., Obrist, W.D., Amarnek, W.S., & Sackeim, H.A. (1980). Differences in the distribution of gray and white matter in human cerebral hemispheres. Science, **207**, 1226-1228.
- Gurdjian, E.S. & Gurdjian, E.S. (1976). Cerebral contusions: Reevaluation of the mechanism of their development. Journal of Trauma, **16**, 35-51.
- Halstead, W.C. (1947). Brain and intelligence. Chicago: University of Chicago Press.
- Harada, M., Minani, R., Haltori, E., Nakamura, K., Kabashima, K., Shikai, I., & Sakai,

- V. (1976). Sleep in brain damaged patients: An all night study of 105 cases. Kumamoto Medical Journal, 29, 110-127.
- Hauri, P. (1982). The sleep disorders. Kalamazoo, MI: The Upjohn Co.
- Hauri, P. (1989). Primary insomnia. In M.H. Kryger, T. Roth, & W.C. Dement (Eds.), Principles and practice of sleep medicine (pp. 442-447). Philadelphia: W.B. Saunders Co.
- Heck, E.T. & Bryer, J.B. (1986). Superior sorting and categorizing ability in a case of bilateral frontal lobe atrophy: An exception to the rule. Journal of Clinical and Experimental Neuropsychology, 8, 313-316.
- Herscovitch, J. & Broughton, R.J. (1981a). Performance deficits following short-term partial sleep deprivation and subsequent recovery oversleeping. Canadian Journal of Psychology, 35, 309-322.
- Herscovitch, J. & Broughton, R. (1981b). Sensitivity of the Stanford Sleepiness Scale to the effects of cumulative partial sleep deprivation and recovery oversleeping. Sleep, 4, 83-92.
- Herscovitch, J., Stuss, D., & Broughton, R. (1980). Changes in cognitive processing following short-term cumulative partial sleep deprivation and recovery oversleeping. Journal of Clinical Neuropsychology, 2, 301-319.
- Hoddes, E., Dement, W., & Zarcone, V. (1972). The development and use of the Stanford Sleepiness Scale. Psychophysiology, 9, 150. (Abstract)
- Hoddes, E., Zarcone, V., Smythe, H., Phillips, R., & Dement, W.C. (1973). Quantification of sleepiness: A new approach. Psychophysiology, 10, 431-436.
- Holbourn, A.H.S. (1943). Mechanics of head injuries. Lancet, 245 (2), 438-441.
- Ichise, M. Chung, D.G., Wang, P., Wortzman, G., Gray, B.G., & Franks, W. (1994). Technetium-99m-HMPAO SPECT, CT and MRI in the evaluation of patients with chronic traumatic brain injury: A correlation with neuropsychological performance. Journal of Nuclear Medicine, 35, 217-226.
- Ivan, L.P. (1984). The impact of head trauma on society. Canadian Journal of Neurological Sciences, 11, 417-420.
- Jamieson, K.G. & Yelland, J.D.N. (1968). Extradural hematoma. Report of 167 cases. Journal of Neurosurgery, 29, 13-23.

- Jasper, H.H. (1958). Report of the committee on methods of clinical examination in electroencephalography. Electroencephalography and Clinical Neurophysiology, 10, 370-375.
- Jennett, B. (1972). Prognosis after severe head injuries. Clinical Neurosurgery, 19, 200-207.
- Jennett, B. & Bond, M. (1975). Assessment of outcome after severe brain damage: A practical scale. Lancet, 1, 480-487.
- Jennett, B. & Teasdale, G. (1981). Management of head injuries. Philadelphia: F.A. Davis Co.
- Johns, M. W. (1991). A new method for measuring daytime sleepiness: The Epworth Sleepiness Scale. Sleep, 14, 540-545.
- Johnson, L.C., Spinweber, C.L., Gomez, S.A., & Matteson, L.T. (1990). Daytime sleepiness, performance, mood, nocturnal sleep: The effect of benzodiazepine and caffeine on their relationship. Sleep, 13, 121-135.
- Johnson, S.C., Bigler, E.D., Burr, R.B., & D.D. Blatter (1994). White matter atrophy, ventricular dilation, and intellectual functioning following traumatic brain injury. Neuropsychology, 8, 307-315.
- Jones, B.E. (1989). Basic mechanisms of sleep-wake states. In M.H. Kryger, T. Roth, & W.C. Dement (Eds.), Principles and practice of sleep medicine (pp. 121-138). Philadelphia: W.B. Saunders Co.
- Jouvet, M. (1962). Recherches sur les structures nerveuses et les mécanismes responsables des différentes phases du sommeil physiologique. Archives Italiennes de Biologie, 100, 125-206.
- Jouvet, M., Pellin, B., Mounier, D. (1962). Polygraphic study of the different sleep phases during chronic disturbances of consciousness (prolonged comas). Electroencephalography and Clinical Neurophysiology, 14, 138P.
- Kalsbeck, W., McLaurin, R., Harris, B., & Miller, J. (1980). The national head and spinal cord injury survey: Major findings. Journal of Neurosurgery, 53, 519-531.
- Katz, M.M. & Lyerly, S.B. (1963). Methods for measuring adjustment and social behavior in the community: I Rationale, description, discriminative validity, and scale development. Psychological Reports, 13, 503-535.
- Kirk, R.E. (1982). Experimental design: Procedures for the behavioral sciences (2nd ed.). Belmont, Calif: Brooks/Cole Publishing Co.

- Klensch, H. (1973). Die diagnostische Valenz der Reaktions zeitmessung bei venschiedenen zerebralem Erkrankungen. Fortsch. Neuro!. Psychiat., 41, 475.
- Klonoff, P.S., Costa, L.D., & Snow, W.G. (1986). Predictors and indicators of quality of life in patients with closed-head injury. Journal of Clinical and Experimental Neuropsychology, 8, 469-485.
- Klove, H. (1963). Clinical neuropsychology. Medical Clinics of North America, 46, 1647-1658.
- Kowatch, R.A. (1989). Sleep and head injury. Psychiatric Medicine, 7, 37-41.
- Lessard, C.S., Sances, A. & Larson, S.J. (1974). Period analysis of signals during sleep and post-traumatic coma. Aerospace Medicine, June, 664-668.
- Langfitt, T.W., Obrist, W.D., Alavi, A., Grossman, R., Zimmerman, R., Jaggi, J. Uzzell, B., Reivich, M. & Patton, D. (1987). Regional structure and function in head injured patients: Correlation of CT, MRI, PET, CBF, and neuropsychological assessment. In H.S. Levin, J. Grafman, & H.M. Eisenberg (Eds.), Neurobehavioral recovery from head injury (pp. 30-42). New York: Oxford University Press.
- Lavie, P. & Scherson, A. (1981). Ultrashort sleep-waking cycle: 1. Evidence of ultradian rhythmicity in "sleepability". Electroencephalography and Clinical Neurophysiology, 52, 163-174
- Levin, H.S., Amparo, E., Eisenberg, H.M., William, D.H., High, W.M. Jr., McArdle, C.B., & Weiner, R.L. (1987). Magnetic resonance imaging and computerized tomography in relation to the behavioral sequelae of mild and moderate injuries. Journal of Neurosurgery, 66, 706-713.
- Levin, H.S., Benton, A.L., & Grossman, R.G. (1982). Neuropsychological consequences of closed head injury. New York: Oxford University Press.
- Levin, H.S., Grafman, J., & Eisenberg, H.M. (1987) (Eds.). Neurobehavioral recovery from head injury. New York: Oxford University Press.
- Levin, H.S., Grossman, R.G., Rose, J.E., & Teasdale, G. (1979). Long term neuropsychological outcome of closed head injury. Journal of Neurosurgery, 50, 412-422.
- Levine, B. Roehrs, T., Stepanski, E., Zorick, F., & Roth, T. (1987). Fragmenting sleep diminishes its recuperative value. Sleep, 590-599.
- Lezak, M.D. (1978). Subtle sequelae of brain damage: Perplexity, distractibility, and fatigue.

American Journal of Physical Medicine, 57, 9-15.

Lishman, W.A. (1988). Physiogenesis and psychogenesis in the post-concussional syndrome. British Journal of Psychiatry, 153, 460-469.

Lubin, A., Hord, D., Tracy, M.L., & Johnson, L.C. (1976). Effects of exercise, bedrest, and napping on performance decrement during 40 hours. Psychophysiology, 13, 334-339.

Lugaresi, E., Medori, R., Montagna, P., Baruzzi, A., Cortelli, P., Lugaresi, A., Tinuper, P., Zuconi, M., & Gambetti, P. (1986). Fatal familial insomnia and dysautonomia with selective degeneration of thalamic nuclei. New England Journal of Medicine, 315, 997-1003.

Magnes, J., Moruzzi, G., & Pompeiano, O. (1961). Synchronization of the produced by low-frequency electrical stimulation of the region of the solitary tract. Archives Italiennes de Biologie, 99, 33-67.

Malloy, P., Bihle, A., Duffy, J., & Cimino, C. (1993). The orbitomedial syndrome. Journal of Clinical Neuropsychology, 8, 185-201.

Mancia, M. & Mariotti, M. (1985). Brain mechanisms of synchronous sleep. Clinical Neuropharmacology, 8 (Supplement 1), 41-48.

Mandleberg, I.A. & Brooks, D.N. (1975). Cognitive recovery after severe head injury. 1. Serial testing on the Wechsler Adult Intelligence Scale. Journal of Neurology, Neurosurgery, and Psychiatry, 38, 1121-1126.

Marascuilo, L.A. & McSweeney, M. (1977). Nonparametric and distribution-free methods for social sciences. Monterey: Brooks/Cole.

Markand, O.N. & Dyken, M.L. (1976). Sleep abnormalities in patients with brainstem lesions. Neurology, 26, 769-776.

Marshall, L.F., Becker, D.P., Bowers, S.A., Cayard, C., Eisenberg, H., Gross, C.R., Grossman, R.G., Jane, J.A., Kunitz, S.C., Rimel, R., Tabaddor, K., & Warren, J. (1983). The national coma data bank. Part I: Design, purpose, goals, and results. Journal of Neurosurgery, 59, 276-284.

Matthews, C.G. & Klove, H. (1964). Instruction manual for the Adult Neuropsychological Test Battery. Madison: University of Wisconsin Medical School.

Mazziotta, J.C. & Phelps, M.E. (1986). Positron emission tomography studies of the brain. In M.E. Phelps, J.C. Mazziotta, & H.R. Schelbert (Eds.), Positron emission

- tomography and autoradiography: Principles and applications for the brain and heart (pp. 493-579). New York: Raven Press.
- McCallum, W.C. & Cummins, B. (1973). The effects of brain lesions on the contingent negative variation in neurosurgical patients. Electroencephalography and Clinical Neurophysiology, 35, 449-456.
- McGinty, D. & Szymusiak, R. (1994). Neurobiology of sleep. In N.A. Saunders & C.E. Sullivan (Eds.), Lung biology in health and disease: Sleep & breathing (Vol. 71) (2nd ed.) (pp. 1-26). New York: Marcel Dekker Inc.
- McLean, A., Temkin, N.R., Dikmen, S., & Wyler A.R. (1983). The behavioral sequelae of head injury. Journal of Clinical Neuropsychology, 5, 361-376.
- McMillan, T.M. & Glucksman, E.E. (1987). The neuropsychology of moderate head injury. Journal of neurology, Neurosurgery, and Psychiatry, 50, 393-397.
- Mesulam, M.M. (1985). Principles of behavioral neurology. Philadelphia: F.A. Davis Co.
- Miller, E. (1970). Simple and choice reaction time following severe head injury. Cortex, 6, 121-127.
- Miller, E. & Cruzat, A. (1981). A note on the effects of irrelevant information on task performance after mild and severe head injury. British Journal of Social and Clinical Psychology, 20, 69-70.
- Miller, J.D. (1987). Brain edema in human head injury. In F. Cohadon, A Baethmann, K.G. Go, & J.D. Miller (Eds.), Traumatic brain edema (pp. 99-122). Padova: Liviana Press.
- Miller, J.D., Becker, D.P., Ward, J.D., Sullivan, H.G., Adams, W.E., & Rosner, M.J. (1977). Significance of intracranial hypertension in severe head injury. Journal of Neurosurgery, 47, 503-516.
- Miller, R.G. (1966). Simultaneous statistical inference. New York: McGraw-Hill.
- Milner, B. (1963). Effects of different brain lesions on card sorting: The role of the frontal lobes. Archives of Neurology, 9, 90-100.
- Monk, T.H. (1991) (Ed.). Sleep, sleepiness, and performance. New York: John Wiley & Sons Ltd.
- Moruzzi, G. & Magoun, H.W. (1949). Brain stem reticular formation and activation of the . Electroencephalography and Clinical Neurophysiology, 1, 455-473.

- Mountain, M.A. & Snow, W.G. (1993). Wisconsin Card Sorting Test as a measure of frontal pathology: A review. The Clinical Neuropsychologist, 7, 108-118.
- Nehemkis, A.M. & Lewinsohn, P.M. (1972). Effects of left and right cerebral lesions in the naming process. Perceptual and Motor Skills, 35, 787-798.
- Nedd, K., Sfakianakis, G., Ganz, W., Uricchio, B., Vernberg, D., Villanueva, P., Jabir, A.M., Bartlett, J., & Keena, J. (1993). ^{99m}Tc-HMPAO SPECT of the brain in mild to moderate traumatic brain injury patients compared with CT - a prospective study. Brain Injury, 7, 469-479.
- Neisser, Y. (1967). Cognitive psychology. New York: Appleton-Century-Crofts.
- Norrman, B. & Svahn, K. (1961). A follow-up study of severe brain injuries. Acta Psychiatrica Scandinavica, 37, 236-264.
- Oddy, M., Coughlan, T., Tyerman, A., & Jenkins, D. (1985). Social adjustment after closed-head injury: A further follow-up seven years after injury. Journal of Neurology, Neurosurgery, and Psychiatry, 48, 564-568.
- Ommaya, A.K. (1983). Workshop organized by the National Highway Traffic Safety Administration. U.S.A.
- Ommaya, A.K. & Gennarelli, T.A. (1974). Cerebral concussion and traumatic unconsciousness: Correlation of experimental and clinical observations on blunt head injuries. Brain, 97, 633-654.
- Ommaya, A.K., Grubb, R.L., & Naumann, R.A. (1971). Coup and contre-coup injury: Observations on the mechanics of visible brain injuries in the rhesus monkey. Journal of Neurosurgery, 35, 503-516.
- Oppenheimer, D.R. (1968). Microscopic lesions in the brain following head injury. Journal of Neurology, Neurosurgery, and Psychiatry, 31, 299-306.
- O'Shanick, G.J. (1986). Neuropsychiatric complications in head injury. Advances in Psychosomatic Medicine, 16, 173-193.
- Parkinson, D., Stephensen, S., & Phillips, S. (1985). Head injuries: A prospective, computerized study. The Canadian Journal of Surgery, 28, 79-83.
- Passouant, P., Cadilhac, J., Delange, M., Baldy-Moulinier, M., & El Kassabgui, M. (1965). Different electrical stages and cyclic organization of post-traumatic comas: Polygraphic recordings of long duration. Electroencephalography and Clinical Neurophysiology, 18, 726P.

- Patten, S.B. & Lauderdale, W.M. (1992). Delayed sleep phase disorder after traumatic brain injury. Journal of the American Academy of Child and Adolescent Psychiatry, 31, 100-102.
- Peach, R.K. (1992). Factors underlying neuropsychological test performance in chronic severe traumatic brain injury. Journal of Speech and Hearing Research, 35, 810-818.
- Peterson, L.R. & Peterson, M.J. (1959). Short-term retention of individual verbal items. Journal of Experimental Psychology, 58, 193-198.
- Ponsford, J. & Kinsella, G. (1992). Attentional deficits following closed-head injury. Journal of Clinical and Experimental Neuropsychology, 14, 822-838.
- Posner, M.I. & Boies, S.W. (1971). Components of attention. Psychological Review, 78, 391-408.
- Posner, M.I. & Rafal, R.D. (1987). Cognitive theories of attention and the rehabilitation of attentional deficits. In M.J. Meier, A.L. Benton, & L. Diller (Eds.), Neuropsychological rehabilitation. Edinburgh: Churchill Livingstone.
- Prigatano, G.P. (1986) (Ed.). Neuropsychological rehabilitation after brain injury. Baltimore: Johns Hopkins University Press.
- Prigatano, G.P. (1989). Work, love and play after brain injury. Bulletin of the Menninger Clinic, 53, 414-431.
- Prigatano, G.P., Stahl, M.L., Orr, W.C., & Zeiner, H.K. (1982). Sleep and dreaming disturbances in closed head injury patients. Journal of Neurology, Neurosurgery, and Psychiatry, 45, 78-80.
- Pudenz, R.H. & Shelden, C.H. (1946). The lucite calvarium - A method for direct observation of the brain; II. Cranial trauma and brain movement. Journal of Neurosurgery, 3, 487-505.
- Rechtschaffen, A. & Kales, A. (1968). A manual of standardized terminology, techniques, and scoring system for sleep stages of human subjects. Washington: Public Health Service, U.S. Government Printing Office.
- Reitan, R.M. (1958). Validity of the Trail Making Test as an indication of organic brain damage. Perceptual and Motor Skills, 8, 271-276.
- Reitan, R.M. & Davison, L.A. (1974). Clinical neuropsychology: Current status and applications. New York: Winston/Wiley.
- Richardson, G.S., Carskadon, M.A., Flagg, W., Van den Hoed, J., Dement, W.C., &

- Mitler, M.M. (1978). Excessive daytime sleepiness in man: Multiple sleep latency measurement in narcoleptic and control subjects. Electroencephalography and Clinical Neurophysiology, 45, 621-627.
- Richardson, J.T.F. (1990). Clinical and neuropsychological aspects of closed head injury. New York: Taylor & Francis.
- Rizzo, P.A., Amabile, G., Caporali, M., Spadaro, M., Zanasi, M., & Morocutti, C. (1978). A CNV study in a group of patients with traumatic head injuries. Electroencephalography and Clinical Neurophysiology, 45, 281-285.
- Robinson, A.L., Heaton, R.K., Lehman, R.A.W., & Stilson, D.W. (1980). The utility of the Wisconsin Card Sorting Test in detecting and localizing frontal lobe lesions. Journal of Consulting and Clinical Psychology, 48, 605-614.
- Ron, S., Algom, D., Hary, D., & Cohen, M. (1980). Time-related changes in the distribution of sleep stages in brain injured patients. Electroencephalography and Clinical Neurophysiology, 48, 432-441.
- Rosvold, H.E. & Mirsky, A. (1964). Symposium on the frontal granular cortex and behavior, The Pennsylvania State University. Unpublished study. In J.M. Warren & K. Akert (Eds.), The frontal granular cortex and behavior, (pp. 332-334). New York: McGraw-Hill.
- Ruesch, J. (1944a). Intellectual impairment in head injuries. American Journal of Psychiatry, 100, 480-496.
- Ruesch, J. (1944b). Dark adaptation, negative after images, tachistoscopic examinations, and reaction time in head injuries. Journal of Neurosurgery, 1, 243-251.
- Russell, W.R. (1971). The traumatic amnesias. London: Oxford University Press.
- Sangal, R.B. & Sangal, J.M. (1994). Closed head injury, excessive daytime sleepiness, and abnormal visual P300 brain topography. APSS.
- Schacter, D.L. (1977). theta waves and psychological phenomena: A review and analysis. Biological Psychology, 5, 47-82.
- Seigel, J.M. (1989). Brainstem mechanisms generating REM sleep. In M.H. Kryger, T. Roth, & W.C. Dement (Eds.), Principles and practice of sleep medicine (pp. 104-120). Philadelphia: W.B. Saunders Co.
- Shiffrin, R.M. & Schneider, W. (1977). Controlled and automatic human information processing: II. Perceptual learning, autonomic attending, and a general theory.

Psychological Review, 84, 127-190.

- Smith, A. (1973). Symbol Digit Modalities Test. Los Angeles: Western Psychological Services.
- Snow, R.B., Zimmerman, R.D., Gandy, S.E., & Deck, M.D.F. (1986). Comparison of magnetic resonance imaging and computed tomography in the evaluation of head injury. Neurosurgery, 18, 45-52.
- SPSS (1990). Reference guide. Chicago: SPSS incorporated.
- Stepanski, E., Zorick, F., Roehrs, T., Young, D. & Roth, T. (1988). Daytime alertness in patients with chronic insomnia compared with asymptomatic control subjects. Sleep, 11, 54-60.
- Sterman, M.B. & Clemente, C.D. (1962). Forebrain inhibitory mechanisms: Cortical synchronization induced by basal forebrain stimulation. Experimental Neurology, 6, 91-102.
- Strich, S.J. (1956). Diffuse degeneration of the cerebral white matter in severe dementia following head injury. Journal of Neurology, Neurosurgery, and Psychiatry, 19, 163-185.
- Strich, S.J. (1961). Shearing of nerve fibers as a cause of brain damage due to head injury. Lancet, 2, 443-448.
- Strich, S.J. (1970). Lesions in the cerebral hemispheres after blunt head injury. In S. Sevitt & H.B. Stoner (Eds.), The Pathology of Trauma. London: BMA House.
- Stroop, J.R. (1935). Studies of interference in serial verbal reactions. Journal of Experimental Psychology, 18, 643-662.
- Stuss, D.T. (1987). Contribution of frontal lobe injury to cognitive impairment after closed head injury: Methods of assessment and recent findings. In H.S. Levin, J. Grafman, & H.M. Eisenberg (Eds.), Neurobehavioral recovery from head injury (pp. 166-177). New York: Oxford University Press.
- Stuss, D.T., Ely, P., Hugenholtz, H., Richard, M.T., LaRochelle, S., Poirier, C.A., & Bell, I. (1985). Subtle neuropsychological deficits in patients with good recovery after closed head injury. Neurosurgery, 17, 41-47.
- Stuss, D.T., Stethem, L.L., Hugenholtz, H., Picton, T., Pivik, J., & Richard, M.T. (1989). Reaction time after head injury: Fatigue, divided and focus attention and consistency of performance. Journal of Neurology, Neurosurgery, and Psychiatry, 52, 742-748.

- Stuss, D.T., Stethem, L.L., Hugenholtz, H., & Richard, M. T. (1989). Traumatic brain injury: A comparison of three clinical tests, and analysis of recovery. The Clinical Neuropsychologist, 3, 145-156.
- Suffield, J.B. (1985). The cognitive psychophysiology of diffuse damage: Contingent Negative Variation (CNV) evidence of over- and underprocessing after severe closed head injury. Ph.D. thesis. Carleton University.
- Tabbador, K., Mattis, S., & Zazula, T. (1984). Cognitive sequelae and recovery course after moderate and severe head injury. Neurosurgery, 14, 701-708.
- Teasdale, G. & Jennett, B. (1974). Assessment of coma and impaired consciousness. Lancet, 2, 81-84.
- Teasdale, G. & Mendelow, D. (1984). Pathophysiology of head injuries. In N. Brooks (Ed.), Closed head injury: Psychological, social, and family consequences (pp. 4-36). New York: Oxford University Press.
- Tebano, M.T., Cameroni, M., Gallozzi, G., Loizzo, A., Palazzino, G., Pezzini, G., & Ricci, G.F. (1989). EEG spectral analysis after minor head injury in man. Electroencephalography and Clinical Neurophysiology, 70, 185-189.
- Teuber, H.L., Battersby, W.S., & Bender, M.B. (1951). Performance of complex visual tasks after cerebral lesions. Journal of Nervous and Mental Disease, 114, 413-429.
- Thatcher, R.W., Walker, R.A., Gerson, I., & Geisler, F.H. (1989). EEG discriminant analysis of mild head trauma. Electroencephalography and Clinical Neurophysiology, 73, 94-106.
- Thomas, C. (1977). Deficits of memory and attention following closed head injury. M.Sc. thesis. Oxford University.
- Tromp, E. & Mulder, T. (1991). Slowness of information processing after traumatic head injury. Journal of Clinical and Experimental Neuropsychology, 13, 821-830.
- Valley, V. & Broughton, R.J. (1983). The physiological (EEG) nature of drowsiness and its relation to performance deficits in narcoleptics. Journal of Clinical and Experimental Neuropsychology, 55, 243-251.
- Van Zomeren, A.H. (1981). Reaction time and attention after closed head injury. Lisse: Swets & Zeitlinger.
- Van Zomeren, A.H. & Brouwer, W.H. (1987). Head injury and concepts of attention. In H.S. Levin, J. Grafman, & H.M. Eisenberg (Eds.), Neurobehavioral recovery from

head injury (pp. 398-415). New York: Oxford University Press.

- Van Zomeren, A.H., Brouwer, W.H., & Deelman, B. G. (1984). Attention deficits: The riddles of selectivity, speed, and alertness. In D.N. Brooks (Ed.), Closed head injury: Psychological, social, and family consequences. Oxford: Oxford University Press.
- Van Zomeren, A.H. & Deelman, B.G. (1976). Differential effects of simple and choice reaction after closed head injury. Clinical Neurology & Neurosurgery, 79, 81-90.
- Van Zomeren, A.H. & Deelman, B.G. (1978). Long-term recovery of visual reaction time after closed head injury. Journal of Neurology, Neurosurgery, and Psychiatry, 41, 452-457.
- Van Zomeren, A.H. & Van Den Burg, W. (1985). Residual complaints of patients two years after severe head injury. Journal of Neurology, Neurosurgery, and Psychiatry, 48, 21-28.
- Villablanca, J.R., Marcus, R.J., & Olmstead, C.E. (1976). Effects of caudate nuclei or frontal cortex ablations in cats. II. Sleep-wakefulness, , and motor activity. Experimental Neurology, 53, 31-50.
- Volk, S., Simon, O., Schulz, H., Hansert, E., & Wilde-Fray, J. (1984). The structure of wakefulness and its relationship to daytime sleep in narcoleptic patients. Electroencephalography and Clinical Neurophysiology, 57, 119-128.
- Von Economo, C. (1930). Sleep as a problem of localization. Journal of Nervous and Mental Diseases, 71, 249-259
- Webb, W.B. & Agnew, H.W. Jr. (1974). The effects of chronic limitation of sleep length. Psychophysiology, 11, 265-274.
- Webb, M. & Kirker, J. G. (1981). Severe post-traumatic insomnia treated with L-5-hydroxytryptophan. The Lancet, 1365-1366.
- Wechsler, D. (1944). The measurement of adult intelligence (3rd ed.). Baltimore: Williams & Wilkins.
- Wechsler, D. (1945). A standardized memory scale for clinical use. Journal of Psychology, 19, 87-95.
- Wechsler, D. (1981). Manual for the Wechsler Adult Intelligence Scale - Revised. New York: The Psychological Corporation.
- Wechsler, D. (1987). Manual for the Wechsler Memory Scale - Revised. New York: The

Psychological Corporation.

- Wilkinson, R.T. & Stretton, M. (1971). Performance after awakening at different times of night. Psychonomic Science, 23, 283-285.
- Wilkinson, R.T. (1992). The measurement of sleepiness. In R.J. Broughton & R.D. Ogilvie (Eds.), Sleep, Arousal, and Performance (pp. 254-265). Boston: Birkhauser.
- Will, R.G., Young, J.P.R., & Thomas, D.J. (1988). Kleine-Levin Syndrome: Report of two cases with onset of symptoms precipitated by head trauma. British Journal of Psychiatry, 152, 410-412.
- Wilson, J.L. & Wyper, D. (1992). Neuroimaging and neuropsychological functioning following closed head injury: CT, MRI, and SPECT. Journal of Head Trauma Rehabilitation, 7, 29-39.
- Winogron (1986). Reaction time and speed-accuracy tradeoff strategies in head-injured and normal children. Ph.D. thesis. Carleton University.
- Yerkes, R.M. & Dodson, J.D. (1908). The relation of strength of stimulus to rapidity of habit formation. Journal of Comparative Neurology and Psychology, 18, 459-482.

Appendix A

Sleep Questionnaire for TBI Subjects

CONFIDENTIAL

Initials:

Night time

What time do you usually go to bed on weekdays? _____ weekends?

What time do you usually get up on weekdays?_____weekends?_____

How many hours do you sleep per night?

less than 7 7-8 more than 8 ; give number:

Do you have problems falling asleep?

rarely **occasionally** **frequently** **always**

(1-2/week) (3-6/week) (7/week)

How long do you take to fall asleep (on average)? _____

Do you have difficulty breathing at night? Yes No

Have you been told that you snore while asleep? Yes _____ No _____

Are you aware or have others told you that you are experiencing problems such as:

leg jerks **Yes** **No**

	Yes	No
talking while asleep		

	Yes	No
walking while asleep		

grinding teeth while asleep Yes No _____

	Yes	No
wetting the bed while asleep		

having disturbing dreams	Yes	No

Do you feel refreshed when you awaken in the morning?

rarely____ occasionally____ frequently____ always____

In general, are you satisfied with the quality of your sleep? Yes____ No____

Day time

Do you feel sleepy during the day?

rarely____ occasionally____ frequently____ always____

Do you nap on weekdays?

rarely____ occasionally____ frequently____ always____

Do you nap on weekends (or holidays)?

rarely____ occasionally____ frequently____ always____

If you nap, when do these naps occur during the day?

morning____ afternoon____ evening____

How long are your naps (on average)?_____

Do you feel refreshed after napping?

rarely____ occasionally____ frequently____ always____

Following your accident, have your sleep habits changed? Yes____ No____

If so, in what respect?_____

Occupation

What is your present occupation?

work____ what type?_____

school____ what type?_____

If you stay at home, what are your hobbies?_____

While performing during any of these occupations (i.e., work, school, or hobbies), do you have difficulty remaining alert?

rarely____ occasionally____ frequently____ always____

Medication and others

Are you taking medication? Yes____ No____

If so, for what purpose?_____

Please specify type and frequency of use_____

Do you take non-prescribed drugs (e.g., marijuana)? Yes____ No____

If so, specify type and frequency of use_____

Do you smoke? Yes____ No____

If so, quantity and frequency_____

Do you drink alcoholic beverages? Yes____ No____

If so, type, quantity, and frequency_____

Do you drink coffee and/or tea? Yes____ No____

If so, type, quantity, and frequency_____

How is your appetite in general?

little____ normal____ big____

Thank you for completing this questionnaire

Name:

Address:

Phone number:

Sex:

Age:

Date of birth:

Marital status:

Education:

Previous occupation:

Handedness:

Appendix B

Information for subjects

The purpose of this study you have been asked to participate in is to investigate sleep-wake states and performance in brain injured and control subjects.

First, you will come to the sleep laboratory on 2 consecutive nights for sleep EEG recording. The first night will serve as an adaptation night and to rule out sleep problems such as sleep apnea and periodic leg movements. Since you will be awakened at 7:00 in the morning, you should adjust your sleep time a few nights prior to coming to the lab (i.e., going to bed earlier or later) so you can adapt to this awakening schedule and obtain your normal amount of sleep. Bring with you whatever you normally wear to sleep. Electrodes will be applied on you to record your brain activity, respiration, and leg movements. Following the second night, you will remain in the lab until the end of the afternoon so repeated testing (including napping and simple reaction time task) can be performed. Breakfast will be provided. Please bring a lunch for noontime (a refrigerator will be available). Finally, a few weeks thereafter, a brief neuropsychological assessment lasting 1/2 day will be administered.

This study should improve our understanding of the nature of sleep-wakefulness patterns after brain injury. Furthermore, recommendations concerning strategies to minimize the effects of posttraumatic sleepiness could be formulated based upon these findings.

All matters pertaining to confidentiality and the protection of human rights will be conducted according to the hospital ethical principles for human experimentation.

END

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FIN