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Putting a Human Face to Severe Traumatic Brain Injury Research:

A Review of Neuropsychological Rehabilitation,

Obsessive-Compulsive Disorder and Caregiver Burden

with Respect to the Case of BP

A Thesis Presented in Partial Fulfilment of the Requirements
for the Degree of Master of Arts in Psychology at
Massey University

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2003

Abstract

The following thesis is a review of several issues relating to severe brain injury. The theories and research literature were also paired with the case study of a young man who had suffered a severe TBI three years ago, and the outcomes and rehabilitation that he has faced since and continued to face. The case study and theory can be read separately, but it is together that they may help to put a human face on the TBI literature. Rehabilitation principles and theories are described in order to present a picture of an ideal rehabilitation plan, and then contrasted by the case study to demonstrate the difficulties that are inherent in severe TBI. Despite careful adherence to rehabilitation principles, the rehabilitation process remains difficult and lengthy. The research literature regarding obsessive-compulsive disorder and TBI are discussed in reference to aspects of the current case study and impulse control disorders in general. The caregiver burden research literature, particularly with regard to that of TBI, is also reviewed, with reference again to the current case study. A plea is made for more flexible, functionally-relevant rehabilitation models, that attempt to take into account the true complexities involved in severe TBI rehabilitation. The use of case studies in future TBI research, along with the larger-sampled empirical studies, may aid our understanding of TBI and its rehabilitation from a more human real-life perspective.

Acknowledgements

Much thanks go to my supervisor Dr Janet Leathem. Her knowledge and experience were invaluable, and her enthusiasm contagious. Most of all, her belief that I had something important to contribute, and her insistence to ensure that I do so, was crucial to completing this thesis. Working with her has been a pleasure, and I hope to do so more in the future.

A big thank you also to Senior Clinical Psychologist Tracey Barnfield. Her supervision with the caregiving and social rehabilitation of BP has been assured, and has provided the skills, perspective and encouragement necessary for what has been a difficult yet rewarding task. I look forward to learning from her and continuing to work with her in the future.

I would like to thank BP's parents, for the permission of accessing their son's medical records, for the offering of all medical information they possessed, and for the great flexibility they have afforded me in the completion of this thesis. More than this however, I offer great thanks for opening their home to me, and trusting me in the continuing caregiving and guidance of their son. Their encouragement and help has been unending.

The many rehabilitation professionals who have contributed and continue to contribute to BP's rehabilitation are acknowledged.

Thank you to Regan for the constant support (and occasionally, the necessary push) throughout the completion of this thesis. I look forward to returning the favour. Much thanks to friends and family for their unwavering encouragement and support.

Last, but definitely not least, my thanks go to BP himself. Throughout the time working with him I have learned a lot about severe TBI and about myself. Thank you for allowing me to share your life with others in the hope of aiding understanding of severe TBI. It will always be appreciated.

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Chapter 1: Overview

The idea for this thesis grew from spending time with and caring for a young man with a severe traumatic brain injury (TBI), who demonstrates a wide range of problem behaviours associated with frontal lobe dysfunction.

On the 13th of November 1999, BP, then an 18-year-old male, fell eight metres from a roof, landing headfirst on concrete. He suffered a severe brain injury, and has since received extensive rehabilitation in hospital, at a private rehabilitation centre, and at home. Nevertheless, several problem behaviours have persisted 3 years after the injury, especially those regarding impulsivity and social inappropriateness. He has been known to (especially when fatigued) wander off without regard for personal safety, and thus requires constant supervision 24 hours a day. He currently lives at home with his parents. His attention, both sustained and selective, is severely limited, and his executive function (e.g., planning, organisation) is impaired. His insight into his own behaviour and limitations is impaired, and he has considerable difficulties with motivation and initiation.

BP's story is relevant to care professionals and psychologists dealing in the area of brain injury, because it puts a human face on the types of injuries and outcomes that can come as the result of a severe TBI. It is through closer examination of real-life cases that we can gain a truer and more functionally relevant understanding of the outcomes and rehabilitation involved in severe TBI.

Perhaps more importantly however, through illustrating a case such as this, a largely unrecognised phenomenon is explored. That is, despite careful adherence to

rehabilitation principles, and with the best efforts from a multi-disciplinary rehabilitation team, improvement in psychological sequelae can often be hard to come by. In fact, it is inherent in the nature of rehabilitation that sometimes the best intentions based in the best theories do not necessarily end up with the optimal outcome. For this reason, rehabilitation of severe TBI is a lengthy process, often continuing years after the initial injury. Given the variable nature of rehabilitation effectiveness, it is important to reconsider the nature of “successful” rehabilitation for the family, the rehabilitation professionals, and the client themselves. A clearer understanding of this complex and varying relationship between rehabilitation interventions and outcomes will help all those involved in the rehabilitation process to not lose hope when interventions do not appear to be benefiting the TBI individual at a functional level. This is, in fact, a natural part of the rehabilitation process.

This thesis will present reviews of the main aspects critical to the understanding of severe TBI, with the story of BP threaded throughout to illustrate and emphasize these features. BP's story may be read separately, as can the review, but that reading both together provides the most compelling evidence for the difficulties inherent in TBI rehabilitation.

Chapter 2 discusses the epidemiology and outcomes of TBI, with particular attention paid to those of severe TBI. Within this chapter is a discussion of the case of BP, including the details of his injury, a history of his early recovery, and the nature of his current impairments.

Chapter 3 discusses the research literature regarding brain injury rehabilitation. This is followed by a discussion of the history of BP's multi-disciplinary rehabilitation, with

particular attention paid to the behaviour modification and behaviour management strategies employed to deal with his impairments on a functional everyday level. In an attempt to demonstrate the difficulties inherent in rehabilitation for severe TBI, both the theories and principles of rehabilitation that research indicates to be most effective, and the history of BP's rehabilitation interventions are compared and considered together.

One aspect particular to BP's case that appears to have come as a direct result of his brain injury involves behaviour similar in nature to those diagnosed with obsessive-compulsive disorder (OCD). Since the injury, BP has found he has a certain affinity with the number three in many facets of his life. However, BP's number-related OCD-like behaviours differ from idiopathic OCD in many ways (particularly the fact that no anxiety is present). Chapter 4 discusses the behaviours that BP demonstrates that are reminiscent of idiopathic OCD, and details the research into OCD following TBI, and into the brain mechanisms associated with the development of idiopathic OCD. It is hoped that by making such links between OCD and specific lesions, especially in a TBI case study such as this, our understanding of the neural mechanisms involved in OCD may be enhanced. It also highlights the blurring between compulsions, perseverative behaviours characteristic of frontal lobe injury, and impulse control disorders. The investigation of a possible overlap between impulse control disorders and OCD is discussed, in order to provide a better understanding of BP's impulsive behaviours regarding his constant need for stimulation (e.g., coffee, sugar, alcohol, attention from others).

Chapter 5 investigates the research of caregiver burden. A focus of this review is the nature of caregiver burden for those caring for a person with severe brain injury, and

what particular aspects of the TBI individual (e.g., specific behaviours: impatience, aggression, rapid mood change) have been found to be associated with higher levels of psychological distress. A particular focus is placed on the effect that the "over-the-top" behaviour exhibited by such clients as BP has on their caregivers. BP sometimes refers to me as being like a "big brother" or a best friend and feels compelled to hug often. At the same time fluctuations in mood will often result in shouting, pretending to be angry, or other behaviours in great contrast only minutes or seconds afterwards. Such extremes of closeness and inappropriateness are taxing on caregivers, both parents and otherwise. It is hypothesised that those caregivers who are caring for TBI individuals who demonstrate rapid fluctuations in expressed mood, or emotional incontinence, are likely to have higher levels of psychological distress (as these are the behaviours that have been particular distressing for myself as a caregiver of BP). Caregiver burden as experienced by BP's father, and the nature of resilience, are also discussed.

It is important to keep in mind that these aspects of BP don't occur in isolation. There are also many other issues that warrant attention with BP that aren't being focused on in this thesis. An attempt to bring together the ideas in this thesis and put them in the context of BP's whole person and rehabilitation is made in the conclusion (Chapter 6). The difficulties inherent in rehabilitation for the severely brain-injured are re-emphasised. An examination of a case study such as this, can demonstrate the true complexity that is traumatic brain injury and its rehabilitation process.

Chapter 2: Traumatic Brain Injury

Epidemiology

In industrialised nations traumatic brain injury (TBI) is very common with annual incidence rates ranging between 132 and 430 per 100,000. New Zealand figures from acute hospital care (Traumatic Brain Injury Rehabilitation Guidelines, 1998) suggest that about 6,000 people are afflicted with TBI each year, equating to a rate of about 180 per 100,000 per year. However, the general consensus is that incidence rates do not reflect the true extent of TBI, as most sufferers from mild brain injuries are not hospitalised (Agency for Health Care Policy and Research, 1998). Extrapolation from United States data reporting an annual rate of 618 per 100,000 mild to moderate TBI (Sosin, Snieszek, & Thurman, 1996), would indicate that in New Zealand, the number of people who suffer from a brain injury (mild, moderate or severe) could be in excess of 30,000 annually.

All studies investigating the ages of those who suffer traumatic brain injuries have shown that the highest risk age group is 15-24 years (Kraus, McArthur, Silverman & Jayaraman, 1996). Typically, the risk of brain injury is quite high under 5 years, decreases up to early adolescence, then peaks sometime after age 15, only to slowly decrease again from the early 20's on into middle age, and increase again from about 60 years of age, largely as a result of falls (Kraus, McArthur, Silverman & Jayaraman, 1996). It is noteworthy that multiple significant life changes occur during late adolescence and young adulthood, the very point in life when traumatic brain injuries are most common (e.g., career choices, moving out of home and becoming more independent, the development of sexual relations and identity). Accordingly age-

specific issues must be taken into account when contemplating the care and rehabilitation of those who suffer brain injuries.

Gender is another important risk factor for TBI. All incidence reports have shown that males are far more likely to suffer a TBI than females, with most U.S. data showing a rate ratio of 2.0 to 2.8 (Kraus, McArthur, Silverman & Jayaraman, 1996). A study of the incidence of TBI in South Australia revealed similar results, with males 2.3 times more likely to suffer a TBI than females (Hillier, Hiller & Metzer, 1997). Reasons for this difference in incidence rates can be largely attributed to the high number of men riding motorcycles (particularly susceptible to accidents, Kraus et al., 1984) and being in motor vehicle accidents involving speed and/or alcohol, as well as the predominance of men in violent assaults and involved in sports injuries. This discrepancy between the rates of TBI of males and females is one that needs to be addressed in the prevention of TBI. Drink-drive campaigns targeted at young males are aimed at doing exactly that.

Causes of TBI

The causes of traumatic brain injury are numerous, but several trends are evident. Motor vehicle accidents account for approximately half of all injuries in most studies (Naugle, 1990) and a disproportionately high number of these are motorcycle accidents. A study by Kraus et al. (1984) of the San Diego area found that motorcyclists accounted for 21% of all motor vehicle related TBI, compared to the 62% that were motor vehicle occupants, 12% pedestrians and 6% bicyclists. Falls are the second leading cause of head trauma, and are especially common amongst the very young and the elderly. Naugle reported that falls accounted for approximately

28% of the epidemiological literature. Assaults and injuries from sports games or recreational activities make up the majority of other causes of TBI.

It has been firmly established that the risk of suffering a TBI is greatly increased when alcohol is involved (Smith & Kraus, 1988, cited in Kraus, McArthur, Silverman & Jayaraman, 1996). One study (Kraus, Morgenstern, Fife, Conroy & Nourjah, 1989) found that, of those diagnosed with a brain injury and tested for blood alcohol concentration (BAC), 56 percent had a positive result. The number of those who tested positive for BAC varied according to severity of injury, with alcohol found to be more likely to be involved in mild brain injuries (71%) than in moderate or severe injuries (49%). However, the study also demonstrated the possible selection bias by emergency hospital staff of who gets tested for BAC, with males, young adults and those injured from falls, less likely to be tested.

Severity

Traumatic brain injuries are usually described in terms of ‘mild’, ‘moderate’ or ‘severe’. Two measures commonly referred to when defining TBI severity are the Glasgow Coma Scale (GCS) and Posttraumatic Amnesia (PTA). The GCS includes three scales, measuring eye opening (1-4), verbal response (1-5) and motor response (1-6), as demonstrated at the time of the injury. Therefore, a measure of 3/15 would be given both to someone who is in the deepest state of coma, or is in fact dead. A score on the GCS of 13 or greater is typically referred to as a ‘mild’ brain injury, with a score between 9 and 12 indicating a ‘moderate’ injury, and a score of less than 8 being referred to as ‘severe’.

PTA refers to the time from injury to the recovery of a continuous memory thread (although memory impairment often continues past this point) (Traumatic Brain Injury Rehabilitation Guidelines, 1998). Using PTA as a point of reference for brain injury severity, it is common that less than 24 hours is referred to as 'mild', a day to a week as 'moderate', and over seven days as a 'severe' brain injury. While these cut-off points between severity levels are largely arbitrary, regardless of the measure used, the consequences generally become greater as severity increases (Traumatic Brain Injury Rehabilitation Guidelines, 1998).

Severe TBI (according to either Glasgow Coma Scale or PTA definition) comprises approximately 10% of all TBIs (Hillier, Hiller & Metzer, 1997). While this a relatively small percentage, severe TBI accounts for a disproportionately high percentage of mortality, morbidity and costs of care associated with traumatic brain injury (Gray, 2000). Due to the greater consequences of a severe brain injury, those with severe TBI tend to make up the bulk of people referred to TBI rehabilitation programmes; e.g., Wong, Dornan and Schentag (1993) reported that 91.6% of admissions to an Ontario TBI rehabilitation programme were sufferers of a severe TBI. Burke et al. (2000) emphasised that over the last 15-20 years those TBI sufferers at each end of the severity spectrum are least likely to receive adequate rehabilitation, with the very severe often falling between the cracks because they are not considered as likely to benefit from rehabilitation. Considering the huge costs involved in the care of the survivors of severe TBI (especially as severe TBI survival rates may be increasing as a result of improvements in acute medical care; Gray, 2000), the relatively low percentage of 10% is not a fair indicator of the extent of the problem.

Immediate outcomes

The immediate outcomes of a traumatic brain injury are dependent on many things, including the type of injury (e.g., whether or not it was of acceleration-deceleration type, such as a fall or most motor vehicle accidents), the velocity of impact, whether it was a closed or penetrating head injury, and whether the brain injury was diffuse or focal.

Closed head injury (CHI), in which the skull is intact, is far more common than an open head injury, where the bulk of the brain injury is caused by an object penetrating the skull (Alabama Department of Rehabilitation Services, 2002). CHI results in two categories of brain injury: primary brain injury, occurring immediately at the time of impact, and secondary injury, which refers to the many different processes that the brain undergoes in the hours, days and weeks proceeding the primary damage. Primary brain injury is usually permanent and largely unresponsive to therapeutic measures (Pang, 1989). Hence, it generally constitutes the limiting factor for neurological recovery.

Primary brain injury

Primary brain injury can be caused by mechanisms independent of acceleration, or by forces that involve acceleration and deceleration to the head. The latter usually results in much more severe brain injury than if a force is directed at a fixed skull (Pang, 1989). The majority of head injuries involve some type of acceleration to the head (Pang). For instance, in a fall, the accelerating forehead hits an unyielding surface, the skull decelerates to a halt, but the brain keeps moving forward until it is stopped by slamming into the rough surfaces of the front of the skull. This lesion can be reasonably focal (although not as focal as say, the injury that would be caused by a

golf ball hitting a head at high velocity), but is often accompanied by a more diffuse or widespread damage, as distortion of the shape of the brain produces shearing strains. These shearing strains lead to what Stritch (1956, cited in Levin, 1990) coined diffuse axonal injury (DAI). A closer look at the differences between focal and diffuse injuries (especially of DAI) follows.

Focal versus diffuse injury

Incidence rates for focal or diffuse brain injuries are difficult to obtain, as the difference between the two is often not clear-cut; i.e., diffuse brain injuries do not affect all brain structures equally, and it is very rare to find a focal injury (especially a severe one) where some diffuse reactions did not take place (Lezak, 1995). Behavioural changes following TBI are largely affected by whether the damage is widespread, or if it is not, which brain structures have been most affected. Focal brain injuries can cause significant neuropsychological impairments, however, often other capacities are very well preserved. While focal brain lesions appear to affect cognition and behaviour permanently and negatively (Prigatano, 1999), losses can often be compensated for to some extent by those aspects of cognitive function that remain largely intact.

Nonfocal or diffuse brain injury also occurs in most closed head injuries, especially those sustained in conditions of rapid acceleration and/or deceleration, such as motor vehicle accidents or falls (Lezak, 1995). Gennarelli (1994, cited in Wallesch et al., 2001) emphasises that a focal lesion (which mainly involve the cortical grey matter) is just one of three mechanisms by which blunt head injury may cause traumatic brain damage. The others are diffuse axonal injury (DAI) and secondary injury caused by oedema and space occupying lesions. DAI refers to stretched and sheared axons from

impact, and the generalised damage that occurs as a result of torn axonal fibres, damage to supportive glial cell structure, and the degeneration of neuronal fibres distal to the focal area of shearing or contusion (Bigler, 1990). While distributed throughout the brain, severe cases of DAI tend to display focal lesions in the corpus callosum and in the dorsolateral quadrants of the rostral brainstem, as well as widespread damage to axons evident at a microscopic level (Graham, 1995).

Secondary brain injury

Secondary injury follows the initial damage of focal lesions and diffuse axonal injury. These include shock in areas remote to the damage, oedema or swelling of traumatised tissue (where inflowing fluid to the damaged area increases pressure and as a result depresses neuronal function), reductions in blood flow, changes in neurotransmitter release, reduction in glucose usage, and autoneurotoxicity (Kolb & Whishaw, 1996). Figure 1 shows the many types of, and mechanisms underlying, secondary brain injury.

A large part of the reason for the damage following the initial injury can be attributed to an increase in intracranial pressure (ICP). Several epiphenomena contribute to the increase in ICP, including brain swelling or oedema, and the development of haematomas (as well as the ICP increase that can occur at a primary injury level, e.g., from a depressed skull fracture). An increase in fluid or blood in the brain does not cause a huge increase in ICP at first, as compensatory mechanisms (e.g., cerebrospinal fluid being pushed into the spinal subarachnoid space) help to relieve the pressure.

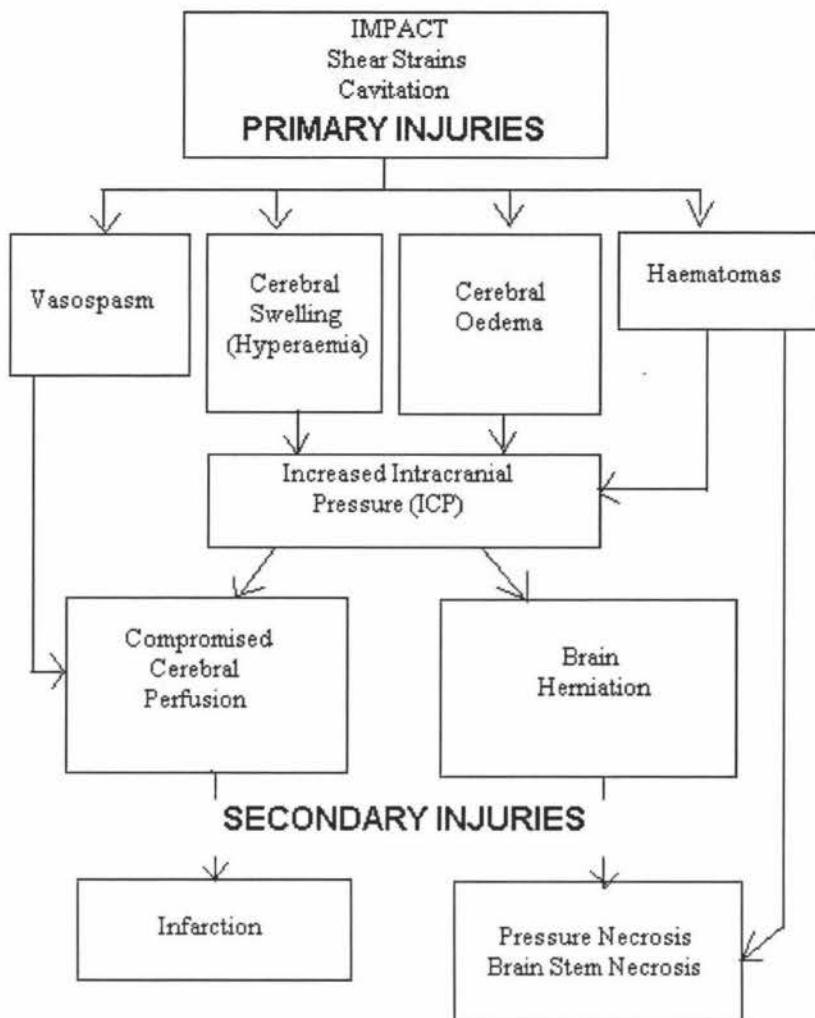


Figure 1. The process from primary brain injury to secondary brain injury (adapted from Pang, 1989, p.10).

This ability to counter initial increases in intracranial pressure is called 'compliance' (Pang, 1989). However, these mechanisms operate only to a certain point, and at the point when the brain is no longer able to compensate for this extra fluid, ICP begins to increase at a faster and faster rate (called 'elastance'; Pang). Raised ICP is potentially fatal, as it may push the brain down onto the brainstem, suppressing neuronal function in this vital area. Raised ICP can also shift the brain, tearing and twisting axons in much the same way as seen with diffuse axonal injury (DAI). Often cerebral blood flow is disrupted too, and while the brain is usually able to 'autoregulate' its own fuel

supply when changes occur to cardiovascular function, brain trauma and ischaemia can render autoregulation inactive. Thus, after a head injury, small increases in ICP (e.g., from 20 mm Hg to 25 mm Hg) can reduce cerebral blood flow enough to cause ischaemic encephalopathy (Pang). In summary, the effect of the epiphenomena that follow primary brain injury is to increase ICP, which in turn causes tissue death. A closer look at these epiphenomena follows.

Oedema (Swelling)

Cerebral oedema occurs frequently following head injury (Graham, 1995). In fact, it is the most common form of secondary injury (Bigler, 1990). It basically refers to the swelling of brain tissue following trauma (Kolb & Whishaw, 1996), or the increase in the water content of the brain (Pang, 1989). Cerebral oedema occurs in the area surrounding the lesion but it can also affect distal areas through pressure and other mechanisms (Kolb & Whishaw). Hence the effects of swelling can be both focal and diffuse (Naugle, Cullum, & Bigler, 1998).

Haematomas

While haematomas (resulting from cerebral haemorrhage) are often referred to as a type of primary brain injury, they also contribute to increasing intracranial pressure (ICP), thus leading to secondary injury. The outcomes are very different depending on the type of haemorrhage (i.e., rate and site of accumulation). An extradural haematoma forms when blood from torn meningeal blood vessels causes an enlarging haematoma to form between the dura mater (the outside membrane layer around the brain) and the skull, pushing against the brain, indenting and flattening it. Intradural haematomas include subdural haematomas (formed beneath the dura; usually more extensive due to freer movement of blood in the subdural space), intracerebral

haematomas (occurring within brain tissue and found in 16% of the Glasgow database; Graham, 1995), or a “burst” lobe (an intracerebral haemorrhage in conjunction with a subdural haematoma).

Of all intracerebral haematomas 5-10% are caused by a subarachnoid haemorrhage (SAH), where the blood is expelled into the subarachnoid space (Mitchell, 2002). This is the space between the pia mater, the fine layer of membranes clinging to the surface of the brain, and the adjacent layer, the arachnoid mater, so-called because of the spidery appearance of the blood vessels within it. This haemorrhage is sometimes followed by serious complications. One of the most serious of these is vasospasm, where the arteries react to the blood around them by going into spasm, resulting in ischaemia (loss of blood and oxygen) to the parts of the brain that the artery supplies (Ogden, 1996). This vasospasm-related ischaemia has been identified as the leading cause of death and disability following a subarachnoid haemorrhage (Kassel, Sasaki, Colohan, & Nazar, 1985, cited in Dowling & Dacey, 1996). Also of great concern is the possibility of re-haemorrhage, which is often fatal (Dowling & Dacey, 1996). While head trauma is the most common cause of SAH (Martin et al., 1992), the effects of the initial cerebral trauma usually overshadow any direct effects of the haemorrhage (Dowling & Dacey). Other possible complications following SAH include hydrocephalus (with the resultant problematic increase in intracranial pressure), and epilepsy, which has been shown to develop in about 15% of those who suffer a SAH (Rose & Sarner, 1965, cited in Dowling & Dacey).

Vasospasm

Vasospasm can also occur as a direct result of a traumatic brain injury, rather than as a consequence of a haemorrhage. It refers to the sudden contraction of the muscle

within the wall of a blood vessel, which decreases its internal diameter, causing a decrease in blood flow, but an increase in “systemic vascular resistance” (Online Medical Dictionary, 1997). Hence, it has a much more direct effect on cerebral blood flow than that of oedema or haematoma, which affect blood flow through the resultant increase in intracranial pressure (ICP).

While immediate outcomes are extremely important, particularly with regard to acute care, the rehabilitation of those who have suffered a traumatic brain injury is typically more concerned with the long-term cognitive, emotional, behavioural and psychosocial outcomes of TBI. These long-term outcomes of TBI will be discussed in general, before focusing on the different outcomes that result from more specific types of brain injury (e.g., severity, age, closed head injury, subarachnoid haemorrhage).

BP: The injury

On November 13th, 1999, BP, then aged 18, climbed onto a roof while intoxicated at a party and was dancing on it when he lost his footing, and fell eight metres onto concrete, landing on his head. With acceleration of 9.8 m/s (i.e., gravity), his head was probably travelling at approximately 12.5 m/s, or 45 km/hr, at the point of impact¹. Whilst this is not as fast as many motor vehicle accidents, it may be reasonable to refer to this as a ‘high velocity’ impact.

He sustained a depressed fracture of the skull involving the right frontal and temporal bones. Small bone fragments were displaced intracranially. The fracture extended into the base of the skull to the left petrous temporal bone. The right

¹ Gravitational Potential Energy = Kinetic Energy

$9.8 \times \text{mass} \times \text{height} = \frac{1}{2} \times \text{mass} \times (\text{velocity})^2$

$9.8 \times \text{height} = \frac{1}{2} \times (\text{velocity})^2$

$(\text{velocity})^2 = 2 \times 9.8 \times 8 \text{metres}$

$(\text{velocity})^2 = 156.8$

$\text{velocity} = 12.5 \text{ m/s}$

$\text{velocity} = 45 \text{ km/hr}$ (multiply by 3.6 to convert to km/hr)

lateral orbital wall was depressed. Petechial haemorrhages were found in both frontal lobes and in the left basal ganglia, indicative of the diffuse axonal injury (DAI) that frequently occurs in high acceleration/ deceleration impact injuries of the sort that BP had sustained. There was a haemorrhage into both sylvian fissures at the base of the brain.

The overall result of the injury to BP's brain, was a frontal and right fronto-temporal laceration associated with a right forehead depressed skull fracture. At the scene of the accident his Global Coma Scale was recorded as 3/15. An initial CT scan showed bifrontal contusion, and there was a cerebral oedema and extensive sub-arachnoid haemorrhage. A subsequent CT scan 2 days later showed mild ventricular enlargement and some resolution of the haematoma and cerebral contusion.

As well as the injuries to the brain BP sustained a broken nose, a broken collarbone and lacerated thumb tendons. These other injuries, as well as his asthma, should be taken into account as possible factors contributing to the secondary brain damage (e.g., his oxygen intake may be depleted, resulting in more cell death from depleted cerebral glucose level and blood flow).

Initial Management

BP was stabilised in the Intensive Care Unit (ICU), and for the first few days was deeply unconsciousness. His right pupil was 4mm in diameter, while his left pupil was 5mm and unresponsive to light. There was an occasional flicker of movement on his left arm and leg, but no movement was observed on his right side. BP was managed in ICU under heavy sedation, antibiotics and intravenous fluids. A cerebrospinal fluid leak that had developed resolved itself within 3 or 4 days. Within the first week BP began to demonstrate a motor response to pain, but did not open his eyes during the first three weeks of his recovery.

Early Recovery

By the 20th of December, 2 1/2 weeks later, BP's father noted signs of attentiveness towards people and tasks, for varying lengths of time between a few minutes and 20 minutes. BP was able to turn pages of a book, and appeared to listen to stories. On occasion he could make a choice between two objects, and play some finger games, but as at that time was unable to reliably indicate yes or no in either a verbal or non-verbal way.

By the 23rd of December, BP was more wide-awake, attending to people in his room, and able to sit supported on the edge of his bed. He was able to take turns in passing or throwing a ball. Some purposeful (non-verbal) communication was beginning to emerge, such as withdrawing his hand and not relinquishing the ball. It was reported that when requested to verbalise, BP appeared to try to open his mouth, but no purposeful vocalisation was possible. BP's purposeful communication continued to improve, so that on December 29th he was observed to use tapping of his fingers to indicate yes or no. He would look left and right, tracking people as they moved. Ability to swallow was also returning (in attempting to drink water or eat dairy food), but was still difficult. By December 31st, BP could respond to simple commands and could move his limbs. When shown the letter B, it was observed that he could write his full name with his left hand. Function of his right hand (his dominant hand before the injury) was not at such a level to perform this task.

BP continued to show considerable progress over the following two weeks. His ability to follow simple commands improved, such as putting a pen in a glass (a task he was observed doing on the 10th of January, 2000), but following complex commands remained difficult.

On the 12th of January, the Huskins Non Verbal Apraxia Screening Test was administered, showing significant decrement on the Oral Praxis subscale (9½/20), while his score for ideomotor praxis was 16/20. When testing for constructional praxis, it was shown that he was able to draw a circle, triangle and a house, and spontaneously write words. He was also able to complete a 'sentences and opposite task', with minimal prompting.

Spontaneous speech did not return until the 14th of January, when he made a request for food.

On the 15th of January, BP was tested for his Median Somatosensory Evoked Potentials. The cortical potentials were found to be absent on the left side, and preceding wave forms were of a lower amplitude than on the right. Right-hemisphere potentials were all found to be within normal range. Cortical potentials were retained, and of normal latency. Somatosensory evoked potentials involve applying a sensory stimulus (often a small electric shock to the median nerve of the wrist) and testing to see if there is the expected neural response. The

results here indicated that the somatosensory pathways (presumably in the primary somatosensory cortex given the nature of BP's injury) were significantly damaged on both sides, particularly on the left (where no response was measured). On the 17th of January, 2000, BP was transferred to a private rehabilitation centre.

An attempt to track BP's progress in a visual way throughout the time of his hospital stay has been made by graphing his GCS scores as recorded over this time. The result can be seen in Figure 2.

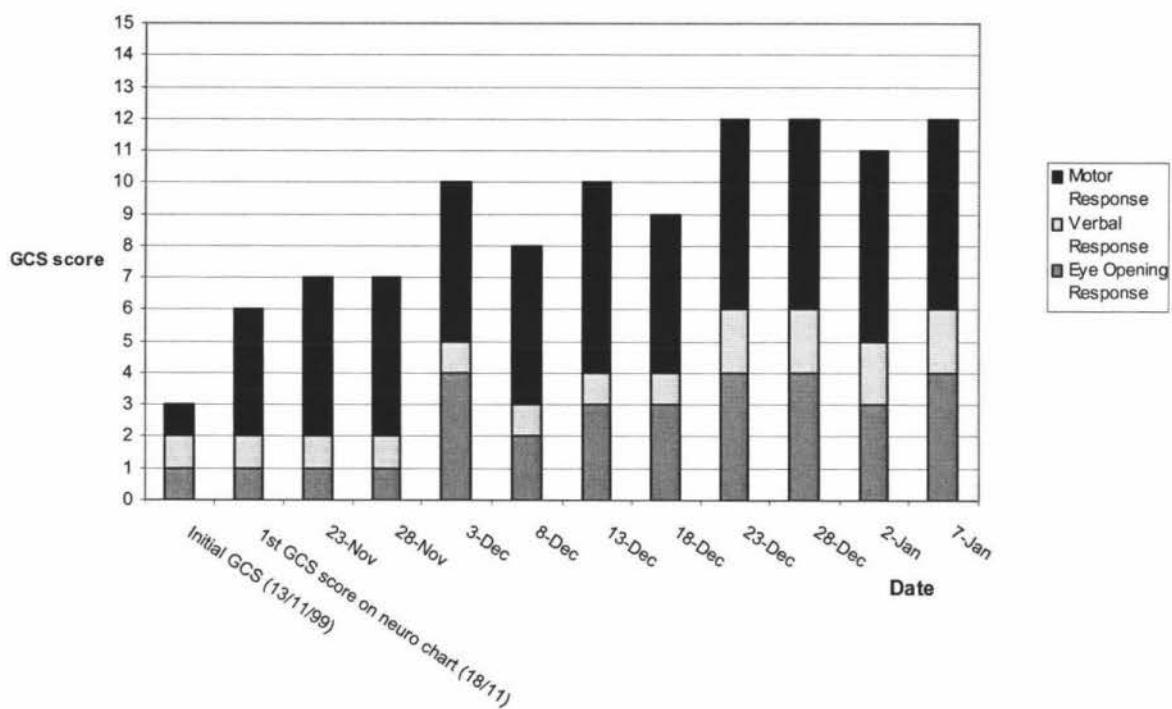


Figure 2. Global Coma Scale (GCS) scores (and subscale scores) for BP throughout the acute phase of recovery (13/11/99 to 7/1/00).

Figure 2. Global Coma Scale (GCS) scores for BP for first three weeks post-injury. GCS scores were plotted for every 5 days from the point of BP's injury to the last recorded GCS score on his neurological chart. The first recorded reading for the day was used. This graph shows in a visual way that BP showed relatively consistent and steady progress over the first three weeks of his recovery. Improvement in motor response was the most rapid, improving from a score of 1 (no response) at the scene of the injury, to a score of 4 (flexion withdrawal) within the first few days. It remained at 5 (localises pain) or 6 (obeys commands- the highest score) for the rest of this time. Verbal response was much slower however, with no response seen until over two months post-injury. In fact, at the time of discharge, minimal verbal response was possible. The major fluctuations in total GCS score were largely due to the more erratic scores of eye-opening response. On the 3rd of December, BP demonstrated spontaneous eye opening, the first time he had opened his eyes at all. This explains the jump in GCS score on the graph for this date. BP showed some variation in eye-opening response from this point on, but the points plotted do give good indication of BP's typical response at each particular point in his recovery.

The best indication of BP's current level of brain damage is an MRI scan performed on November 2nd 2000 which showed extensive bifrontal gliosis, more marked on the right, with volume loss, and minor gliosis present at the anterior right temporal lobe. Appearances were noted as being consistent with severe frontal trauma, with no other lesions present. These abnormalities are clear on the three MRIs shown in Figure 3.

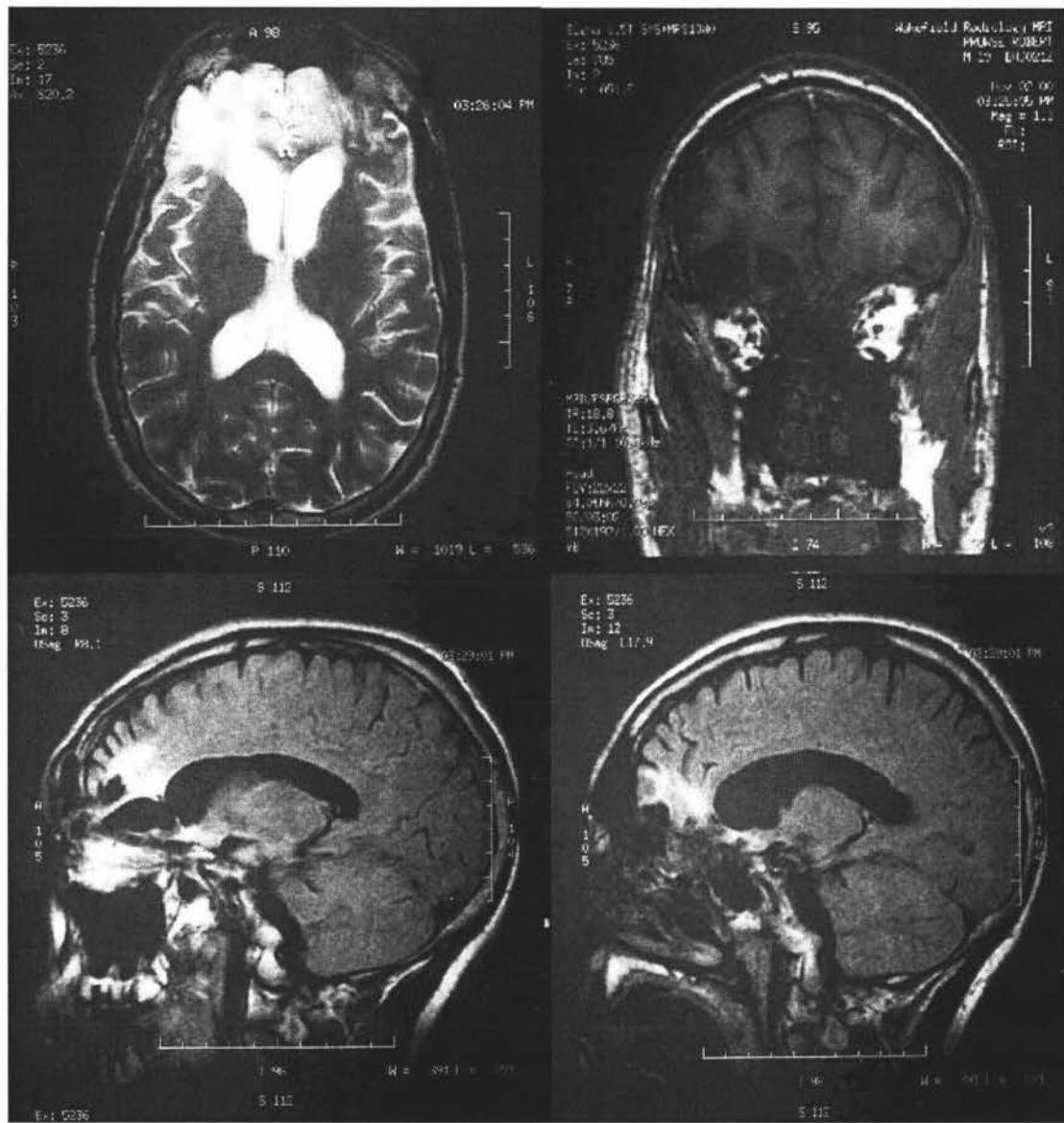


Figure 3. MRI scans taken of BP's brain in November 2000. The horizontal and coronal views (top left and top right respectively) show the large bifrontal gliosis, particularly prominent in the right hemisphere (the left side of each image). A sagittal view through both hemispheres (the bottom two images) shows that damage was extensive in the orbital frontal regions of both sides of BP's brain.

Long-term outcomes

Long-term outcomes of traumatic brain injury can only be discussed in a very general sense, as the nature of impairment is largely dependent on several factors. These include the location and type of the lesion, how diffuse the damage was, the state of the individual before the accident (with regard to age, reserve capacity, medical and psychiatric complications, psychosocial factors such as level of social and family support, level of education, and socio-economic status), the time since injury and the nature and extent of any post-injury rehabilitation. One broad way of assessing the outcome of TBI sufferers is to use the Glasgow Outcome Scale (Jennett & Bond, 1975). This scale categorised patients into one of five outcomes: death, persistent vegetative state, severe disability, moderate disability, or good recovery. This rating has been criticised for being crude and insensitive to subtle improvements to a patient's status, for its insensitivity to improvements or if overall change has not been great (Satz et al, 1998). Obviously such changes are crucial to rehabilitation, as these improving capacities can be used to compensate for other more impaired areas of functioning, as well as giving great hope to the patient and their loved ones.

Possible sequelae of TBI can be grouped into cognitive, behavioural/personality impairments, or physical outcomes of TBI. An attempt to do so follows. It must be kept in mind, however, that due to the extremely varied outcomes from traumatic brain injury due to the variables mentioned above, these disorders and impairments can only be discussed very briefly and broadly.

Cognitive outcomes

Memory dysfunction is very common following TBI, and memory complaints continue to be the most common reason for referral for neuropsychological assistance

to neuropsychologists (J. M. Leathem, personal communication, March, 2000). This is because memory dysfunction (of different types) has been linked to a multitude of neural structures throughout the whole brain (Tranel & Damasio, 1995), so that a lesion is likely to affect one or more of such structures. Impairments in other areas of cognitive function are also common, including difficulties with attention, perception, visuo-spatial abilities, executive function (organisation and planning) and language. It is common for those with TBI to suffer from a combination of these cognitive impairments rather than any in isolation. A list of possible cognitive sequelae of TBI (paying particular attention to severe TBI), the common neuropsychological measures used to assess each cognitive realm, and example studies of these impairments, can be found in Table 1.

Personality/emotional outcomes

Personality changes are particularly difficult to deal with following a traumatic brain injury, for the individual and their family and friends alike. These changes may include disinhibited and inappropriate behaviours and comments, childish behaviours, unprovoked aggression, all of which can be extremely distressing for loved ones. In fact, some families may take the position that the person who came home from the hospital is a different person altogether to the one they knew so well before the injury. As a result friends may stop coming to visit and a progression towards socially isolation often begins.

Emotional outcomes are also varied and have wide-ranging consequences for the brain-injured individual, family and friends. Mood disturbance, particularly major depression, is common following TBI (Newburn, 1998). Apathy and decreased motivation with or without associated depression, are also common (Kant, Duffy, &

Pivovarnik, 1998). The many types of personality and emotional changes of (particularly severe) TBI, and example studies of such changes can be seen in Table 2.

Physical outcomes

Physical impairments are quite likely to show rapid recovery post TBI. Lezak and O'Brien (1990) found that only 17% of their TBI sample had moderate or severe difficulty walking 5 years post-injury, much lower than was found soon after the injury. This may be so because physical injuries are more likely to be addressed (through physiotherapy) than say, social interaction difficulties. Physical impairments have also been shown to not have the same impact on carer burden as other TBI sequelae, such as social aggression or cognitive disability (Frank, 1994). Still, these physical outcomes can affect many areas of a person's daily life following a TBI. For instance, the inability to engage in some leisure activities as a result of the injury may contribute to the decreased social contacts and increased social isolation.

One common physical outcome following TBI is the development of post-traumatic epilepsy. These can be defined as 'early' (usually within the time when ICP is elevated) or 'late' (which has much greater implications with respect to the need for treatment of ongoing epilepsy; Long, 1989). Whilst not as high as that for missile injuries (30% or more; Annegers et al., 1980), the incidence of late seizures amongst those admitted to hospital following a closed head injury (CHI) is approximately 5% (Long, 1989), as opposed to the 2% or so epilepsy rates in the general population (Snyder, 1998). The incidence becomes much higher when looking at particular subgroups of CHI. Early posttraumatic seizures, depressed skull fractures and intracranial haematomas all dramatically increase the risk of late seizures amongst survivors of CHI (Long). One study showed the rate of late seizures after an

Table 1. Long-term cognitive outcomes observed following traumatic brain injury (TBI)

Faculty	Impairments observed as a result of TBI	Some common neuropsychological measures used	Example study	Outcomes observed in that study
Learning/ Memory	<p><u>Short-term memory:</u> Reduced capacity to hold information, whether visual or verbal. Difficulty encoding information, making it relevant and meaningful, and therefore not being able to form memories so easily.</p> <p><u>Long-term memory:</u> Reduced capacity to retrieve memories from long-term store when needed. Impaired ability to make memories contextually significant and meaningful, therefore making them difficult to retrieve (they are not stored in meaningful ways). Impairment in meta-memory, so that the need to rehearse and re-learn information may not be understood. Also, a decreased use of strategies (both external and internal) to aid learning and memory retrieval, either due to a lack of awareness of their usefulness or need, or because the concept of using strategies is forgotten at the time they are needed. Interference (of other information) causing quick degradation of earlier memories.</p> <p><u>Working memory:</u> Reduced ability to hold information in a temporary store, and mentally manipulate that information. The inability to rehearse units of information in order to cement it as a long-term memory.</p>	<p>-Rey-Osterrieth Complex Figure Test (visual memory, short-term and long-term, recognition and recall).</p> <p>-Rey Auditory Verbal Learning Test (verbal memory, short-term, long-term, learning curve, interference).</p> <p>-Digit Span (WAIS).</p> <p>-Logical Memory (WMS).</p> <p>-Rivermead Behavioural Memory Test (everyday memory)</p> <p>-Benton test of visual retention.</p>	<p>Tabaddor, Mattis, & Zazula (1984)</p>	<p>68 patients with moderate-to-severe brain injury were given neuropsychological assessments. Amongst other deficits, memory at baseline was found to be well below norms: 6 standard deviations below for verbal memory (as measured by the Mattis-Kovner test of verbal retention) and approximately 5 standard deviations below for non-verbal memory (as measured by the Benton test of visual retention). While non-verbal memory was shown to improve quite a lot from baseline to 6 months, there was no improvement from 6 months to 1 year, leaving scores still approximately 4 standard deviations below norms. Verbal memory continued to show improvement after the 6-month mark, but was still well below norms.</p> <p>25 severe TBI patients and 25 matched controls were given neuropsychological tests designed to measure performance of the slave systems of working memory (digit span and spatial delayed response task), as well as a dual-task paradigm to assess performance of the central executive system (CES) (in line with Baddeley's theories of working memory). Performance on these was also compared to scores on tests of executive function. The TBI group had slower reaction</p>

**Language/
Communication**

Anomia or dysnomia (i.e., an inability, or difficulty with, word-finding or naming). Aphasia (difficulty with the production of speech or written language, or with the comprehension thereof). Inappropriate use of language (e.g., using inappropriate words, excessive swearing, disinhibited use of language). Decreased speed in the use of language (either in comprehension or in output). Decreased ability to understand non-verbal language (i.e., body language), or to perceive and understand intonation and sarcasm.

**Attention/
Concentration**

Sustained Attention: Difficulty in concentrating for extended periods of time. Fatigue. Unable to continue on one task without taking breaks or performance decreasing.

Selective Attention: Decreased ability to shut out distracting stimuli. Increased difficulty in focusing on one task (e.g., carrying on a conversation while the television is on in the background).

-Boston Naming Test.
-Comprehension
(WAIS).
-Word Fluency Test.

Continuous
Performance Tests
(CPT)

Stroop Test.

Levin, Song,
Ewing-Cobbs,
Chapman, &
Mendelsohn
(2001)

Loken,
Thornton, Otto
& Long (1995)

Wallesch,
Curio, Kutz,
Jost, Bartels, &
Synowitz,
(2001)

times on the tasks performed alone, and greater decrements in performance for the dual-task conditions. Executive function was also shown to be impaired, while principal components analysis uncovered a common factor between executive function performance and the dual-task paradigm. These results support the notion that working memory is compromised by severe TBI, and that this impairment may be attributed to CES dysfunction.

122 children who had suffered a CHI (78 severe and 44 mild) and 104 uninjured controls were compared on a word fluency test. Results indicated that those children who had suffered a severe CHI performed at a lower level for word fluency than both the mild brain injury and control groups. The TBI group were also followed over a period of up to 5 years, and it was found that word fluency recovery was slower for those who had suffered a severe brain injury when compared to the mildly brain injured group. Age when injured also had an effect on word fluency (those that suffered the injury the earliest had slower recovery).

20 severe CHI patients were compared to 20 normal matched controls on a visual CPT, with three levels of complexity. The CHI patients showed a vigilance decrement across all levels of complexity compared to the control group.

In neuropsychological assessments of 60 TBI patients, a marked and stable difference was found in many areas for those with focal contusions (particularly behavioural), while those who suffer diffuse axonal injury (DAI) were much more likely to show marked changes in the

<p>Divided Attention: Difficulty switching attention between different stimuli. Impaired performance on complex tasks as a result, and making it difficult to perform more than one activity at the same time (e.g., driving, where several things must be attended to simultaneously).</p>	<p>Trail Making Test (B).</p>	<p>Park, Moscovitch & Robinson (1999)</p>	<p>first weeks post-injury, that mostly dissipated. The DAI group did however show continued difficulties with attention, with some interference as measured by lower scores on the Stroop test than normal controls.</p>
<p>Executive Function (e.g., Organisation and Planning)</p>	<p>Reduced ability to shift cognitive set. Rigidity of thought. Difficulty in planning sequenced behaviour. Reduced ability to generate goals, and to break them down into subgoals in order to reach the final goal. Reduced ability to organise ones' thoughts and actions, and to self-regulate (i.e., learn from one's mistakes)</p>	<p>Wisconsin Card Sorting Task. Tower of London/Hanoi Task. Object Alternation Task. Picture Arrangement (WAIS). 20 Questions.</p>	<p>Goel & Grafman (1995)</p> <p>6 severe TBI patients and 6 controls were given the PASAT and a lag recognition test separately and concurrently. The TBI group performed as well on each test when presented separately, but performed at a lower level than the control group when the tasks were presented simultaneously. Divided attention impairments were implicated, particularly when working memory is employed. A meta-analysis of studies investigating divided attention following TBI was also performed, and it was found that divided attention was impaired on tasks that involved controlled memory processes using either semantic or episodic memory. On the other hand, the meta-analysis uncovered that divided attention for tasks involving perceptual or motor memory, or that can be performed relatively automatically, is relatively intact following TBI..</p> <p>20 adult patients with lesions in the prefrontal cortex were compared to controls on the Tower of Hanoi task. The brain-injured group's scores were significantly lower. The researchers discovered that the difference was largely due to an inability to see or resolve a goal-subgoal conflict, and failure to inhibit an initial response.</p>

Judgement/ Perception	Difficulty in understanding others' actions, intentions, conversations, or facial expressions. Difficulty in choosing the best action to take in a given situation. Problems with generalising from one situation to another, or distinguishing different behaviours for different situations.	Comprehension (WAIS).	Schlund & Pace (2000)	Participants included 4 adult TBI patients and 4 normal controls. Both groups were made to press a response key, under concurrent response-reinforcement contingencies, in order to receive a reward. They were also asked to make judgements about the size of that reward based on the response contingencies. The TBI group performed significantly worse, suggesting that judgement of causality is impaired following TBI.
Visuo-spatial	Impairment in visual processing such that sight, perception of the world, and/or visual representation may be distorted. Difficulty visualising or making cognitive maps. Easily disorientated. Difficulty with visual scanning, and/or depth perception.	Rey-Osterrieth Complex Figure Test (copy), Picture Completion, (WAIS) Block Design (WAIS), Symbol Search (WAIS).	Schenk & Zhil (1997)	32 patients who had suffered a brain injury were tested for global visual motion perception. Three were found to have significantly impaired global visual motion perception. Given that one of these patients had a lesion in the left hemisphere, and two in the right, it was suggested that an area for the perception of visual motion can be found in both hemispheres (each assigned to the contralateral visual half-field), and that this area is situated in the posterior medial temporal gyrus.
Speed of Information Processing	Increased time taken to complete tasks, particularly as complexity increases. Decreased ability to process material quickly and efficiently.	Paced Auditory Serial Addition Test (PASAT). Completion times for WAIS tasks.	Godfrey, Knight, Marsh, Moroney & Bishara (1989)	In a study of 18 adults who had suffered a severe brain injury, information-processing speed was significantly impaired compared to a control group, measured by the speed of performance on both a word rotation task and a visual search task.

Table 2. Long-term personality changes and emotional outcomes observed following traumatic brain injury (TBI)

Faculty	Impairments observed as a result of TBI	Example study	Outcomes observed in that study
Agitation	General restlessness and irritability.	Masson et al. (1996)	231 people recovering from a TBI and 80 controls (who suffered a lower-limb injury) were assessed for cognitive, behavioural and physical impairments 5 years post-injury. Amongst the impairments, it was found that 46% of the TBI group (of those who answered the question) still complained of irritability 5 years on, compared to 14% of the lower-limb-injured group. Severity of injury was also a factor, as 63% of those who had suffered a severe TBI complained of irritability, compared to 60% of the moderate injury group, and 38% of mild brain injury sufferers.
Impulsiveness	Usually associated with the frontal lobes. Disinhibited and inappropriate behaviour. Difficulty controlling one's own behaviour and impulses. Excessive impatience.	Kersel, Marsh, Havill & Sleigh (2001)	65 adults who had suffered a severe TBI were assessed for levels of psychosocial functioning at 6-months and 1-year post-injury. At both assessments, the most common behavioural problem mentioned was impatience (51% at 6-months, compared to 49% suffering from irritability, 44% argumentative, 42% anger, 42% depression, and 41% disinterest, to name a few. The rates were similar at 1-year post-injury as at 6 months).
Motivation	Apathy and disinterest. Difficulties with initiation of behaviour, and motivating for change. Lack of direction. Decreased ability to set (and/or interest in setting) long-term goals.	Kant, Duffy & Pivovarnik (1998)	83 patients who had suffered a CHI (62 mild, 8 moderate, 9 severe, 4 unknown) were assessed for apathy using the Apathy Evaluation Scale (AES), and for depression using the Beck Depression Inventory (BDI). 11% were discovered to have apathy without depression, 11% had depression without apathy, and 60% exhibited both apathy and depression. Patients with severe injury were more likely to exhibit apathy alone, rather than both apathy and depression combined.
Socialisation	Social withdrawal. Inappropriateness in social behaviour (e.g., inappropriate joking, interrupting) and/or impaired display of emotions (e.g., flat affect). Disinhibition in social situations.	Galski, Tompkins, & Johnston (1998)	30 TBI patients and 10 matched controls were assessed for their competence in conversational discourse, and their levels of psychosocial adjustment. The TBI group was found to be impaired on four different areas of conversational discourse, and was also found to have lower levels on six measures of psychosocial adjustment. These included a lower level of community integration, less participation in social activities, poorer general quality of life, greater depression, and endorsed more symptoms of psychosocial maladjustment. The results indicate that many TBI sufferers, due in some part to impairments in social interaction, have difficulty reintegrating back into society fully.

Aggression	Increased aggressiveness/vulnerability to aggression. Anger. Possible violent outbursts. Self-injury (e.g., cutting one's arm, banging head against wall)	Andrews, Rose & Johnson (1998)	27 children who had suffered a TBI (8 mild, 9 moderate, 10 severe) and 27 controls were assessed with regard to various social and behavioural indicators. Those children that had suffered a TBI were more aggressive and displayed more antisocial behaviour than the control group (as scored by the DeBlois Aggressive and Antisocial Behaviour Scales [DAABS]). The TBI group was also found to have higher levels of loneliness and maladaptive behaviour, and lower levels of self-esteem and adaptive behaviour. Severity of brain injury was not found to influence the social and behavioural constructs being measured (although this could be due to the small sample size).
Self-awareness	Impaired insight. Lack of understanding of own losses/deficits/inappropriateness. Often associated with damage to frontal lobes.	Port, Willmott & Charlton (2002)	The levels of insight of 30 mild-moderate TBI patients and their significant others were measured using the Awareness of Deficit questionnaire (ADQ), low-moderate levels of difficulty into self-awareness were reported, although their data suggested that the awareness of significant others may also be limited during the early recovery phase.
Mood disturbances	Mood swings, depression, excessive crying/laughing, or mania.	Glen, O'Neil-Pirozzi, Goldstein, Burke, & Jacob (2001)	41 TBI outpatients were assessed for depression using the Beck Depression Inventory-II (BDI-II). 24 (59%) were found to have clinically significant depression ($BDI-II < 13$). 14 of these depressed outpatients, or 34%, scored in the moderate or severe depression categories.
Anxiety	Generalised anxiety, panic attacks, phobias, obsessive-compulsive disorder, post-traumatic stress disorder (PTSD).	Williams, Evans, Wilson, & Needham (2002)	66 survivors of severe TBI were assessed for prevalence of PTSD, using the Impact of Event Scale (IES). It was found that of the sample, 18.2% had clinically significant PTSD. 6.1% of those diagnosed with PTSD were rated as having severe symptoms. These figures indicate that the anxiety disorder PTSD is more prevalent following TBI than in the general population.
Sexuality	Sexual dysfunction, sexual identity confusion, inappropriate sexual behaviour.	Sandel, Williams, Dellapietra, & Derogatis (1996)	52 outpatients who had suffered a TBI completed the Derogatis Interview for Sexual Function (DISF) to determine the presence and/or extent of sexual dysfunction following brain injury. The majority of the sample (63.5%) had suffered a very severe TBI ($PTA > 4$ wks). Results showed that sexual functioning was lower than that of non-injured populations, but only reached statistical significance on 2 of the 5 subscales: Orgasm and Drive/Desire. Ratings of sexual functioning were higher for those with frontal lobe lesions, right hemisphere lesions, and for those who had had their injury more recently. Severity of TBI did not have an effect on sexual functioning.

Table 3. Long-term physical/somatic outcomes observed following TBI

Faculty	Some possible consequences of TBI	Usually associated with early/late stage of recovery?	Example study	Reported outcome
Headaches	Persistent headaches or migraines. Headaches triggered by certain things (e.g., smells, bright lights, fatigue).	More often associated with early stages, especially following mild TBI (i.e., post-concussion syndrome)	Couch & Bearss (2001)	Participants consisted of 92 TBI patients who had developed headaches post-injury. The effect of TBI severity on presence of chronic daily headache (CDH) was investigated. For those with moderate or severe brain injuries, 27% had CDH compared to 69% with no headache, while for the mild brain injury group, 80% suffered from CDH compared to 11% with no headache. The reasons for this inverse relationship between injury severity and presence of CDH were unclear.
Dizziness/Balance	Dizziness/vertigo. Loss of balance.	More often associated with early stages, especially following mild TBI (i.e., post-concussion syndrome)	Masson et al. (1996)	231 people recovering from a TBI and 80 controls (who suffered a lower-limb injury) were assessed for cognitive, behavioural and physical impairments 5 years post-injury. 32.4% of TBI patients reported experiencing dizziness, compared to 12.5% of the controls. Severity of injury was not found to have an effect on the occurrence of dizziness 5 years on.
Motor Disturbance	Paralysis of particular body parts. Hemiparesis/hemiplegia. Uncontrolled movements and/or shaking. Impairment in fine motor movements (e.g., finger movements). Impairments in muscle movement and/or co-ordination.	Motor dysfunction may recover over time, but may also remain indefinitely.	Haaland, Temkin, Randahl, & Dikmen (1994)	40 TBI patients (of differing severity), who had not suffered peripheral upper body injuries, were examined for fine motor skills (grip strength and finger tapping ability) at 1-month and 1-year post injury. Results were compared against a control group of 88 people who had not suffered a head injury. The TBI patients were shown to be impaired on both finger tapping and grip strength one month after their injuries, when compared to the control group. At 1-year post-injury, the TBI group continued to demonstrate deficits in finger tapping, but there was no significant difference between the two groups on grip strength. Results suggest that fine motor control may continue to be impaired years after a TBI, and that grip strength appears to recover at a faster rate than finger tapping,

Fatigue	Excessive fatigue (possibly after minimal exertion of energy), resulting in tiredness, impaired attention, and impaired cognitive functioning. General weakness.	Often associated with diffuse injury. Can remain in the very long-term.	Leathem & Babbage (2000)	probably due to the speed required. 88 TBI patients (predominantly severe) from an outpatient neuropsychology service in New Zealand were given the Symptom Checklist 90-Revised (SCL-90-R), to investigate the extent to which the symptoms of brain injury artificially inflate its scores. 89.8% of the respondents reported "feeling low in energy/slowed" in the week leading up to filling in the checklist, with more than 10% citing extreme difficulty. 75% said they had experienced the feeling that "everything is an effort", while 66% also said they had felt weak in parts of their body. It was concluded that caution must be taken when interpreting TBI patients' scores on the SCL-90-R.
Sleep disturbance	Insomnia. Disturbance waking cycles. Excessive tiredness during waking hours.	Found in both early and late stages of recovery (although not regarded a common outcome).	Leathem & Babbage (2000)	88 TBI patients (predominantly severe) from an outpatient neuropsychology service in New Zealand were given the Symptom Checklist 90-Revised (SCL-90-R), to investigate the extent to which the symptoms of brain injury artificially inflate its scores. 70.6% of the sample said they had "sleep that is restless/disturbed" in the week prior, with greater than 10% citing extreme difficulty. 64.8% of respondents said they had had "trouble falling asleep". Once again, greater than 10% of the sample cited extreme difficulty with this area. It was concluded that caution must be taken when interpreting TBI patients' scores on the SCL-90-R.
Vision	Loss of vision. Blurred vision. Visual neglect. Nystagmus. Impaired visual scanning. Colour disturbance. Altered depth of field. Prosopagnosia.	Vision loss at early stage of recovery may return but often remains a long-term outcome.	Schlageter, Gray, Hall, Shaw & Gammatt (1993)	51 inpatients in a TBI rehabilitation programme were tested by an optometrist for visual impairments and compared to a control group of 23 hospital staff. The assessment was done within days of the patients' admission to the rehabilitation facility, and included many different areas of visual functioning (pursuits, saccades, ocular posturing, stereopsis, extra-ocular movements, and near/far eso-exotropia). 59% of the TBI group were found to have significant impairment in one or more of these areas. Those with impairments in pursuits or saccades were also treated with a regime of vision exercises, however, the results did not support the introduction of an inpatient vision programme.

Hearing	Partial or full loss of hearing. Tinnitus. Perception of sounds that aren't present in the environment.	Hearing loss at early stage of recovery may return but often remains a long-term outcome.	Lubinsky, Steger Moscato, & Willer (1997)	A nation-wide Canadian household survey was conducted in order to determine the prevalence of self-reported disabilities. From this sample, the responses of 454 people who said they had suffered a TBI were investigated in order to estimate the prevalence of speaking and hearing disabilities following TBI. To determine prevalence of hearing impairments, the survey had used the everyday example of hearing conversations. 74.3% of the TBI group reported having trouble hearing conversations with one person, while 96.2% reported having trouble hearing a group conversation amongst 3 people, whether or not a hearing aid is being used. 78.6% of TBI patients also reported having trouble hearing a conversation over the telephone. Results from a previous study using these survey results to determine disability in the whole population were also reported, with 19.6% reporting trouble with their hearing, much lower than the TBI sample.
Taste	Loss of taste. Disturbed taste (e.g., perception of unpleasant flavours). Acquired dislike for foods enjoyed post-injury (or acquired liking for previously disliked foods). Disrupted saliva production.	Found in both early and late stages of recovery.	Fujii, Fujita, Hiramatsu & Miyamoto (1998)	The disappearance of two peoples' pre-morbid food aversions following TBI were discussed. One case, a 19-year-old woman who had suffered a severe brain injury as the result of a motorcycle crash, disliked Spanish paprika and meat very much, but 1 month post injury had found herself to like the taste of both. The second case, a 27-year-old man who had suffered a severe TBI in a traffic accident, found he liked the taste of Spanish paprika, onions, raw fish, horseradish, and two types of vegetables, all foods he intensely disliked before his accident. While his dislike for horseradish returned 6-months post-injury, his preference for the other foods did not change over this time. CT scans showed the right dorsolateral cortex to be extremely injured in Case 1 (1 month post-injury), while Case 2's CT scan showed damage to the right posterior temporal lobe, the entire left temporal lobe, and the posterior part of the left frontal orbital surface.
Smell	Decreased sensitivity. Total loss of smell. Perception of persisting smells that aren't present in the environment.	Found in both early and late stages of recovery.	Ogawa & Rutka (1999)	Olfactory dysfunction was assessed in 365 workers who had suffered an occupation-related 'head injury', most of whom had been suffering from some dizziness post-injury. 13.7% of

				participants were found to have olfactory dysfunction. 68% of these were categorised as anosmic (complete loss of smell), while the other 32% had either hyposmia (partial loss) or dysosmia (distorted sense of smell). These figures were slightly higher than previous literature had suggested, probably due only work-related TBI being assessed (falls accounted for most of the accidents). Olfactory dysfunction was found to occur more frequently for those with moderate to severe injury (33% of those with LOC>1 hour, compared to 10.6% for those with LOC<1 hour). Olfactory dysfunction was also found to more common amongst those who had suffered a skull fracture (50%), and was twice as likely to occur following frontal, occipital, skull base and midface fractures compared to temporal and parietal fractures.
Touch	Decreased sensitivity. Total loss of touch.	Found in both early and late stages of recovery (although not regarded a common outcome).	Pandey, Mohanty & Mandal (2000)	4 groups of focal brain-damaged (1-left hemisphere, involvement of parietal lobe; 2-left, no parietal involvement; 3-right hemisphere, parietal; 4-right, not parietal), and a control group of non-brain injured medical patients, were tested for their tactual recognition. Participants were asked to match up letter or numbers by picking out the appropriate shape with their hands. Results showed that those with parietal lobe lesions performed at a lower level than those with focal lesions situated in other lobes, indicating that the parietal lobe is important for tactual recognition. The left hemisphere injured group had more difficulty with tactual recognition of letters, while those with lesions situated in the right hemisphere had more difficulty with recognition of figures.
Sexual dysfunction	Erectile dysfunction or other physical conditions impacting on sexual performance.	Found in both early and late stages of recovery.	Leathem & Babbage (2000)	88 TBI patients (predominantly severe) from an outpatient neuropsychology service in New Zealand were given the Symptom Checklist 90-Revised (SCL-90-R), to investigate the extent to which the symptoms of brain injury artificially inflate its scores. 48.3% of the sample reported to having felt a "loss of sexual interest/pleasure" in the week prior to the assessment. Greater than 10% of respondents reported that they had had extreme difficulty with this. It was concluded that

Epilepsy	Development of seizures post-injury, that may or may not disappear over time.	Found in both early and late stages of recovery, although early seizures often occur during increased ICP and do not have the same implications for ongoing epilepsy as do late seizures.	Annegers & Coan (2000)	caution must be taken when interpreting TBI patients' scores on the SCL-90-R. 4541 TBI patients were followed up in order to examine how characteristics of brain injury are associated with the development of post-traumatic seizures. Seizures were found to be far more likely following a severe brain injury (17.2 of this group suffered from post-traumatic seizures compared to 2.9% of the moderate and 1.5% of the mild injury groups). The risk of post-traumatic epilepsy increased for those who suffered subdural haematomas, skull fractures, longer periods of loss of consciousness (< 1 day), or were over the age of 65.
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intracranial haematoma to be 26%, which increased to 44% when just looking at moderate and severe TBI (PTA > 24 hours; Jennett and Bond, 1975). Epilepsy is important to monitor and treat, even if seizures are infrequent. For instance, those who use dangerous equipment or drive motor vehicles could be putting themselves at great risk of further injury, while those who have cognitive or behavioural impairments may have these symptoms exacerbated during the confusion of the ictal and post-ictal periods.

A list of common physical outcomes of traumatic brain injury can be found in Table 3, along with which particular stage of recovery and severity of injury is usually associated with each somatic complaint.

Psychosocial outcomes

Lezak and O'Brien (1990) showed that, of 39 patients who had suffered a TBI, only 18 were working or at school three years post-injury, while only 6 of these were considered to be functioning at premorbid work or education levels. Furthermore, at 5 years post-injury, 5 of the 39 patients had been hospitalised for psychiatric problems, 6 were in care facilities such as a nursing home, 11 had suffered subsequent accidents (mostly motor vehicle-associated), and one had served a jail sentence. These findings are grave indeed, and help to elucidate the true extent of the problem that is the psychosocial outcomes of traumatic brain injury.

Psychosocial adjustment has been identified as the most impaired of all areas of functioning, following a severe TBI (Oddy, Coughlan, Tyerman, & Jenkins, 1985, cited in Godfrey, Marsh & Partidge, 1987). Often those who suffer a head injury find they have fewer interests and hobbies, and make and receive fewer visits, and

generally spend less time on leisure pursuits after their injury. This increasing social isolation can lead to increased dependence on family and caregivers (increasing their burden), and may make the person more inclined to withdrawal socially, and become depressed. Guilt for the increasing dependence and burden on carers may also increase stress and depression. Friends, now finding their depressed friend is even more difficult to spend time with, may reduce contact further, thus social isolation continues to get worse. This is exacerbated by the lack of opportunity to establish new contacts and friends, partially due to the majority of severe TBI sufferers living at home with their families (Morton & Wehman, 1995). Meanwhile, difficulties regarding returning to work or academic study may make the TBI patient to feel worthless, adding to the depression he or she is already experiencing.

A number of studies have demonstrated the effects that a TBI can have on a person's psychosocial functioning. When compared to an outpatient and non-clinical group, Newton and Johnson (1985) found that a group of severely head-injured patients had poor social adjustment, demonstrated social interaction difficulties, and (perhaps as a result) had higher social anxiety and lower self-esteem. Marsh and Knight (1991) demonstrated that the social interactions of 18 sufferers of very severe closed-head injury were impaired in many ways 6 years on from their injury. Their conversations were characterised by lack of fluency and clarity (due to word-finding deficiencies and inability to express themselves clearly), and they appeared disinterested when compared to a control group. These deficits would not be very reinforcing for other people to want to engage in a conversation, providing another means by which social isolation can occur. Another study of 100 survivors of blunt head injury found that level of overall psychosocial reintegration was rated as 'good' for only 24.1% of the

sample, while 42.5% and 33.3% were rated as 'substantially limited' and 'poor' respectively (Tate, Lulham, Broe, Strettles, & Pfaff, 1989).

It is easy to see how the psychosocial consequences of TBI act upon the sufferer to only worsen their situation, and how important it is to acknowledge the downward spiral of psychosocial outcomes and try to intervene early. It is probably fair to say that the impact of a traumatic brain injury on a person's relationships within their home, workplace and with their friends is the most troubling and difficult to remedy of all sequelae of TBI. Caregiver burden, a particular concern within the psychosocial outcomes of TBI, will be discussed at greater length in Chapter 5.

Severe brain injury: Long term outcomes

Most of those that survive the severest forms of head injury are healthy adolescents or young adults who have a further life expectancy of half a century or more. Due largely to the improvements in resuscitation and treatment of complications following a traumatic brain injury, more and more survivors from severe TBI are able to live for a long time post-injury (Lewin, Marshall & Roberts, 1979). However, advances in rehabilitation and in treatment for those surviving a severe TBI have not kept up with the medical advances in keeping patients alive. Of those who survive the early post-injury period of a severe TBI (which may be as low as 50%; Lewin, Marshall & Roberts, 1979), most require long-term caregiver assistance after a long hospital stay (Gray, 2000). Also, since personality, cognitive and behavioural changes are more likely to be greater after a severe TBI, the psychosocial problems of social isolation and decreased social networks and leisure activities will be accentuated. One of the greater problems in mental health today is trying to tackle the decreased quality of life in such people that results from the psychosocial changes to the lives.

Severe traumatic brain injury usually results in a combination of diffuse axonal injury (DAI) and more focal injuries pertaining to the nature of the injury. The individual is also affected by other injuries to other parts of the body. These three combined result in a great variety of possible outcomes (Morse & Montgomery, 1992). An overview of the possible outcomes of TBI can be found in Tables 1, 2 and 3, and, in general, the outcomes for *severe* long-term injury are the same, only to a greater extent. In fact, it is fair to say that most of these outcomes mentioned only occur following moderate to severe TBI, apart from a few known sequelae of post-concussion syndrome (the cluster of symptoms that commonly follow a mild TBI) that are mentioned in Table 3. As a basic rule, the more severe the TBI, the greater the level of impairment.

Several studies have investigated this relationship between brain injury severity and long-term outcome in survivors. Even amongst a severely brain injured population the effect of severity can be seen. Bishara, Partridge, Godfrey and Knight (1992) considered long-term outcomes of 93 severe closed head-injured patients (i.e., their Global Outcome Scale levels at 6 months and 12 months post-injury), and discovered that the only factor that contributed significantly to outcome was length of post-traumatic amnesia (PTA), a common indicator for severity of injury.

The outcome of a traumatic brain injury is also affected by several other factors pertaining to the injury. For example, the extent to which the lesions are diffuse or focal can have a great impact on predicted outcome. Prigatano (1999) reported that diffuse brain injuries, particular when severe, are often associated with a poorer psychosocial outcome. Maxwell, Povlishock and Graham (1997, cited in Wallesch et al, 2001) argue that severe diffuse axonal injury (DAI) is the primary cause of

neurological and neuropsychological impairments post-injury. Severe DAI has also been reported to be the most common cause of the vegetative state and severe disability until death, and the cause of 35% of all deaths after suffering a head injury (Graham, 1995). The quantity of social behaviours emitted is negatively correlated with brain injury severity, suggesting that social withdrawal is more pronounced for those with severe brain injury (Godfrey, Marsh & Partridge, 1987).

Age is another factor that can be taken into account when assessing the likely outcome of a severe TBI patient. The research has usually demonstrated that children (those aged 19 and under) generally have faster recovery and overall better outcome than adults, while the elderly have particularly poor outcomes (Marion, 1996). Some findings have not found such a result however. For instance, Thomsen (1989) investigated the effect of age on very late outcome in 40 people, aged 15-44 when they suffered a severe blunt TBI ($PTA \geq 1$ month). The frequency of 13 late cognitive, behavioural, social and subjective problems were compared at 2 1/2 and 10-15 years, and were actually found to be *positively* correlated to age at injury. It appears there is still some debate as to whether age at injury is a reliable factor in the prediction of long-term outcome following TBI.

It has been suggested that alcohol consumption at the time of injury can not only be looked upon as a pre-disposing factor for the likelihood of TBI, but can also affect outcome. Patients who have a positive Blood Alcohol Content (BAC) have impaired their processes of initial recovery in several ways, and enter hospital with a lower level of consciousness (Barnfield, 1995). Pre-injury alcohol abuse has also been shown to affect TBI outcome. For instance, Dikmen, Donovan, Løberg, Machamer and Temkin (1993) examined the relationship between pre-injury alcohol abuse and

outcome, and found that both pre-injury alcohol consumption and severity of injury were good predictors of neuropsychological impairment at 1-month and 1-year post-injury. While it is clear that alcohol and TBI are related in many ways, the effects of the two on each other are so complex as to make it very difficult to determine the exact effects of alcohol consumption on outcome (e.g., head injuries are more likely amongst substance abusers; the effects of TBI can lead many to substance abuse). Nevertheless, the relationship is an important one to consider when predicting outcome.

Another possible influencing factor on long-term outcome is the presence or absence of a subarachnoid haemorrhage (SAH). While cognitive and psychosocial impairments vary greatly amongst those who suffer a SAH, generally the outcome is quite poor. Bonita, Beaglehole and North (1983) reported that more than half of the victims of a SAH either die or become demented. Other common outcomes following a SAH include memory deficits, impairments in perceptual speed and accuracy, problems with cognitive flexibility and concept formation and abstraction, and sometimes difficulties in visuospatial constructive abilities (Ogden, 1996). The location of a SAH has not been found to correlate very well with either the severity of impairment or the type of cognitive difficulties a patient may suffer (Ogden, Mee & Henning, 1993). Other psychosocial outcomes from SAH include sleepiness, a lack of motivation and initiative, anxiety, depression, irritability, attention and concentration difficulties, and very often fatigue. One study found that 12 months after an SAH, 86% of those described as having had a “good recovery” still suffered from excessive fatigue (Ogden, Mee & Henning, 1994).

Initial outcome can also be used as quite a good predictor of long-term outcome. Najenson, Grosswasser, Mendelsohn and Hackett (1980) followed 147 severely head-injured patients through their in-patient rehabilitation, with their ability to work (at 6-month or more follow-up) used as the measure of recovery outcome. They found that of all initial predictors, cognitive status (as judged by neuropsychological assessment) and the presence of behavioural disturbances (including 'frontal syndrome' and unawareness of their disability) were the best predictors of unemployment at follow-up. Communication disorders (26.5% were dysphasic at discharge) and locomotor disability also influenced vocational outcome, but to a lesser extent. Accurate prediction of late outcome based on cognitive, behavioural and physical symptoms soon after injury is an important goal for neuropsychologists and medical specialists, not least because it means the individual, family members and loved ones can adjust to the long-term level of disability that can be expected. Unfortunately while these predictors are all helpful to gauge the likely long-term outcome, such prediction of outcome is a very inexact science indeed. The recovery process varies greatly from person to person, and while it is vital to provide loved ones with some idea of what to expect in the future, it must always be acknowledged that the long-term outcome may be much better or worse than these early predictions.

One of the best predictors of long-term outcome is the area of lesion. This is particularly true if the damage is focal. In general, the more focal a lesion, the more selective impairments will be (and the more preserved capacities are left to compensate for any losses). While more and more is being discovered with regard to the exact nature of behaviours and their particular neural substrates (e.g., the hippocampal complex with short-term memory and learning; the occipital lobe and visual abilities; etc.), there is still a lot that neuropsychologists have yet to uncover.

Perhaps the most complex and least understood areas of the brain are the frontal lobes. Damage to this area can result in a whole manner of cognitive and behavioural outcomes, making care and rehabilitation particularly difficult. While a discussion of the known relationships between brain structures and behaviours would be very lengthy, a discussion of the frontal lobes and their place in traumatic brain injury research follows.

Frontal lobe injury

The frontal lobes are particularly susceptible to major damage from trauma (Stuss & Benson, 1986). The frontal lobes make up over a third of the human cerebral cortex and are made up of diverse anatomical units, each with connections to cortical and subcortical regions, as well as to each other (Damasio & Anderson, 1993). For this reason, lesions situated in the frontal lobe have diverse and wide-ranging effects.

The expression ‘frontal lobe syndrome’ is one that is often used to describe a cluster of symptoms that is characteristic of many people suffering from a frontal lobe lesion. This combination of behaviours include social disinhibition (as in tactless, and showing a tendency to do things in public that are usually restricted to private situations and intimate relationships), childishness, self-centredness, unrealistic attitudes and expectations, and an impaired self-awareness and insight. (Wood, 1990).

These features are in fact produced by damage to the orbito-frontal cortex. It has also been suggested that it is this area that is also responsible for learning the associations between response and reward (Passingham, 1995). Impairment in being able to form such associations freely may make learning of the adverse consequences of problem behaviours difficult- a fact that has important implications for both the everyday functioning and the rehabilitation of individuals with orbito-frontal dysfunction.

Frontal lobe dysfunction has also been shown to impair attention, memory (particularly what is referred to as ‘working memory’, or a three-part system that “temporarily holds and manipulates information as we perform cognitive tasks” (Matlin, 1994, p. 126), verbal fluency, language formation, reading comprehension, writing ability, motor function, visual perception, response to changing consistencies, planning, awareness and insight, appropriateness of affect, inhibition, and to alter a person’s personality substantially. These changes in the personality and behaviour (e.g., inappropriateness, aggression, disinhibition) make reintegration back into society and work difficult, and place particular stress on family, friends and carers.

While the exact functioning of the frontal lobes remains a mystery due to its sheer complexity, some particular functions of the frontal lobes have been localised to a limited extent. Damage to the lateral convexity of the frontal lobe is often associated with problems of executive function (Wood, 1990). For people who have suffered a TBI affecting this area, the ability to plan future behaviours may become so impaired as to make even the simplest of activities overwhelmingly difficult. Impairment is also seen on tasks that involve planning and sequencing of responses. If left to their own devices, daily behaviour for such patients often becomes a highly repetitious routine, while long-term goals are typically unrealistic or illogical (Damasio & Anderson, 1993). Another common impairment for such patients is the inability to shift sets easily, i.e., they display a cognitive inflexibility that makes problem solving particularly difficult.

Behavioural deficits related to these localised parts of the frontal lobes have been investigated most clearly when looking at conditions other than TBI, where more

exact lesions are likely to occur (Stuss & Benson, 1986). Separate syndromes are rarely found in isolation amongst TBI patients, especially when examining the effects of severe TBI (Wood, 1990). Most will tend to show several characteristics of the different syndromes associated with frontal lobe dysfunction. Probably the single-most common problem though, and the most harmful to social relationships, according to Wood, is that of social disinhibition. It is this facet of frontal lobe dysfunction that may make a person seem so altered from 'their previous selves' causing peers and friends to gradually distance themselves. It is a challenge to the sufferer, to families, to friends and to those involved in rehabilitation, to overcome this apparently wide gap between what was expected of the person pre-injury, and what can be expected now and in the future.

BP: Outcomes

In this thesis the case is presented of BP, a young man who sustained a severe frontal lobe injury in a fall from a roof. Now, nearly three years on, despite extensive rehabilitation, he continues to demonstrate cognitive and behavioural impairments in line with frontal lobe damage.

At the time of his accident, BP was an 18-year-old final year high school student (Bursary), intending to study design at tertiary level the following year. He was a capable student, especially skilled in design and drawing. He lived in the Wellington area, with his mother, father and younger sister. A sister a year older was attending design school and lived away from home. His mother worked in the education sector, while his father worked from home as a chartered accountant. It would be fair to say then that BP comes from a high socio-economic status household. The family shared a keen interest in music, with BP playing guitar, piano and harmonica. He was also a keen sportsman, being particularly proficient in soccer, which he played at representative level for many years. He also was a keen skateboarder. He had many friends, mostly from his schooling, or from skateboarding circles.

Inpatient rehabilitation

On admission to the brain injury rehabilitation centre approximately 2 months post-injury, BP was still not communicating verbally. Post-traumatic amnesia lasted approximately 1 month into his stay here, resulting in a total PTA length of about 3 months. While in the rehabilitation centre, BP made rapid recovery in many areas, particularly in his physical and verbal capabilities. However, progress with his attentional difficulties was slow. He was unable to stick to tasks and needed frequent prompting with regard to grooming and personal care. He became fatigued easily and his motivation was minimal. Most marked were behavioural problems. BP would wander without regard for personal safety. He continued to display inappropriate social behaviour, such as talking to complete strangers, asking them personal questions and hugging them. He was unable to pick up on cues, being completely unaware of the inappropriateness of his actions. At the same time he would often display aggressive behaviour (albeit in a joking manner) to family members, friends and staff.

2 ½ months later, and after several home trials, BP moved back into his parents' home, where he continues to reside. Although the recommendations of the private rehabilitation centre at this time were that BP was not ready to move back into the community, the wishes of both BP and his parents were that he should move back home.

BP in home situation

Whilst his ability to apply himself to some tasks, his sustained attention and, to some extent, his awareness into his own limitations has improved a little, his inappropriate behaviour continues to be a problem. He has a tendency to wander and feels compelled to talk to everyone he sees (often making out he knows the person when the person is in fact a complete stranger), invading their privacy (e.g., walking into an unknown house or asking personal questions), and acting in inappropriate ways (e.g., threatening to be violent, making lewd sexual remarks and actions (again, usually in a joking manner), excessive swearing, lying, and shouting and singing loudly in places where he shouldn't (e.g., the cinema)). His impulsivity makes it extremely difficult to control himself from engaging in these behaviours, even when he is remorseful after being removed from the situation.

His mood also fluctuates, with significant depressive episodes, for which a course of citalopram (Cipramil) was trialled (starting early 2001 and continuing to this time). The depression can largely be attributed to an increased awareness of the extent of his loss, and the realisation that his brain injury will affect him for the rest of his life. This has arisen particularly at this time, as he sees his friends going flatting, going to university, and having the freedom to go out and drink in the weekends. Carbamazepine (Tegretol) is also being taken for control of seizures, although there have been few, and none have been observed for a long time.

This collection of behavioural difficulties mean that 24 hours a day oversight is necessary to prevent/offset harm to both BP and those around him.

A neuropsychological assessment for BP approximately 4 months post-injury, was conducted to determine post-injury strengths and weaknesses (Cavit ABI, 2000). It would also provide a baseline against which to measure improvements in cognitive functioning as a result of BP's rehabilitation. The results of that neuropsychological assessment are shown in Table 4.

To compare this neuropsychological assessment as at March 2000 with his current level of functioning, the findings from a recent neuropsychological assessment taken in November 25th 2002 is provided in Table 5 (Cavit ABI, 2002). It is clear from this assessment that while BP has shown a general improvement across the board, he continues to have substantial difficulties with executive function and reasoning skills, indicative of his functional behavioural impairments.

Table 4. Results for BP's neuropsychological assessment taken March 2000 (Cavit, 2000)

-Attention and Concentration: BP's sustained attention fluctuated between 40 minutes and less than 5 minutes, and he became fatigued easily. He scored within the average range for Digits Forwards (scaled score of 8) and Digits Backwards (6). A demonstrated inability to order verbal information and inhibit over-learned sequences was considered indicative of frontal dysfunction.

-Conceptual and Verbal Abilities: This was informally assessed due to time constraints and speech production difficulties. BP demonstrated a large vocabulary consistent with his education (7th form), but had frequent word finding difficulty. Fairly concrete conceptual abilities were expressed, although he was able to name abstract similarities between concepts and comprehend everyday situations. BP obtained a scaled score of 6 on picture arrangement.

-Performance and planning: BP demonstrated above average mathematics ability (scaled score of 11), although had some difficulty when there were a large number of calculations (probably due to impaired working memory rather than math skills). He scored at the low-average level on block design (scaled score of 8), largely due to slow manual manipulation of the blocks and slowed information processing. He obtained a scaled score of 10 on four nonverbal reasoning tasks: pattern completion, classification, analogy and serial reasoning (site tests), though he had difficulty with multiple reasoning steps. He performed poorly at picture completion (scaled score of 4), although his visual and attentional difficulties were thought to have contributed to this.

-Memory: BP performed in the low range for immediate auditory recall, having difficulty remembering details of a story (logical memory delayed recall scaled score of 4) and recalling themes. He demonstrated recall of 4/8 discrete pairs of words on first presentation (paired associates). Over 4 trials BP increased his recall from 5/12 to 7/12 of a word list (AVLT). There were a number of intrusions from previously learned information. BP's recognition was much higher than his recall, suggestive of a retrieval deficit rather than encoding, suggesting that memory aides and mnemonic techniques may be useful. Below average working memory performance, especially when ordering letters and numbers (letter number sequencing, scaled score 5), and in memorising and recalling strings of numbers (digit span, scale score 8), was observed.

-Summary: It was suggested that BP's lack of insight into his deficits, his concrete approach, impaired conceptual abstraction and impaired memory indicated a potential safety issue if he was unsupervised. Rehabilitation it was suggested should primarily focus on managing BP's difficult behaviours, his fatigue, and his impaired concentration, as these were all limiting his performance.

Table 5. Results from BP's neuropsychological assessment taken November 2002 (Cavit, 2002).

<p>-Overall Cognitive Functioning: BP had shown a slight improvement, from the below average range, to the bottom end of the average range, since his last neuropsychological assessment (March 2000). However, this does not reflect the huge variation in cognitive abilities that BP displays.</p> <p>-Conceptual and Verbal Abilities: BP had shown an overall improvement in concept formation, which was assessed as average (in contrast to a more concrete thinking pattern two and a half years prior). His score on the Comprehension subtest was in the low average range however, reflecting his continuing difficulties with judgement of his behaviour and of social situations.</p> <p>-Attention and Concentration: BP was able to attend to the test procedure for blocks of 40-45 minutes. Digits Forward remained at an average level, while Digits backwards indicated significantly impaired working memory. This working memory also affected his performance on the Arithmetic subtest, as more complex questions needed to be repeated to enable the information to remain in BP's working memory. BP did not appear to have difficulty with shifting sets, suggesting that his attention/concentration impairment is primary caused by a deficit in working memory.</p> <p>-Executive Function: BP appeared to have no difficulties switching set on the Trail Making Test. Some perseveration was observed on the Design Fluency and Colour Word Interference tests. BP demonstrated the cognitive ability to monitor and modify behaviour by correcting errors made on the Colour Word Interference test. Performance on the Card Sorting Test was one standard deviation below his expected pre-injury level of functioning, 20 Questions was also one standard deviation below the mean. On this test, BP tended to ask questions based on one feature (e.g. colour), after which he would resort to guessing targets. BP's performance on the Word Context Test indicated a difficulty in integrating multiple types of information. His spatial planning was shown to be significantly impaired, based on a well below average score on the Tower test. As number of required moves increased, BP's performance deteriorated, and a high number of rule violations suggested impairment in maintaining cognitive set.</p> <p>-Perceptual Organisational Skills: An improvement since last assessment on Block Design was observed, but performance on the Picture Completion Subtest was impaired, suggesting difficulty separating relevant from irrelevant information.</p> <p>-Speed of Information Processing: BP appeared within average range, but was slowed down significantly by his visual and motor impairments.</p> <p>-Memory and Learning: Significant improvements were noted in BP's memory. His score on the Auditory Immediate Memory Index was within the superior range. AVLT performance was at the 34th percentile (5/12 on the first trial and 8/12 on the last), consistent with his last assessment. Performance on the Visual Immediate Memory Index was at the top end of the average range. Delayed memory performance for all subtests was lower than immediate memory performance, suggesting high rates of forgetting.</p> <p>-Summary: BP had made some significant improvements since his initial assessment in March 2000. Ongoing problems were noted in working memory, social judgement and (deductive) reasoning, spatial planning and executive function. Ability to monitor and modify behaviour appeared to have improved somewhat, which suggests an increased ability to be able to respond to verbal feedback in daily functioning. BP's pattern of performance was characteristic of significant dysexecutive function, in line with his pattern of functional impairments (e.g., inappropriate and disinhibited social judgement and behaviour, impulsivity, impairments in planning, problem solving and deductive reasoning) he displays on a functional level.</p>

In April 2002, my role in BP's life began. This role was largely as a caregiver, as BP still required constant supervision at this time. The similarity in our ages (BP was 19 and I was 23 at that time) was considered to be a bonus by the supervising clinical psychologist as it was felt BP might be able to observe the modelling of appropriate behaviour from a peer. It was also felt that a background of study in neuropsychology and psychology in general would perhaps put me in a better position to administer different techniques with regard to social rehabilitation and behaviour modification. Officially the role was designed to be that of both a carer, and a provider of 'social rehabilitation'. It was planned that I would receive supervision once a month from a clinical psychologist. Briefly, my task was to include BP in the following:

- planning of the day ahead and outings,
- role-playing and setting of boundaries for social settings,
- providing relevant feedback with regard to social behaviour, and of setting up contingencies to help guide behaviour toward appropriate social norms. The goal for this was to enable BP to practice appropriate social behaviour, in order to help him regain as much independence in his daily life as possible
- Given the difficulties BP continued to have with regard to his impairments in responding to social cues and deficits in self-awareness, a primary goal was always to keep him safe. This issue of safety was and is paramount to the role.

A picture of BP in April 2001

It is important that when discussing different aspects of BP, that these should include not only his impairments, but also his strengths, or otherwise (many elements essential to BP's character and the sense of who he is are neither strengths nor weaknesses, but rather idiosyncrasies).

At the point that the writer met BP, the situation was as follows:

- BP showed many of the characteristics commonly referred to as 'frontal lobe syndrome'.
- He appeared rather hyperactive, with appropriate social interactions peppered with jokes, impersonations, changes in voice (both in pitch and volume), and swearing.
- Inappropriate touching was sometimes a feature (a common example being squeezing a person's hand when being shaken, or trying to hug

someone, regardless of whether they are a stranger, a friend or a family member).

- Dysfunction in the area of social interaction was particularly marked in unfamiliar situations. BP, in line with his impulsiveness, felt compelled to interact with as many different people as possible, and outings would typically involve at least one inappropriate interaction with a stranger. Of note though, while his self-awareness and ability to respond to social cues was severely impaired, BP had developed several techniques (or conversation starters) that from an outsider's standpoint would seem completely appropriate. One such routine was to say to a stranger "Excuse me, but you look familiar. Did you go to (BP's secondary school)? Oh, well my name's BP anyway...". Unfortunately, from this point on, the conversation would usually become more and more marked by inappropriate exchanges, accompanied by an inability to respond to social cues given by the person BP was talking to. Routine patterns of conversation were often demonstrated, and commonly involved jokes (sometimes inappropriately sexual in nature), age (e.g., "Guess how old I am. Go on, guess.") or his injury (which was a rigid series of statements that did not vary from conversation to conversation: "I fell off a two-and-a-half storey roof 1 1/2 years ago, landing down on the pavement on my head; broke both sides of my neck; lost all the bones on that side of my face and the vision from that eye (pointing to the left side of his face), but I've still got it where it counts- down there (points below his belt)".

- BP often attempted to begin a conversation with a stranger by flattery, another demonstration of his preserved capacities in planning and in understanding social interactions. Unfortunately though, the execution was often poor. His flattery usually pertained to age, or sometimes attractiveness, but was often characterised by excessive exaggeration, therefore negating the flattery. For example, BP often would say to a person he has just met "You must be about 23?" when they were in fact in their 50s. On occasion he would guess at an adult's age as being that of a child (e.g., calling a 30-year-old 13, not exactly a form of flattery). Another typical statement would be "Are you a model?", or "Have you been on Shortland Street?". These statements were reinforcing for BP, in that if the flattery worked the person would feel like talking to him, but if not, the response was often a laugh- either way the result was attention.

- BP would sometimes display an emotional level that was not appropriate to either the situation or the person it was directed at (saying "I

love you” 10 times or more in one day, or saying this to someone he had only met for the first time a few minutes prior).

- Awareness of the inappropriateness of his behaviours was limited, and feedback would be either ignored or disregarded a few moments later.
- Humour is a feature of BP’s personality that could and should not be ignored. His intention in a conversation is often to make people laugh, whether by jokes or just by acting “the class clown”. Set jokes that BP would remember would be regurgitated to new people on a daily basis (if not several times a day). Interestingly, over time, these jokes would often change. One joke became much longer than it needed to be, and began to involve new characters and routine phrases that were not integral (and were often distracting) to the joke. Sometimes they were filled with anachronisms (e.g., “A pirate walked into a bar or saloon hundreds of years ago...”, yet the punchline involves a steering wheel. The characters in the joke began to utter the same phrases in the same voices, yet weren’t actually necessary for the joke to work). Often a joke would not make sense, yet even with appropriate feedback he continued to tell it to other people. Often, the joke would be inappropriate to tell in a particular setting (e.g., shouting out a rude joke in the middle of someone’s speech at a bowling club). Yet even though BP’s humour could be considered unusual, inappropriate, and a hindrance to his social rehabilitation, there is no mistaking the charming nature of his sense of humour. It is integral to his identity, and while it needs to be made more appropriate to the situation at hand, its complete eradication should not be considered a goal of his rehabilitation.
- Impulsiveness, typical of frontal lobe dysfunction, was a major feature of BP’s behaviour. If not monitored, BP would wander to somewhere else in the neighbourhood. His wandering was often in complete disregard to his safety, whether it be while he was gone (e.g., opening the front door of a neighbour’s house and walking in, or running across a busy street), or what he was leaving behind (e.g., an unlocked house or a element turned on). Use of the phone also needed to be monitored, so as to prevent him from calling phone numbers of old friends and acquaintances several times a day. BP continued to smoke cigarettes, but due to his impulsiveness, it was important to set time limits between smoking (he always attempted to go 1 hour between each cigarette, but often found himself unable to). The presence of alcohol was also limited, to avoid impulsive drinking that such an opportunity could lead to.

- Limited attention span meant activities could only be focused on for a limited period of time. Most tasks were aimed at 40 minutes to 1 hour long, but were often foregone after 15 minutes. Watching a video or TV would usually be done in small bursts (half an hour or so). This impaired sustained attention was important for the planning of BP's rehabilitation and general daily tasks.
- Fatigue also played a huge part in BP's life. BP became progressively more inappropriate, disinhibited, agitated, impulsive, and impossible to reason with as he became fatigued. For this reason, a "lunchtime nap" was an integral part of BP's daily routine. 2 major activities were planned for each day (such as rehabilitation (physiotherapy twice a week, occupational therapy once a week, and later music therapy once a week) or social outings), with one in the morning, one in the afternoon, and a 1 hour nap in between. It was and is important to pay attention to the signs that might indicate the onset of fatigue, as from this point on BP's behaviour would often deteriorate quite rapidly. In such a situation, if his behaviour became uncontrollable (and he refused to respond to my feedback), at least one of BP's parents was available to be able to take him home (if not at the time, then within an hour or two). Generally though, this measure was a "safety net", and was employed less and less as my understanding of BP's behaviour increased. Some triggers to increasing fatigue were also identified, such as obvious ones like physical exertion, to the not so obvious triggers (e.g., loud music in a given setting).
- Another problem behaviour that BP displayed occasionally was aggression. It is important to distinguish however, that this aggressive behaviour was never serious, but was always performed in a joking manner, or as a means to some end, rather than as aggression or violence itself. In fact, it became apparent quite early, that unless very fatigued BP was not a violent person by nature. Nonetheless, he was often unable to see that in an unfamiliar social setting, the intent of a seemingly aggressive act was not as important as the initial appearance of his behaviour. For instance, he might try to make someone he has just met laugh by putting on an angry voice, raising his fist, saying "I'm going to smash you in" and pretending to punch him or her in the face, only to stop just short of doing so. It is obvious the trouble such behaviours can cause, regardless of the intent of the action. Trying to instill some understanding of this and control of such interactions is obviously highly beneficial both to BP's safety, and his independence.
- Motivation, which has also been linked to frontal lobe function, was another presenting problem for BP. It was very difficult to engage in activities

with BP that he did not find rewarding in the short-term. For instance, while BP was very able to cook a meal once he began, it was almost impossible to convince him to commence the task. BP would be quite content with a bowl of cereal for every meal of the day, if he was left to his own devices. Behaviour was often quite self-centred and self-focused. Helping out around the house was seen as a chore that was to be avoided if at all possible. In a way, this egocentrism was typical of a young teenager, who expects his parents to care for him in all aspects of his life. Instilling some sense of motivation for improvement, for helping others, and for heading towards long-term goals was a crucial step on BP's road to independence. A challenge was in finding activities and goals that BP was passionate about, something he had seemed to have lost since his injury.

- The other major facet that was severely impaired in BP's daily functioning, was his ability to plan and organise his day, week or life. This is a classic executive function task, and one that is often impaired after frontal lobe injury. My role here was to help him with planning ahead, as well as to help him to learn to make plans and organise for himself. It was also important to try to provide as much structure and routine to his weeks as possible. Often if plans were made, they would go out the window in a particular situation, due to his impulsiveness, so his ability to stick to a plan needed to be focused on the particular situation at hand.

The impairments that BP demonstrated could all be discussed in terms of frontal lobe damage. But BP continued to show many preserved capacities. For instance, his remote memory was phenomenal, and his ability to learn new things also quite good. He was also very skilled at mathematics, and had a large vocabulary (although his word finding difficulties sometimes resulted in the insertion of inappropriate words in the middle of sentences). His reading skills were impaired due to visual scanning difficulties, but if given time was shown to be quite skilled on a computer. His visuo-spatial ability also appeared to be highly functional, as demonstrated by a particular fondness (and knack) for Tetris and skateboarding computer games. While he did display some mood fluctuations (controlled for with medication), he was generally a happy and cheerful young man, and was quite a lot of fun to be around at times. BP displayed a few idiosyncratic tendencies (such as doing things in multiples of three, or having somewhat strange habits (e.g., saying "Mackerel, Doorknob, Titty Vegetable, Pardon Me, Pardon Me" every time he passes wind)), but these often just helped to make up the interesting and colourful character that BP was.

The cluster of behavioural and social impairments that BP had made the implementation of rehabilitation very difficult in many ways. Rehabilitation for severe traumatic brain injury involves many people and is a long process. It is made especially difficult by the myriad of difficulties associated with frontal lobe dysfunction, such as the impairments in motivation, self-awareness, disinhibition, social inappropriateness, fatigue, attention span and executive function mentioned above. In the next chapter, the rehabilitation for those with severe frontal lobe dysfunction is outlined, with particular reference to the difficulties of such in the case of BP.

Chapter 3: Brain Injury Rehabilitation

BP exhibits many behaviours and cognitive impairments that warrant rehabilitation. These include being impulsive/disinhibited, social inappropriate and impairments in motivation, attention/concentration, self-awareness/insight, and organisation/planning. The overall goal of rehabilitation is to ensure that BP attains the maximum degree of independence in his everyday functioning, and a successful community reintegration in all areas of his life in spite of his difficulties.

Brain injury rehabilitation is a relatively young discipline with limited empirical research. Nonetheless, there are many references to specific rehabilitation strategies and overall guiding principles (e.g., social learning theories) in the literature that can be used to aid in the rehabilitation for a person with individual needs and limitations (and strengths). TBI rehabilitation must be multi-disciplinary, so that the expertise of those in specific areas of rehabilitation can be combined to create a rehabilitation plan that tackles all the problems at hand. This chapter reviews the TBI rehabilitation literature, with particular reference to those interventions that relate to BP's presenting problems.

Phases of Brain injury Rehabilitation

Only over the last three decades have psychologists begun to direct their attention away from diagnostic assessment procedures, towards the rehabilitation of people with acquired brain injury (Wood, 1994). However there is a dearth of randomized controlled research in the area of rehabilitation efficacy, with much more research needed to clarify what works and for whom (Malec & Basford, 1996). This lack of research is due in part to the lack of commonly agreed outcome measures, as well as

the multi-factorial (and interdisciplinary nature) of brain injury rehabilitation, and the wide variations in severity and presenting problems (Burke et al., 2000).

Rehabilitation can be categorised according to the stage of recovery the person with TBI is in (Malec & Basford, 1996). In the *acute* recovery phase survival and the physical aspects of recovery are the primary focus with the input primarily medical.

Subacute rehabilitation of TBI patients involves the inpatient rehabilitation of those who are still severely disabled and are unable to participate in more intensive rehabilitation. While there are few studies investigating the effects of rehabilitation on those with severe disability or who are not deemed ready for more intensive rehabilitation, there is sufficient evidence to suggest that this subgroup is capable of significant functional recovery (Gray, 2000) and that rehabilitation may help to further ameliorate disability and reduce costs of long-term care for this group.

Postacute rehabilitation refers to those types of rehabilitation that are employed at a later stage of recovery, and are more intensive and focused on remediation of skills.

Postacute rehabilitation is guided by the goal of community reintegration, and can be employed during a period of inpatient care, or in the community many years post-trauma. Outcomes of postacute TBI rehabilitation are typically reported in terms of functional/psychosocial adjustment, living status, and/or vocational status. Malec and Basford (1996) reviewed studies of vocational outcome of TBI patients who had received some form of post-acute rehabilitation with studies that had no or unspecified post-acute rehabilitation. At follow-up 56% of the TBI patients who had received the post-acute rehabilitation were in independent work, training or homemaking, compared to 43% of the other TBI patients. Unemployment levels also differed between the two groups (29% and 47% respectively).

Efficacy of Brain injury Rehabilitation

While there is some indication that rehabilitation is beneficial for those with TBI, the question remains as to which rehabilitation works for whom. Bajo and Fleminger (2002) reviewed the few studies of rehabilitation efficacy after TBI in terms of 6 different patient characteristics (brain injury severity, presenting problem, complicating factors, rehabilitation readiness, demographic and socio-geographical variables). Results indicated that effectiveness of rehabilitation programmes was most affected by severity of brain injury, and that of presenting problems, impairment in executive function (in particular lack of self awareness) was most predictive of failure to respond to some cognitive and behavioural interventions including memory aids, cognitive remediation and operant behaviour modification. The negative effect on rehabilitation of lack of insight, coupled with being slow or unable to learn from one's mistakes (associated with frontal lobe syndrome), has been reiterated elsewhere (Ben-Yishay & Prigatano, 1990; Lam et al. 1988, cited in Malec & Basford 1996; Ogden, 1996). TBI patients with impaired self-awareness may find rehabilitation unnecessary, and insist on pursuing inappropriate short-term goals, providing a significant obstacle to the designing of an appropriate rehabilitation plan. Further limited insight may make it difficult to learn from experience or to gather a full understanding of the negative impact of behaviour (Port, Willmott & Charlton, 2002). While some other studies have not supported these findings, this is generally thought to be due to the lack of reliable, valid and standardised measures for self-awareness (Malec & Basford, 1996).

Wood (1994) described a rehabilitation programme that might be set up for those TBI patients who have behaviour problems as a result of frontal lobe damage. The initial stage would involve the establishing of an awareness in the TBI patient of the

dissimilarity between the behaviour he or she exhibits and that expected by others according social norms. Tools such as video-recordings of behaviour may help to establish this awareness, and to practice more socially appropriate behaviour. Discussion of how behaviour may be affecting people in his immediate environment, and role-playing (e.g., role reversal), may also be helpful in understanding why such behaviour is unacceptable to others. It is assumed that gaining self-awareness in this way (i.e., guiding their own understanding) the awareness gained is at a deeper level of processing than if trained to respond appropriately to specific behaviours in specific situations (Wood, 1994).

Cognitive rehabilitation

The cognitive deficits that arise after TBI are often treated with cognitive rehabilitation. While there is some evidence in the literature that some cognitive processes may be partially restored with the aid of rehabilitation, the majority of studies have not demonstrated a return to pre-morbid levels of cognitive functioning (Wilson, 1997).

Memory

Because memory complaints are so common amongst survivors of TBI research into the remediation and rehabilitation of memory deficits is rich and full. Rehabilitation can involve internal (e.g., the teaching of memory strategies) or external aids (e.g., memory notebooks, labels). Given that BP's memory (bar working memory, which has been shown to be impaired) is largely preserved when compared to his other cognitive and behavioural difficulties, a review of rehabilitation for memory is not included here.

Attention

While memory may be the most identifiable form of impairment in those who have suffered a brain injury, deficits in memory and concentration are equally important (Wood, 1994). Attention is one area of cognitive functioning that may be responsive to rehabilitation (although once again the restoration of function is more likely to be partial than full) (Wilson, 1997). Attention training generally involves goals based on the elimination of errors in increasingly difficult tasks, such as discriminating between relevant and irrelevant information (selective attention), increasing the speed of task completion (speed of information processing), and increasing the length of time of tasks (sustained attention) (Wood, 1994). Other techniques that may be useful for rehabilitation of attention include verbal mediation (i.e., verbalising the task at hand), repetition, controlling the rate of information presentation (i.e., slowed information processing speed may make accurate attention difficult; breaks will also help with the onset of fatigue), and increasing the salience of the information (e.g., linking information to events and people in the TBI person's life). Operant conditioning can be a useful tool in attention training, as rewarding attentive behaviour may increase attention and discrimination between relevant and irrelevant stimuli (Wood, 1990). While computerized attention training has been shown to have improve attention on the training task, there is little evidence to suggest that this generalises to attention in other, real-world settings (Wood, 1990).

Executive function

Executive functioning refers to processes involved in goal completion: anticipation, goal selection, planning, initiation of activity, self-regulation or self-monitoring, and use of feedback (Sohlberg & Mateer, 1989). Impairment in executive function is associated with frontal lobe damage. Given the complex nature of executive

functioning, one would assume that its rehabilitation would also be complex and difficult.

Sohlberg and Mateer (1989) in their review of the literature regarding the rehabilitation of executive function impairments, concluded that *structure* was the most important component. This is both at the individual level (e.g., breaking down activities into their components and rehearsing the effective completion of each component), and the environmental level (e.g., setting routines and planning timetables to adhere to).

Specific techniques in the remediation of executive function impairments include verbalization of a plan of behaviour before and during the execution of an activity. In their study of a group of closed head-injured patients with poor planning ability Cicerone and Wood (1987, cited in Sohlberg & Mateer, 1989) the verbalization of each move in the first stage was faded to whispered instructions in the next phase, and was reduced to the client ‘talking to himself’ in the final phase. The use of real-life situations to aid generalisation of planning abilities was also emphasised. Practice of planning, organising and breaking goals into subgoals has also been shown to be effective in remediating impaired executive function in a group of dysexecutive patients (Von Cramen et al., 1991,1992; cited in Manly et al., 2002). It has been suggested that many people with impaired executive function often neglect overall goals as they become too overly engaged in their current activity. The use of auditory cues to remind the person with dysexecutive syndrome of the current goals has been shown to be effective in a controlled study (Manly et al., 2002).

Sohlberg and Mateer (1989) argued that while a few isolated procedures for specific executive function deficits can be found in the literature, there was a great need for a rehabilitation model that addresses the full range of executive functions. They developed a model of executive function rehabilitation based on the separate areas of 'selection and execution of cognitive plans', 'time management' and 'self-regulation'. Each of these areas was then split up into between four to six subcomponents (e.g., Self-regulation: (1) awareness, (2) impulse control, (3) perseveration, and (4) environmental dependency). Assessment for each specific subcomponent is needed, and rehabilitation activities are suggested for these. The main principles for these tasks involve practice of specific skills, the use of diminishing supports, and the use of behaviour modification programmes. The importance of compensation within the rehabilitation model is also stressed (e.g., external cueing).

Computers in rehabilitation

The use of computers in rehabilitation was a very exciting prospect, particularly in the 1980s, when numerous software rehabilitation packages began to emerge (Wilson, 1997). While some encouraging studies emerged supporting the effectiveness of computer-based rehabilitation methods, these have been very few considering the huge increase in the use of computers in rehabilitation centres, and have not been supported by other studies (Wilson, 1997). There have also been criticisms due to the lack of generalisability to real-world situations (Ben-Yishay & Prigatano, 1990). Nevertheless, given the importance of computers in our world today, the use of computers in everyday life for people with TBI could be very helpful in the learning and maintenance of computer skills (a skill that is particularly useful when considering vocational replacement) and may be useful tools within a more holistic rehabilitation programme.

The future of cognitive rehabilitation will depend on whether further research provides sufficient evidence that direct stimulation of underlying mechanisms is actually reactivating dysfunctional areas, or whether rehabilitation professionals would be best to consider strategies for compensating for loss of function instead (Wood, 1994). Since the literature so far has been unable to show improvements in cognitive functioning as a result of rehabilitation consistently, it is perhaps prudent to look at compensating for impairments of cognitive functioning instead (not that compensation and remediation of function are mutually exclusive- they both are vital to successful rehabilitation). Wilson (1997) suggests three approaches to compensation of lost cognitive ability:

- (1) Avoiding problem areas by changing or restructuring the environment,
- (2) Functional adaptation (i.e., finding another way to achieve the goal), and
- (3) More efficient use of residual skills.

These three approaches are essential in developing a practical rehabilitation plan for a TBI patient, as it is a lot easier to restructure the environment, adopt different methods and use preserved skills in day-to-day living, than it is to improve those skills that have been lost or impaired as a result of a TBI.

Behaviour management

Principles of behavioural assessment and behaviour modification can be extremely useful in designing rehabilitation programmes for people with cognitive or behavioural impairments following TBI. A behavioural assessment would include accurate identification of the problem behaviour (e.g., cognitive impairment, problem behaviours such as aggression, inappropriate behaviour, excessive swearing, non-

compliance, inappropriate sexual behaviour, interpersonal skills deficits, and impulsivity; Wesolowski and Zencius, 1994), the setting of clear goals, baseline measurement of the behaviour, development of a rehabilitation/treatment plan, beginning of treatment, monitoring of progress during treatment, and adjusting the treatment according to observed results (Wilson, 1997).

Contingency management, or the setting up of systematic reinforcement (of pro-social behaviour) and punishment (of problem behaviour) schedules, is used for target behaviours. An assessment phase should contain the definition of the targeted aggressive behaviours, the collection of data on the frequency and severity of these behaviours (baseline data), and hypotheses regarding the antecedents that are triggering them, and the consequences that are maintaining them. Reinforcement schedules are then introduced (e.g., praising every time prosocial and non-aggressive behaviours are used instead of aggression), and the frequency of the behaviour is monitored over a period of time to see if the reinforcement contingencies are decreasing the problem behaviours. Extinction, or the decrease in behaviour frequency in response to the removal of positive reinforcement, is a powerful concept in this model of rehabilitation. One common form of positive reinforcement gained from displaying problem behaviours such as aggression is the attention gained from those in the immediate environment (rehabilitation professionals or otherwise). For this reason, ignoring such behaviours can be a very effective means of decreasing a target behaviour (if it is safe to do so).

Much of the literature regarding behavioural management of challenging behaviours refers to the management of aggression. Aggression from a social science perspective implies that (1) the aggressor is seeking to control a situation so that perceived needs

are met, (2) aggressive behaviour is problematic because people in the environment judge the behaviour as violating norms, and (3) for behaviour to be pro-social it must meet the individual's perceived needs and respect situational norms (Haffey & Scibak, 1989). For this reason it is important to establish: (1) the TBI's individuals perceived needs (i.e., what the person's is hoping to get out of the aggressive behaviour), (2) the TBI individual's awareness of norms being violated (e.g., does he or she realise is it inappropriate to strike out?), and (3) potential alternative behaviours that would meet perceived needs *and* satisfy norms (e.g., encouraging the person to ask politely for what they are after- establishment of this more successful method will be positively reinforcing).

There has been some debate as to whether accurate self-monitoring in TBI sufferers is necessary to benefit from reinforcement schedules (Knight et al., 2002). Differential reinforcements schedules are a common tool in the management of challenging behaviour. This may take the form of differential reinforcement of incompatible behaviour (DRI), of other behaviour (DRO), or of low rates of behaviour (DRL). DRL has been found to be particularly useful in the management of challenging behaviours for people with acquired cognitive impairment (Knight et al., 2002). DRL employs the use of a reward schedule, such that when high frequency challenging behaviours occur at a lower frequency for a given time, a reward is given. The target frequency can start at an easily attainable level, and then decreased throughout the course of the rehabilitation. Knight and colleagues (2002) questioned whether the ability to accurately self-monitor one's behaviour was necessary for DRL to be effective, and found (with the use of three case studies) that, surprisingly, self-monitoring impairments did not appear to be necessary for gains to be made. This provides yet

more support for the employment of behaviour modification techniques and rehabilitative efforts based on the principles of operant conditioning.

Haffey and Scibak (1989) describe impediments to the application of social learning principles.

- Profound impairments in attention may make learning associations between behaviours and consequences extremely difficult.
- Motivation problems may also mean that no reinforcement is found to be powerful enough to elicit an adaptive or prosocial behaviour. In this situation, the avoidance of putting in the substantial effort needed is more reinforcing. In such a situation, Haffey and Scibak suggest the use of limited access to other reinforcers (e.g., phone calls), contingent upon the performance of the target prosocial behaviours (although the ethics of such an approach must be considered, and it should only be used if other approaches have failed).
- Generalisation from one situation to another, may be limited (i.e., substitution of aggressive behaviours for prosocial behaviours may occur only in the specific situation in which it is taught).
- Another reason for ineffectiveness of social learning theories could be due to the reinforcement for some behaviours not being salient to rehabilitation staff, and that problem behaviours are being inadvertently reinforced. On-site reviews by other staff, or the use of video recordings, may help to clarify such inadvertently reinforcing behaviours by rehabilitation providers.
- Lastly, behaviour modification strategies may be seen as degrading and demoralizing.

Specific techniques that may be employed for each are shown in Table 6.

Table 6. Procedures that can be used in the rehabilitation of problem behaviours (Wesolowski & Zencius, 1994)

<i>Noncompliance</i>
- The Premack Principle: Reinforcing a low-frequency behaviour (e.g., cooking a meal) with a high-frequency behaviour (e.g., watching TV)
- Token economies, such as point systems (the accumulation of points for prosocial behaviour, to use for rewards later)
- Leveled programs (the use of reinforcement for different levels (e.g., frequencies) of behaviour as the rehabilitation progresses, starting at easy-to-attain targets and becoming increasing more difficult)
- Behaviour contracts (to prevent arguments on previously agreed targets and reinforcements)
- Antecedent control techniques (e.g., maps, pictures, verbal cues, restructuring of environment)
<i>Aggression</i>
- Devotion of time to altering social conditions rather than just talking about them
- Consequence management (Premack Principle, token economies, behaviour contracts)
- Videotape feedback
- Alternative behaviours (teaching prosocial behaviour)
- Repeated use
- Redirection (of focus from an aggression-provoking stimulus to other stimuli in the environment)
- Stress management training (e.g., teaching breathing techniques in aggression-provoking situations)
<i>Inappropriate sexual behaviour</i>
- Dating Skills Training (e.g., teaching the substitution of inappropriate sexual behaviours for more appropriate ones)
- Scheduled feedback
- Transfer of stimulus control (discriminating between environments, and explaining appropriate places for intimacy)
- Self-monitoring
- Behaviour contracts
<i>Interpersonal skills deficits</i>
- Social skills training (in small groups)
- Point systems
- Contingent and scheduled feedback
- Pre-teaching (practicing beforehand, role-playing)
<i>Impulsivity</i>
- Checklists (instead of verbal instructions)
- Feedback
- Self-recording (to aid self-awareness; without the right motivation though, TBI patients may be prone to use deception and under-report impulsive behaviours)

Deaton (1990) also suggested various strategies for promoting behavioural change in children and adolescents, based on the social learning principles of antecedents and

consequences. Table 7 includes examples of these strategies. Once again, these interventions must be applied consistently, and the frequency of the behaviour must be monitored to assess the interventions' effectiveness. Shifting the control of behaviour from external sources to the person with TBI is essential in order to aid generalisation of learned behaviour to other situations.

The behaviour management plan is usually developed by a clinical psychologist who must also teach specific behavioural strategies to both client and caregiver/s. The plan must foster a realistic sense of hope.

Although anecdotal evidence and case studies have supported the effectiveness of cognitive-behavioural interventions for people with TBI, systematic studies into the efficacy of such approaches is lacking (Malec & Basford, 1996). Webster and Scott (1983, cited in Wood, 1990) demonstrated that a cognitive-behavioural procedure based on a verbal mediation strategy was successful in improving attentional capacity and memory in a patient 2 years post-TBI. This cognitive-behavioural strategy focused on the regulation of some aspects of behaviour in order to shift the individual's focus onto them, elevating their significance, salience, and awareness of the behaviour.

Information Provision & Counselling

The provision of information and of counselling after TBI to assist both victim and family deal with the catastrophic effect of the event and the consequent changes in their lives is important for both client and family. Contact with the person with TBI, family, and other rehabilitation professionals must be maintained. Appropriate and specific feedback (with the negative counterbalanced with positive feedback) at the

Table 7. Intervention strategies for antecedents and consequences of challenging behaviour in children and adolescents (adapted from Deaton, 1990, p.241).

Possible Antecedents	Possible Interventions
Does not understand task demands	-provide clear, concrete instructions, in writing if needed -model task completion
Does not begin task	-give prompts -reinforce each instance of initiative, however minor (e.g., picks up a pencil)
Unable to do task	-simplify task -provide training in underlying skills
Is not motivated	-make task more interesting or relevant -give desired rewards for task completion
Possible Consequences	Possible Interventions
Avoids failure or frustration by not complying	-alternate difficult tasks with easy enjoyable ones -provide tasks at which the child will succeed
Gets out of doing task	-must complete task before going on to other activities, regardless of time to completion
Receives attention for not doing task	-is timed out or ignored while noncompliant -is reinforced with attention for cooperation -other children in environment are reinforced for their cooperation
Gets to assert independence/control over situation	-may be offered choices when appropriate

right times is essential, as is a close working relationship with the family (the family will be the ones who are using the behavioural techniques more than anyone else, and need to know and appreciate how and if they are working) (Wesolowski & Zencius, 1994).

Vocational Rehabilitation

Supported work placements have become a standard feature of most post-acute rehabilitation programmes (Malec & Basford, 1996). This involves matching the interests and skills of the TBI individual with a (often-volunteer) job, supported by the presence of a job coach or occupational therapist. These supports are gradually

decreased as the individual's independence in the role grows. While supported employment is widely regarded as being a successful means of vocational and community reintegration, this may be a lengthy process. Wehman et al. (1993, cited in Malec & Basford, 1996) found that the mean intervention needed during the first 6 months for 'least difficult' TBI clients was 211 hours, while 'most difficult' clients required 426 hours of intervention time. Moreover, while 1-year job retention rates for the former group was at the relatively high level of 78.5%, only 26.7% of those 'most difficult' clients remained in the position at this time. It is clear then that supported work placements, while successfully reintegrating many TBI sufferers into vocational employment, is a time-consuming and expensive approach, with variable results (particularly for those with more profound impairment).

Pharmacotherapy

An interest in using pharmacological interventions for the amelioration of psychological/behavioural disturbances or to aid cognitive rehabilitation after TBI has been growing in recent years, (Lux, 1996). The current consensus is that pharmacotherapy can be extremely useful for some people recovering from TBI, but more rigorous and systematic studies are needed to determine the true effectiveness of pharmacological treatments in TBI rehabilitation (Lux, 1996), especially since some pharmacological agents can have undesirable side effects.

For example, medication is sometimes required due to the co-morbidity of TBI with psychiatric disorders (e.g., depression). While selective serotonin reuptake inhibitors (SSRIs) are widely regarded as the most successful of antidepressants (Zafonte et al., 1999) drug interactions may lessen the effect of antidepressants and may be associated with a greater frequency of post-traumatic seizures. Wroblewski (1990,

cited in Silver & Yudofsky, 1994), in a review of 68 patients taking antidepressant medication, found that 6 patients had seizures during the baseline period (before medication), 16 during antidepressant treatment and 4 after treatment was discontinued. 20% of the sample had seizures shortly after introduction of the antidepressant medication. The bottom line appears to be that antidepressants can be used safely and effectively for those with TBI, but should be introduced cautiously and carefully monitored (Silver & Yudofsky, 1994).

The future of rehabilitation- the example of the Whatever it Takes approach

The Whatever it takes (WIT) approach to community-based TBI rehabilitation services was developed by Willer and Corrigan (1994), and reflects a practical, function-focused, individual-focused method of rehabilitation delivery after TBI. This approach has basically come out of the realisation that there is no quick cure for TBI rehabilitation and that there is unlikely to be one in the near future at least. Instead, practical solutions to problems faced by brain-injured individuals are needed, with a focus on day-to-day problems. There continues to be friction regarding the best way to 'treat' patients, between the medical model (a disability-focused approach, where patients are treated by clinicians for their deficits) and an independent living model (where the goal is self-determination for the TBI person who is encouraged and supported by the removal of boundaries to independence in education, employment or everyday living e.g., break times for fatigue, structural changes to the house).

The medical model of rehabilitation is obviously more suited to the recovery of function in the early stages of recovery, or for those with more profound impairments. While most would agree that the independent living model would be more helpful for restoring the functional independence of community-based TBI individuals, complete

self-determination may not be possible (due to persisting impairments). A combination of these two models, adopting concepts of treating deficits and encouraging the maximum self-determination possible for each individual would be the logical solution. This is the objective of Willer and Corrigan's (1994) model.

The WIT model of TBI rehabilitation adopts 10 principles that are used to guide rehabilitation at every step. These are presented in Table 8. Basically, the WIT model is a realisation of the complexities of TBI rehabilitation, and that rehabilitation programmes need to be based on each individual's needs and capabilities for the different day-to-day problems and situations that apply to them alone. It is also an attempt to incorporate the trial-and-error nature of rehabilitation into a semi-structured model. One never knows what forms of rehabilitation will work for each individual with TBI until they have been tried.

In summary, rehabilitation programmes should be based on theories and on the literature (i.e., what has worked in he past), but then be flexible enough to abandon original ideas and try new rehabilitative techniques when it is clear the first has not been not been effective. In the practice of brain injury rehabilitation, guidelines and experience are probably the best tools that rehabilitation professionals have. The future of TBI rehabilitation is likely to try to adopt real-life, practical solutions to everyday problems, and create models of rehabilitation that is both based in theory, yet is flexible enough to cater for the myriad of outcomes that can be expected from each individual with TBI.

Table 8. The ten principles of the Whatever it Takes (WIT) model of brain injury rehabilitation (adapted from Willer & Corrigan, 1994, pp. 649-657).

- | |
|--|
| (1) No two individuals with acquired brain injury are alike, |
| (2) Skills are more likely to be generalised when taught in the environment where they are used, |
| (3) Environments are easier to change than most people, |
| (4) Community integration should be holistic (i.e., assessments need to be linked to real world situations; strengths are as important as deficits; all aspects of reintegration in different areas of the person's life should be incorporated into a rehabilitation plan), |
| (5) Life is a place-and-train venture (i.e., eventual living situation goals should be attempted as soon as possible, with the necessary supports to make it work, and gradually decreasing supports as independence returns), |
| (6) Natural supports last longer than professionals, |
| (7) Interventions must not do more harm than good (e.g., behaviour management can sometimes demoralise a person with TBI; excessive support may increase dependency and decrease motivation to engage in rehabilitative activities), |
| (8) The service system presents many of the barriers to community integration (e.g., funding; fragmentation of service delivery), |
| (9) Respect for the individual is paramount, and |
| (10) Needs of individuals last a lifetime, so should their resources. |

BP: Multi-disciplinary TBI rehabilitation

BP's rehabilitation from the point of injury up to the time where my intervention began (April 2001) was multifaceted, commencing at the private rehabilitation centre, on through the many interventions set up at home. Speech language therapy was intensive (twice daily at the rehabilitation centre), as well as physiotherapy (for general mobility, but progressively more focused on eliminating the weakness on his right side), occupational therapy, and interventions by psychologists (both for behaviour management and in a more counseling role). All of these interventions have continued up to the present time (although SLT became infrequent after the acute stage of his recovery, and no interventions were felt necessary following a final language/communication review completed November 2002).

As stated earlier, BP sustained a severe TBI, and has received acute, sub-acute, and now post-acute rehabilitation. His multiple difficulties have been assessed

and monitored by a variety of rehabilitation specialists. Various interlinked rehabilitation plans have been developed incorporating as far as possible the theoretical approaches endorsed in the literature, mindful of BP's own individual strengths and weaknesses and those in his environment. The following section details the rehabilitation and the outcomes of that rehabilitation since his discharge from hospital almost 3 years ago. While this rehabilitation has been grouped into separate sections, it is important to remember that these rehabilitation professionals do not work in isolation. The rehabilitation of a person is a collaborative, multi-disciplinary approach. To emphasise this, and to provide a clearer grasp of BP's rehabilitation over the last 3 years, a timeline of the rehabilitation from all rehabilitation providers is shown in Figure 4.

TBI rehabilitation is made very difficult by the nature of frontal lobe dysfunction, and the complexity of real-world situations. The characteristics of impaired self-awareness, attention, executive function, and motivation, as well as non-compliance and constant lying, complicate neuropsychological rehabilitation in their own ways, making it hard to use textbook solutions in everyday environments. Also the need to keep BP safe often has to override the objectivity that may be asked for in a rehabilitation plan.

Psychotherapy

The role of the clinical psychologist has been a vital one in BP's life and the lives of family and caregivers. The provision of information about head injury is important. It was also very important to ensure that the concerns and grief experienced by BP's family and by BP himself were listened to. The counselling role of helping BP and his family to deal with the loss and change resulting from the TBI, and helping them to cope with the significant stress that caregivers of people with TBI often face (see Chapter 5) is vital to the overall functioning of those concerned. Clinical psychologists have tended to occupy the role of overseeing the whole of BP's treatment, and the bringing of the many rehabilitation professionals together for a better structured rehabilitation plan (although the assigned team leader during BP's two-month in-patient stay following hospital discharge was a rehabilitation nurse specialist). BP's parents were instructed of ways in which to approach the challenging behaviour that BP was expressing, based on the social learning theory principles mentioned above. When new behaviours emerged, or problem behaviour began to escalate, the clinical psychologist's role would be to try to determine the causes of this behaviour change, or the consequences that are reinforcing them, and remove

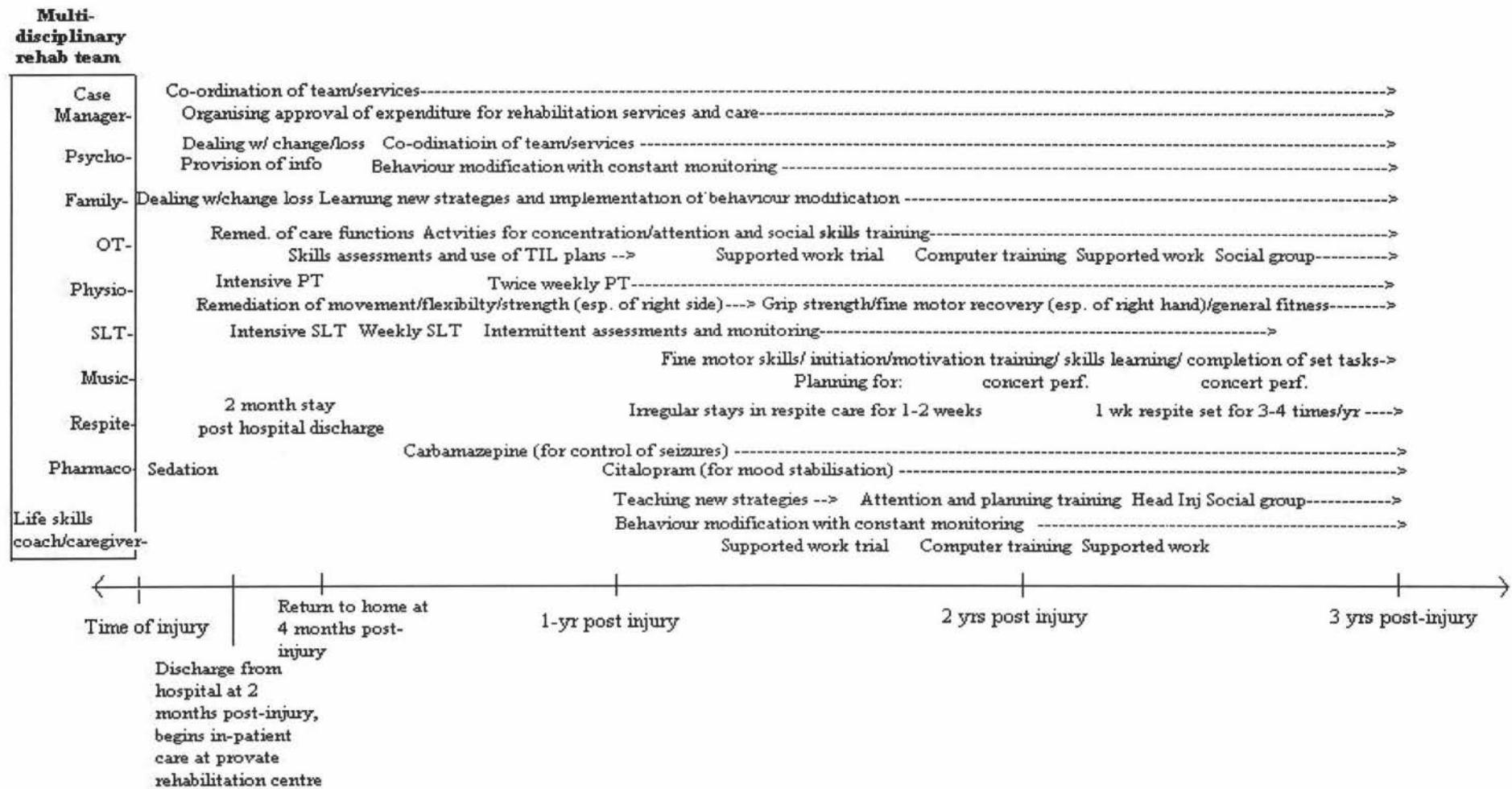


Figure 4. Time-line of BP's multi-disciplinary rehabilitation

these causes, or try to find new strategies to combat them (e.g., using behaviour modification, or thinking about pharmacology changes).

BP's psychologist at the present deals with BP on both an intermittent basis, or if the need arises. Meetings with BP and his parents are set for every few months or so decreased from a more frequent level of every few weeks (e.g., three weeks) (it was felt that frequent sessions with myself were more beneficial than frequent sessions with BP). These sessions are a time where BP can express the issues that are bothering him, what his goals are, and discuss ways in which he may work toward those goals. His parents are able to discuss how BP has been faring, what changes have occurred in his behaviour, his situation, and how they are coping on a day-to-day basis. Strategies can be discussed for eliminating problem behaviours, and substituting them with more adaptive and prosocial behaviour. Behaviour strategies such as pretraining, external cueing, and setting a structured routine to BP's week can be taught and discussed, with a particular focus on maximising independence for BP, minimising stress on a day-to-day level, and increase BP's sense of wellbeing. As problems arise, BP parents are able to contact the psychologist and discuss ways to remedy the situation.

BP's psychologist has been invaluable in the training and supervision for myself too, both as a caregiver, and as a provider of social rehabilitation, over the last two years. Over this time, meetings have been set every 3 or 4 weeks, with the option of contacting her anytime if a particularly challenging behaviour or situation arises, or for any general query. Behavioural management strategies such as pretraining, roleplaying, the setting of consistent contingencies, use of the Premack Principle, verbal cueing, providing routine and structure, and constant feedback are discussed, and explained in a way that can be both general or specific to a given situation. She has provided tools by which to make the day-to-day care of BP more manageable. Ethical dilemmas such as how to deal with inappropriateness of displays of affection, the importance of remaining in the role of a care professional rather than filling gaps in his social support (i.e., the lesson that friends are paid to be so is *not* one that should be taught), and dealing with alcohol and drugs in BP's life (i.e., the conflict between the social norm of drinking and his ability to cope with alcohol) may be discussed and clarified. Overall, as my role as a caregiver is to be a guide in BP's life, BP's clinical psychologist is my guide for doing so. The skills and expertise that she brings in the management of challenging behaviour and in overall scope of BP's

psychosocial functioning (along with those skills and experience of BP's very capable parents) have been the most valuable tools available in dealing with BP's behaviour on an everyday basis.

Family

BP's family has by far the most contact with BP, and so are the best people by which to judge his overall functioning at an everyday level. They also have an extensive knowledge of his life before the accident, so can put his challenging behaviours in perspective of his premorbid personality in a way that no-one else working with BP is able to. A vital part of the rehabilitation of BP has been the part that his parents have played. BP's parents employ the use of behavioural modification techniques, such as the use of reinforcements to prompt helpful behaviour around the house, and are able to see on an everyday level which situations and consequences are likely to lead to the elicitation of problem behaviours. BP's family have been extremely determined to increase the level of experienced independence for their son, and (due to the position as family) may be particularly more motivated to put in substantial effort for small gains than other rehabilitation professionals. The role of the family is vital to the daily functioning and behavioural management of BP, perhaps more so than all other people involved in his rehabilitation combined. Their extensive experience in dealing with BP, both pre- and post-injury, must be incorporated into his overall rehabilitation plan in order for it to be successful. The efforts of BP's parents and family have been substantial and never-ending in his rehabilitation.

Occupational therapy (OT)

Occupational therapists have been involved in the community reintegration of BP in many ways. Initially, rehabilitation in this area focused on the gaining of independence through focusing on BP regaining the ability to self-feed, groom, dress himself, bathe, and toilet (during BP's inpatient stay 2-4 months post-injury). The aiding of the return of these skills was quite successful. Once BP's ability to perform these functions improved, training for independent living (TIL) plans were used to assist in his community reintegration². These were put into effect from the time of BP's return to home (4 months post-injury). Activities such as card games were used to aid cognitive skills, particularly in improving concentration and sustained attention. Going to social situations (e.g., a public library) in order to practice selective attention, and practice appropriate social

² TIL = Training for Independent Living. These are plans, usually in approximately 13 week blocks, where goals and interventions are set out. TIL plans are used extensively by occupational therapists.

behaviour increased the difficulty of these activities (implemented from approximately 1 year post-injury). The use of skills-based activities were employed, such as building a model aeroplane (i.e., concentration, task completion), or grocery shopping (practising sticking to the task at hand and behaving appropriately in social situations) (from approximately 8 months to 1 year and onwards). Difficulties with wavering interest and motivation made many skills-based activities unsuccessful. Other techniques useful for BP's daily functioning include the teaching of mental cues, and relaxation training (18 months post-injury and onwards). Approximately 18 months post-injury supported vocational training was attempted, cleaning out the back at a department store, but the distractibility of customers, lack of motivation and sustained attention, and inappropriate social behaviour (e.g., swearing) made the vocational training situation unsuccessful. A later attempt at supported work placement (at a garden centre) was also discontinued due to distractibility, poor concentration and inappropriate behaviour. A computer-training course (2 years post-injury) was discontinued after two sessions for similar reasons.

A formal (i.e., staffed by a private rehabilitation centre) social group therapy weekly session was introduced to BP's rehabilitation plan approximately 2 ½ years post-injury. This involves a group of TBI individuals with social behaviour problems to practice appropriate and prosocial behaviour in a supportive environment. Activities usually involve group outings (e.g., ten-pin bowling alley, museum), but may also involve such activities as cooking a meal together. BP designed and completed a newsletter for the group on a computer over a period of weeks, as part of his rehabilitation plan. While this was very successful for the one newsletter, and much praise was given regarding it, BP refused to put in the effort required for further issues. Nonetheless, attendance at this social group for two to three hours every Friday continues, with strict boundaries enforced to ensure that it is successful (BP was unable to attend for some time due to the breaking of these boundaries).

OT sessions are currently set at one 1-hour session per week, on top of the Friday social group. These sessions involve the cooking of a meal one week (although very often the initiation of this activity is unsuccessful), followed by a social outing (e.g., museum, a café) the following week. Art therapy classes (in a social setting for those with or without disability) are to be introduced in the near future. TIL plans are usually set at a 13-weekly interval, focusing on specific activities to be undertaken to assist in the rehabilitation of those behaviours that are hindering

proper community and vocational reintegration (e.g., attention, fatigue, insight, social behaviour).

Physiotherapy (PT)

Intensive physiotherapy during an initial in-patient stay at a private rehabilitation centre focused mainly on BP's hemi-paresis (i.e., the weakness on his right side). His initial status was that the limbs on the right side of his body were paralysed, and he showed weakness on his left side, although his left-sided movement quickly returned to close to premorbid levels. Physiotherapy focused on the building of strength and flexibility of his right arm, right hand, and right leg, with the goals including the returning of the ability to walk, run, and grip on this side. Rehabilitation in this area was particularly successful, as BP's motor function (both gross and fine) improved rapidly. Physiotherapy has continued to this day, with the same goal of returning BP to a state where he has freedom of movement, strength and fine motor skills on his right side equal that of his left, as well the promotion of general fitness. These sessions are for one hour, twice a week. While improvements in fine motor skills and strength on his right side continue to improve, this improvement has slowed quite dramatically. The rehabilitation of the fine motor skills in his right hand is a particular focus currently, with the use of small weights and grip machines, as BP's ability to write with this hand remains particularly limited (his premorbid dominant hand was his right hand).

Speech language therapy (SLT)

Intensive SLT during BP's inpatient rehabilitation initially focused on initiation of speech and later shifted to tone, fluency and content as BP's speech production rapidly improved. After discharge, speech and language was monitored and assessed periodically, with SLT continued at two hours per week immediately following discharge (with a review after six weeks). Over the following years, assessments suggested improved listening and interactive skills, improved pitch, tone and cadence of speech.

Difficulties with visual scanning for reading, hand movement for writing, and cognitive and behavioural difficulties for use and content of speech remain a problem, as does fatigue. Currently BP's speech and language skills have been assessed at a level where further SLT intervention was not considered necessary (by BP's original speech language therapist), but that attendance at a social group therapy (see above), music therapy, art therapy and behavioural interventions that

focus on improving other areas of his function (particularly attention) will continue to improve his communicative abilities.

Music Therapy

Music therapy was begun in May 2001. Treatment objectives included improvement of voice quality, verbal fluency, improved concentration/attention, improved interactive/listening skills, improved fine motor skills (particularly with right hand and fingers), and skill learning practice. Plus, BP's love for music means that this therapy can provide a means of self-expression, self-pride, and enjoyment. Tasks have included the playing of piano, guitar, drums and harmonica, singing practice, the learning of new songs, the creation of lyrics to songs, teaching of music theory (and going through Grade 1 music theory workbooks in preparation to sit the Grade 1 theory exam in the near future), and the performance of a 'gig' in a social situation (a private brain injury rehabilitation centre). BP has responded extremely well from this form of rehabilitation. Gains have been made toward the treatment objectives mentioned above, and have provided great enjoyment and a real sense of pride. BP's father, a keen and talented guitarist and musician, has had a lot of input into this therapy, and in the learning of new songs for concerts (of which he plays back-up guitar to. Plus, motivation is one of the greatest obstacles to rehabilitation for BP, but his love of music makes this rehabilitation much more motivating. These sessions are held once a week, for one hour, and are set to continue for at the foreseeable future.

Respite care

The availability of respite care for BP's family is important. While respite care has been rather infrequent since he returned to home, the need for it has required that three stays in a private brain injury rehabilitation centre (for approximately 7 days each time) per year has been set in his rehabilitation plan. Over this time, routines are adhered to as much as possible, such as regular caregiving days, and the appointments of physiotherapy and social group. This respite, while a little distressing for BP (it is a centre for people with more profound impairments, particularly physical impairments, so BP does become bored and feel trapped after a few days), has been vital in letting BP's parents escape on a holiday, go to work functions elsewhere, and just recharge their batteries. This way, the likelihood of anxiety and stress building up in his family is decreased, such that they may enjoy a better quality of life. They are also then able to provide better quality care and rehabilitation and a better home environment for BP than if

burdened with stress, depression or anxiety. BP has also become chronically fatigued at times, causing his general behaviour to deteriorate (perhaps due to the presence of strangers working on his kitchen, making it harder for BP to have adequate rest). Respite care has provided a quiet environment with a very structured timetable, such that chronic fatigue has largely dissipated.

Pharmacotherapy

BP has been taking carbamazepine (Tegretol) for the past 2 ½ years, and the SSRI citalopram (Cipramil) for 2 years. Carbamazepine, while being taken primarily for the control of post-traumatic seizures, may also benefit BP's rehabilitation in other ways. Carbamazepine is known to reduce agitation in TBI patients post-injury (especially in the early recovery phase) (Lux, 1996). It is also thought to have less adverse cognitive side effects than other anticonvulsants (Glenn & Wrobleksi, 1986, cited in Zafonte et al., 1999).

Citalopram, administered to stabilise mood and alleviate depressive or dysphoric episodes, may also have additional beneficial effects. SSRIs such as citalopram can be used to treat emotional lability or emotional incontinence (i.e., where involuntary emotional expression is incongruent with the actual emotions being felt by the patient at the time) after TBI (Lux, 1996). Patients often show improvements in emotional lability within five days of treatment (Silver & Yudofsky, 1994). This is a common post-traumatic symptom exhibited by BP, and the use of citalopram may be helping to control the extent to which BP expresses these emotional outbursts.

Care-Giver/Coach

My role in BP's rehabilitation began in April 2001. This role was to provide 'social rehabilitation' for BP, and to act as a caregiver for BP 8 hours a day, 2 or 3 days per week. BP's clinical psychologist felt that being a similar age to BP (he 19, myself 23), it would enable BP to observe the modelling of appropriate behaviour by a peer. Funding was provided for supervision by the clinical psychologist, in the form of a 1-hour session every three or four weeks. These sessions would involve the training of behaviour modification and management strategies for the behaviour problems and cognitive deficits that BP presents with. Each session would generally consist then of a discussion of how the suggestions from the last session were implemented, what problems existed in this implementation, and how BP reacted to the rehabilitation (i.e., whether or not challenging behaviour decreased). Social learning principles of antecedents and

consequences, reinforcement contingencies, and the modelling of appropriate behaviour guided these strategies. It was also important to consider BP's cognitive limitations in the implementation of behavioural management strategies, and how interventions might impact BP's overall psychosocial functioning.

A typical day of caring for BP would start with the planning of the day ahead on a whiteboard. One major activity is planned for the morning, and one for the afternoon, with a 1-hour rest at midday to make sure fatigue is controlled. Even with rest periods, the inappropriate behaviour has usually been more prominent in the afternoon, so those social activities that would entail substantial effort are scheduled for the morning. A café visit, unless other activities have already been set in advance, is a favourite part of the day for BP, so given the highly reinforcing nature of a café visit it is often placed later in the day, contingent on the completion of tasks. Due to difficulty in motivation for task completion (e.g., cooking a meal, cleaning his room), Monday and Wednesday have been set as days when morning café visits are strictly forbidden (barring exceptional circumstances), and given that a substantial amount of effort has been expended in the morning, this café visit is used as a reward. This has tended to work quite well, although impulsivity and impatience sometimes means that BP will not rationalise and will leave the house on occasion to embark on a café excursion. Reinforcement contingencies (e.g., the cancelling of a café visit the following day) are employed in an attempt to prevent this.

While café visits are used as a reward, they are also used as a rehabilitation exercise. Beforehand, the café visit is planned on BP's whiteboard. The activity is broken down into its components (e.g., getting to the café, entering the café, ordering, drinking the coffee, leaving the café). Set goals are decided on based on behaviours that BP has been having trouble with (e.g., swearing, inappropriate sexual joking). A small number of these goals (4 or 5) are agreed on together, with BP being guided to suggest most of the goals himself. Role-playing is used on occasion, in order to practice appropriate social behaviour in specific situations, and to aid in self-awareness of how others might respond to inappropriate behaviour. Reinforcement contingencies are set beforehand, in order to give incentive to the use of prosocial instead of antisocial behaviours. BP is encouraged to initiate suggestions of rewards for an outing that has been judged to go well (based on the set goals), and these are then modified if needed (i.e., BP often chooses quite catastrophic and unrealistic consequences, such as 'I won't go

to a café for a month'. A response used may be 'That's quite a long time, don't you think? How about if it goes well we will go to that new café we were going to check out, but if not, we'll have to save it for another day?'). Consequences are generally worded in a positive way, with the use of rewards preferred to punishments. It is always better for the initiation of the plan, goals, and consequences to be performed primarily by BP, to promote maximum use of his impaired executive function. Practice of these skills will make planning tasks on his own easier, thus increasing independence. Repetition of goals is requested when reaching the café (to keep them fresh in his mind, and assess that attention and working memory are functioning sufficiently). Feedback, particularly positive feedback for prosocial behaviours (or the lack of an inappropriate behaviour in a situation that has been known to trigger them), is given constantly and is kept specific to the exact behaviour. Appropriate behaviour is modeled, and social cues that BP may not pick up on are pointed out (e.g., interrupting someone who is trying to read the paper). Verbal prompts are given to remind BP of the goals and consequences. A 'two-strike rule' is employed, such that the café visit is deemed over (i.e., a taxi is called to go home) if goals are violated twice in an obvious way (particularly with regard to sexualised behaviour). On return to BP's house, the café visit is discussed in terms of the initial goals, with the focus being on BP practicing the recognition of inappropriate and prosocial behaviour. Ways to improve the activity next time are also discussed. A break-down of the strategies involved in this café visit are given again below, but with particular reference to those aspects of BP's behaviour that may negatively influence the success use of these behavioural management principles.

The use of feedback and operant conditioning paradigms is not restricting to cafes, but is used in all social activities. Other activities that are planned for an morning or afternoon include going to and from therapy sessions (e.g., physiotherapy), lawn bowls (i.e., for improving concentration, social skills, and vigilance, with the goal of lengthening playing time so that he can play a whole Saturday game), and other social settings (e.g., visit to friend's house, movies, museum, squash games, movie theatre). Supported volunteer work has been trialled twice (cleaning in the storeroom of a department store for 6 weeks, and planting trees/ weeding at a bushwalk for several months), but due to motivation and attentional impairments, and the profundity of social inappropriateness these were largely unsuccessful. Nonetheless the experience was a good step toward community integration, and allowed clarification of the types of situations/environments that might make vocational training difficult. The re-

introduction of a work trial will be supported by an occupational therapist as well as myself next time, with stricter boundaries and the possibility of a behaviour contract. Another activity that was trialled was the participation in a computer-training course. This was discontinued after two sessions due to problems with motivation, concentration, and inappropriate behaviour (although the teacher of the course was perhaps not experienced enough with people with frontal lobe disorders, and thus unprepared for BP's behaviour).

At present, due to the lack of success in vocational placements so far, the attention has shifted toward building motivation and routine within the home. Chores are set each week, with activities often contingent on the completion of the chores. These include emptying the dishwasher, cleaning his room, and watering the garden. The cooking of one meal a week for the family is also expected. Music theory exercises and other tasks to do with music therapy are also set to complete each week (e.g., piano or guitar practice, learning the words to songs). There is still a lot of leisure time at home available in the day for BP. This is filled with activities such as playing cards and other games (e.g., Playstation), watching videos, and going down to a nearby park to kick a ball/play cricket/throw a frisbee. While these are considered leisure time, it is important to always keep rehabilitation in mind, such as improving sustained and selective attention and reasoning skills.

Behavioural Strategies

Many of the behavioural strategies mentioned in the section on TBI rehabilitation research are used in the majority of activities BP undergoes. These include:

- Pre-training: BP has difficulty generalising, so this must be specific to each given situation. Role-playing. ‘Putting the shoe on the other foot’ (e.g., “how would you feel/react?”). Breaking down each stage of the situation (incl. coming home at the end- a problem given fatigue). Often focusing on 4 or so very specific things to work on, and testing BP to make sure he can remember them immediately before entering situation, e.g., swearing, sitting at other people’s tables, talking to one person at a time, punching).
- Setting up of consequences (agreed upon by both, both negative and positive. Use of things he enjoys a lot (e.g., cafés) and the Premack principle (e.g., video games)).
- Feedback: Constant feedback is essential, both during and after the task. Focus on positive feedback, but negative also (maybe as things to improve on

next time?) Important for self-awareness/self-monitoring to ask how he thought it went, What went well? What could be improved on? Again, ‘putting shoe on other foot’ (e.g., “How do you think she felt when you did that?”).

- Manipulation of situation: Sitting at table in café far from other patrons. Removal of distracting stimuli (e.g., music). Setting up situation as to provide time for BP to stop his impulses, or to remove himself from situation easily (to have a cigarette, take a break). Not putting him in potentially disastrous situations, unless a rescue plan (e.g., picked up by parents) is available.
- Routine: This is important so BP knows what is expected of him, esp. given executive function impairment. It may help with problems of non-compliance and lack of motivation (e.g., use of timetables, calendars).
- Flexibility: Constantly assessing rate of behaviours, and changing approaches accordingly. Looking for possible ways problem behaviours are being inadvertently reinforced, and changing one’s own behaviour in accordance. Basing rehabilitation first on theory, and trying something else if it isn’t working. Using input from other team members (e.g., psychologist, OT), as well as family, about what they have found works best. Looking for signs of fatigue- tackling situations only if he is prepared. Taking rest breaks when necessary.

An example is the use of applied behaviour analysis (ABA). ABA is based on social learning principles, and involves the accurate assessment of a problem behaviour, the measuring of a baseline level of frequency/severity, the intervention of a behavioural management strategy, the monitoring of changes in frequency of defined behaviours, and the review of whether to discontinue the intervention and try a new one or not. This design has been shown to be quite successful in decreasing such behaviours as aggressiveness and inappropriate sexualised behaviour in those with TBI. The first stage of this involves identifying target behaviours. Due to BP’s impaired self-awareness and insight, he may disagree that a behaviour identified is a problem, or even that it exists. Monitoring the rate of such behaviours becomes very difficult in social settings, due to the need to eschew the distance necessary for such observation in order to rescue BP from unsafe environments. Such intervention is so common, that it is unlikely recording could be completed for long, before BP puts himself in an unsafe situation, or becomes a nuisance to others. Anxiety in the caregiver may make an adequate job of recording difficult. Unfortunately, the times when BP would be most likely to elicit high frequencies of target behaviours, would be

when it is most difficult to stand back and record. In an ideal world, staff numbers would be sufficient so that monitoring of behaviour and general care could both be provided, but budget constraints make this unlikely. Also, BP's compulsion to talk to everyone around him would mean that the monitor would not be left alone to do his/her job. Strictly structured interventions plans such as this are extremely difficult to use for people with severe TBI such as BP, and some degree of flexibility is needed to ensure that the underlying strategies can be employed.

An important aspect of rehabilitation is the ability to learn contingencies, and to change behaviour accordingly. Frontal lobe dysfunction often includes impairment in making such contingencies. The ability to generalise from one particular situation may also be lacking, making learning of such contingencies of little value. Limited insight, common with frontal lobe impairment, may mean that the client has little knowledge that there is anything wrong, and therefore unable to adjust their behaviour accordingly. In fact, often BP feels that he knows best, and that what I might consider to be a problem behaviour is actually just a charming characteristic (this is particularly important with BP, as he constantly tries to make people laugh, but often goes about this in completely inappropriate ways, e.g., pretending to punch people, calling them names, telling nonsensical or 'dirty' jokes).

Another problem that one often encounters in frontal lobe rehabilitation, is the characteristic lack of motivation. Feeling quite content to 'live off ACC' makes it very difficult to engage in rehabilitation and vocational training³. Every day is a constant struggle with just small things (e.g., cooking a meal). This is not due to a lack in ability, as once the task is begun, it is completed swiftly. It is, of course, very important to plan rehabilitation with the TBI patient, in order to discover what activities are particular interesting to them. Without this interest, motivation is extremely difficult. Even with it though, the interest often wanes. Lack of motivation after frontal lobe damage provides a huge obstacle in returning to gainful unemployment, and increases dependency on those around them.

Impairment in insight often makes pre-training and role-playing difficult. BP has limited understanding about the extent of his cognitive and behavioural

³ The Accident Compensation Corporation (ACC) administers New Zealand's accident compensation scheme, providing accident insurance for all New Zealand citizens, residents and temporary visitors to New Zealand. In return people do not have the right to sue for personal injury, other than for exemplary damages. (ACC, 2003).

impairment, so the idea of role-playing going to a café may seem babyish and unnecessary to him. Given that BP is extremely impatient too (due to his impulsivity), in-depth planning of a situation (i.e., role-playing, breaking down the task into its components) is often foregone for a quick run-through before he leaves. While he may feel he is completely up to a task when at home, the impulsive behaviour is so hard to control while out- he truly believes that everything will go well and such pre-training is unnecessary. Insight is especially compromised when fatigued, so it is important to try to reason with BP based on prior trips that have not gone well. This lack in understanding of the extent of his deficits also makes some rehabilitation strategies unsuccessful. He is especially non-compliant when scoring schedules and records are kept regarding outings and behaviour, as these are viewed as childish.

These are just some examples of how implementation of neuropsychological rehabilitation is difficult, particularly for those with frontal lobe dysfunction. The following section relates to some specific aspects of BP's cognitive and behavioural dysfunction, and the ways in which rehabilitation is made difficult for these areas.

Motivation

One of the key difficulties with BP's rehabilitation has been the obstacle of motivation. Motivation may be linked to self-awareness, where lack of insight into one's own impairments may make it difficult to see 'the point' of participating in rehabilitative activities. Executive function impairment is also likely to be closely linked to decreased motivation. For one to be motivated about engaging in an activity, the link must be made between the activity and the rewards (intrinsic or otherwise) likely to be gained from it. This link would be especially difficult to make if the goal is vague or the reward is small. Impulse control disorders may also influence motivation. If the goal is long-term, then nearly always will a shorter-term goal win out. The example of motivation issues in cooking a meal is shown in Table 9.

Also employed in an attempt to increase motivation for change, were Miller and Rollnick's (1991) motivational interviewing techniques, based on treatment for addiction. The key to this approach is to guide the client towards motivation by asking open-ended questions, listening 'reflectively', affirming, and eliciting self-

Table 9. Problems with motivation in rehabilitation activities- the example of cooking a meal.

Introduction
- BP rehabilitation plan includes the cooking of one meal a week for his parents
- Motivation to do so is minimal, and each week it is an almost an impossible task
Roadblocks to motivation
- If he does not cook the meal, his parents will cook anyway (and probably a better meal than he would provide too)
- Limited insight: BP thinks he could cook a meal whenever he wants, so it's a pointless exercise (fails to see routine and motivation to do so are as important as actual cooking skills)
- Attention: Inability to stick at a task for a prolonged period of time makes cooking complex meals difficult. Selective attention difficulties make cooking difficult if distracting environment (e.g., stranger in the house, phone rings, music is playing)
- Executive function: Has difficulty making concrete connections between the practicing of independent living skills and the long-term goals of functional independence (i.e., cooking for himself all the time- not just eating cereal).
- Frontal lobe syndrome: Childishness and selfishness means doing tasks for others has little incentive. Self-rewards are primary goal.
- Impulse control: Difficulty preventing himself from stopping cooking to have a cigarette/ call someone.
Possible solution to these problems
- Set up a reward schedule (e.g., his parents cooking a favourite meal the next night; inviting friends over for dinner on the nights that he cooks)
- Removal of distracting stimuli (e.g., no music; phone off the hook)
- Choosing quick meals that BP likes to eat
- Making sure plenty of good feedback is given for completing the task
- Plan a set time for cooking when it is known everyone will be out
- Have cooking a meal on a set day at a set time, so that the activity becomes a return (as has been the case with other activities such as physiotherapy)
- Compromise if necessary (e.g., I'll chop the onion if you do the rest of the vegetables)
Current situation
While sometimes these techniques work, the final result is that cooking is done very infrequently. Perhaps cooking is really not BP's thing? Still, motivation for other household chores is also limited, and it appears with the best intentions it remains VERY difficult to cook a weekly meal.

motivational statements, in order to invoke motivation change from within the individual. Techniques for guiding clients towards wanting to make a change for themselves are provided. However, so far the employment of these techniques has not been successful. It is likely that the BP's impaired executive function makes it difficult for cognitive-driven rehabilitation to be successful. Nonetheless, some techniques to counteract resistance (e.g., not ordering BP to do things but to give him choices and make him make the decisions on his own) have been useful on occasion. These techniques continue to be employed and perhaps in the long-term Miller and Rollnick's (1991) motivational training will be successful in improving motivation to engage in some tasks.

Social inappropriateness

Given the nature of the role of a ‘social rehabilitation provider’, the problem of social inappropriateness should be the major concern and focus. This social inappropriateness is pervasive and comes in many forms. Typical inappropriate behaviours in a social environment involve the compulsion to talk to strangers, often with a reference to age (e.g., “Guess how old I am”). Discussions about how such questions about age violate social norms have been unsuccessful in diminishing this behaviour. In fact, over the last year, BP has begun to flatter people more and more when guessing their age. Sometimes this flattery is absurdly excessive, causing the person to laugh. Laughter has been found to be extremely reinforcing for BP, making it difficult to stop him from ‘acting the fool’ when in the presence of strangers, and it has reinforced BP’s joke telling. Contingencies have been set in place regarding the inappropriate joking with strangers, and have also involved the teaching of appropriate times and ways to joke (e.g., having a joke-telling session at home or with friends, where jokes are shared).

One particular roadblock to effective social rehabilitation has been the typical responses of the general public to BP’s inappropriate behaviours. While in many ways politeness is a virtue and is welcome with regard to accepting BP into one’s environment, many people are often too taken aback and confused to let BP know if behaviour is inappropriate and is violating social norms. It would be much more helpful for BP to be given feedback about such behaviours. For instance, if BP were to hug a person he has just met, they would usually not say that they felt it was violating their space and made them feel uncomfortable, even if that was the case (although his caregiver certainly would say something to BP in such a situation). While this is a better reaction than to get angry, the best response would be to say, “I don’t feel comfortable hugging you, as we have only just met. I only hug my friends and loved ones. You can expect a hug out of me once we have known each other for some time”. It may seem harsh, but giving BP the message that hugging strangers is not out of the ordinary does not help BP to learn to distinguish appropriate behaviour from inappropriate. The example of tackling social inappropriateness in a café is given in Table 10 below, including how BP’s impairments may make rehabilitation in this environment difficult.

Table 10. Examples of a social skills training exercise, and the difficulties inherent in the task- the example of a café visit.

<p><i>Example: Social skills training in a café situation</i></p> <ul style="list-style-type: none"> - Setting up of plan beforehand on a whiteboard (i.e., breaking down the activity into components, e.g., getting to café, entering the café, buying the coffee, behaviour while there, leaving café, coming straight home). - Role-playing of task at hand - Setting up of boundaries/ rules/ behaviours to work on (this is done together with BP, where those problem behaviours in the specific situation are discussed and what particular behaviours need to be focused on. E.g., 1) Trying not to speak to strangers, 2) No pretend aggression, 3) No sexual joking, 4) Sticking to the plan, 5) No swearing) - Setting up of verbal cues that can be used to remind BP of the social goals (e.g., “chill”, “no. 1”), and to aid BP in picking up on social cues from others (e.g., if they are annoyed or busy) - Setting up of consequences beforehand (e.g., if this goes well we will go to visit a friend tomorrow) - Making BP repeat the goals as we enter the café (i.e., to make them fresh in his mind; to see if his working memory can hold all five goals in his head) - Giving specific feedback (both negative and positive) throughout the activity, particularly regarding the stated goals earlier - Using cue words and aiding BP with the recognition of social cues from others during the activity - Modelling of appropriate behaviour during the activity (and at all times) - Use of a “two-strike” rule (call for a taxi to go home after two presentations of problem behaviours) - Specific feedback about how the activity has gone at the point of leaving the café - A review of how the goals have been met once home (i.e., Asking BP “How did you think that went? Which of the goals do you think were best met? Which weren’t? Why do you think that was? What do you think we could change to make sure it goes better next time? Then going on to point out those behaviours that were inappropriate and how we could do better next time. <p>Roadblocks to the social rehabilitation plan</p> <ul style="list-style-type: none"> - Impulse control: very difficult to go through the plan, role-play, etc, without BP becoming impatient and walking out the door. A compromise of a quick run-through is the norm instead. In the café situation, even with the best intentions beforehand, inappropriate behaviour often comes out. The difficulty of training for impulse control with little able to be done ‘in the moment’. Are consequences and pre-planning attacking the real problem? - Fatigue: Towards the end of an activity fatigue can set in, causing the frequency of problem behaviours to increase. - Attention: Easily distractible, so big crowds, loud music or small children may make it difficult to focus on the goals. - Self-awareness: difficulty in recognising what behaviours are inappropriate and why - Executive dysfunction: difficulty with integrating the sub-goals into one whole. Impairment in ability to generalise from one specific situation to another (e.g., if there are differences in the café trip such as knowing someone there) - Motivation: More reinforcing for BP to engage in inappropriate conversations with strangers than having a quiet cup of coffee, despite set reinforcement contingencies <p>Possible solutions to these problems</p> <ul style="list-style-type: none"> - Planning such trips for times when BP has shown he can control inappropriate

behaviour the best

- Managing fatigue (i.e., enforcing rest breaks or quiet time before tackling social situations; being aware of behaviour that is indicative of fatigue when preparing the activity)
- Choosing situations where crowds or loud music are unlikely
- Constant feedback regarding inappropriateness of some behaviours, and why they are inappropriate (e.g., role-reversal- "how would you feel?")
- Combining social visits with friends with café trips (hence providing more motivation for improved behaviour. Unfortunately the result is usually the opposite, depending on the friend)
- Encouraging appropriate conversations with known staff and known customers

Current situation

Difficulty with such social situations but improving slowly with the practicing of appropriate behaviour.

Impulsivity

While consequences are important determinants of behaviour, and therefore warrant attention and manipulation, the question remains as to how salient consequences are in each moment of a given situation, for someone with impulse control impairment. Often BP finds himself displaying behaviours (e.g., saying inappropriate things) without being able to stop himself. They appear in the moment. The consequence of this is often remorse and guilt for the situation, and a feeling of helplessness. While behaviour modification has been shown to be helpful in treating impulse control disorders, BP's best efforts may still not be enough to prevent some impulsive behaviours.

The importance of structuring the environment (i.e., dealing with antecedents) becomes particularly important for those with impulse control disorders. An initial assessment of which antecedents are triggering impulsive behaviours is necessary. Counting impulsive behaviours in specific contexts where they are to be reduced can be extremely difficult. If BP makes an aggressive or sexual remark to a stranger (or it appears he is about to), it is necessary to intervene. This is in order to protect both BP and the stranger. This intervention is hindering an objective assessment of the frequency of the specified problem behaviour. Nevertheless, it is necessary for BP's safety under my role as his caregiver. Instead, situations that elicit problem behaviours are generally inferred from experience with BP in these situations. These situations can then be avoided. The example of environmental and situational manipulation in a lawn bowls excursion is provided in Table 11 below.

Table 11. Managing the environment in a rehabilitation exercise- the example of a lawn bowls excursion.

Managing fatigue:

- Monitor for signs of fatigue (e.g., increased inappropriate behaviour, putting on strange voices, mania (or being 'OTT'), slurred speech, eye looking glassy, particularly impulsive),
- Cancel the trip if fatigued (or postponing it until after a rest)
- Planning the trip for immediate following a rest (or a restful activity)
- Arranging to arrive during the middle of the other bowling club members lunch break, so that BP may practice socialising with them, but not for an extended period of time so that fatigue sets in
- Checking schedules to ensure that no event is on (in order to avoid crowds)
- Setting a minimum number of ends to be played, and extending this over time (vigilance/sustained attention training)

Managing social behaviour

- At arrival, choosing to sit with people that he knows (so that he may practice appropriate social behaviour)
- Avoiding interacting with those who do not respond well to BP's behaviour
- Avoiding those who tend to make BP 'show off' in inappropriate ways
- Choosing a bowling green that is not immediately beside other members, but is still near enough to players that he may practice controlling impulsive behaviour (i.e., trying not to interrupt their game)
- Playing on the practice bowling green if championship matches are being played
- Using verbal cues to remind BP of goals and to aid him in suppressing inappropriate social behaviour.

Overall, it is clear that BP has many people, professional or otherwise, involved in his rehabilitation. Each brings their own skills and experience to the particular problems that BP presents with. However, the change in BP's inappropriate behaviour and executive function over the past 2½ years has been slight. Considering the best behavioural theories and strategies that the rehabilitation literature can provide have been used, the question must be asked- What are we doing wrong? While many situations may have been better-approached in hindsight, the general consensus is that even with the best tools and practices, rehabilitation is an extremely lengthy and difficult process, particularly for those with severe frontal impairment. Nonetheless, there have been improvements in some areas of BP's social functioning (e.g., BP used to wander often, this is now an infrequent occurrence). These improvements help to inspire those involved in BP's rehabilitation to continue to base their rehabilitation on theory, be flexible to change if a strategy is not successful, and to persist.

Conclusion

Research in the efficacy of brain injury rehabilitation is limited. The focus of neuropsychology has shifted in recent times, away from diagnosis and assessment, and towards brain injury rehabilitation. The research literature on TBI rehabilitation is likely to expand at a very rapid rate, thus providing support for the use of strategies and interventions, both old and new. In the meantime, TBI rehabilitation must be based in theory (e.g., social learning principles). But rehabilitation must also very, above all else, specific to the situation, environment and specific strengths and weakness of each individual. Rehabilitation must remain flexible, so that interventions can be altered as they are found to be unsuccessful, or if conditions change. Rehabilitation models must incorporate this trial-and-error approach, and be focused on the ability for person with TBI to function on an everyday level. People don't live in a clinic- the general nature of our complex world means that rehabilitation providers must be extremely flexible and adaptable to whatever obstacles come their way.

People with severe TBI have a multitude of issues that complicate their rehabilitation. It appears that even with the best intentions and a multi-disciplinary approach, rehabilitation can be a very lengthy and difficult process. The best rehabilitation professionals can do is keep people safe, base rehabilitation on theory and the literature, and adapt it to the individual and changing circumstances. This way it is hoped that everyday functioning ability and self-determination can be maximised. The individuals strengths must be emphasised and encouraged, the environment must be manipulated in a way to increase everyday functioning, and respect for the individual must be kept in the forefront of the rehabilitation professionals' minds.

Chapter 4: Obsessive-compulsive symptoms following TBI

Besides the many frontal lobe-related behaviours following BP's injury that have been described previously (such as impulsivity, social inappropriateness, and lack of insight), other behaviours emerged that resembled obsessive-compulsive disorder (OCD). These behaviours did not come to the attention of those clinicians involved in BP's rehabilitation, probably because BP did not report them as problems, and because they would only see him for short periods of time. Most importantly however is that fact that more pertinent problems have needed this attention (for his general functioning, independence and safety). The OCD-like behaviour involved BP's affinity with the number three and its multiples, the influence of which could be seen throughout many of his daily activities. There appeared to be no compulsive behaviours associated with the number three or otherwise prior to the accident.

Examples of the influence of the number three and its multiples on BP's behaviour are numerous. For example, BP has a tendency to click his fingers, and will almost invariably do this three times in any one go. Other such stereotyped motor behaviours as making a clicking noise with his mouth, clapping, or punching out (which are done in a joking, rather than an aggressive, manner, usually on another person's arm, although often the third punch is unreasonably hard) will almost always follow the three-times rule. He sometimes asks to be patted on the back (e.g., if he does something praise-worthy) three or six times. If two or five pats are given (although in most cases none are given), he will ask for another and will persist until he gets it. BP has considerable insight into this affinity with the number three, and will explain that the reason why he needs another pat is to make up a multiple of three. Neither BP nor his family can explain why the number three is so important, or where it came from.

A clear example of BP's affinity with the number three can be seen when he plays video games on his Playstation (which he does often). When selecting levels and other options on the main menu before beginning a game, BP will move up and down the menu, so that when he finally selects starting a game, he will have pressed the buttons a multiple of three times. Often the number of button presses totals 18 (which takes considerable time). During the game (almost always Tony Hawk Pro Skater, a game he plays a *lot*), he may remark if the numbers in a score add up to a multiple of three (e.g., 26,304 points:

$2+6+3+0+4=15$), and say that the score was “a lucky number”, or more commonly “*his* lucky number”. More often he may just look pleased and say something like “Yes- 15”.

Other examples of the influence of the number three include the number of card games we may play (usually first to six wins) or ends of lawn bowls (he prefers 6 ends). Variation from this number is sometimes met with resistance, although if fatigued or distracted he may cut this number short. Setting a microwave for times that feature the number three, such as heating a pie for 3 minutes and 33 seconds, has also been observed.

The major difference between BP’s counting compulsions and those that would be exhibited by someone suffering from obsessive compulsive disorder appears to be the distinct lack of anxiety accompanying these compulsions. When asked about how he feels when doing one of the aforementioned activities by a number other than three, the most extreme description he has agreed to feeling is “uncomfortable”, and that he “prefers” the number three and its multiples. On this basis alone BP would not qualify for a clinical diagnosis of OCD. Further, his compulsions are not designed to ward off obsessions or anxiety at all, but are ritualistic behaviours that are present in and of themselves. Given that the counting behaviours did not cause BP distress or anxiety, it is not surprising that he or others have failed to mention them to clinicians involved in his rehabilitation.

It is possibly more helpful, in the light of BP’s executive dysfunction, to see these behaviours more in terms of his impulsivity and perseveration (i.e., a cognitive rigidity that arises out of difficulties in switching cognitive set for different situations). Perhaps it is a way for BP to provide some structure to his life when structure and organisation is lacking (due to executive function impairment). The distinction between the perseveration and ritualistic behaviours shown by many people that have suffered a traumatic brain injury (especially of the frontal lobes) and the compulsive behaviours of OCD is often not clear, and their similarities suggest they may have similar neural substrates. Proposed psychobiological bases and the murky distinction between perseveration and compulsions will be discussed later.

While these behaviours appear to have largely subsided of late, it is interesting to speculate on the link between TBI and OCD. Why and how often do OCD-like symptoms appear following TBI, and why did they subside for BP over time? Possible answers to these questions will be considered later in this chapter. In order to clarify the relationships, the nature of obsessive-compulsive disorder as a clinical diagnosis will be discussed here, followed by the theories of aetiology paying particular attention to biological theories and the incidence of OCD following TBI. It is important to keep in mind however that while some of BP's behaviours may be reminiscent of some of the compulsions of OCD sufferers (hence the investigation of the neuropsychological basis of OCD), there are many fundamental differences between BP and those with classic, idiopathic OCD.

Obsessive-compulsive disorder (OCD)

Obsessive-compulsive disorder (OCD), once thought to be a rare condition, is now considered one of the more prevalent psychiatric disorders (Stein, 2002). Recent studies have estimated a lifetime prevalence rate for OCD of 2.5% and 1-year prevalence rate of between 1.5 and 2.1% (American Psychiatric Association [APA], 1994). It is considered amongst the group of anxiety disorders, and is characterised by the presence of obsessions (intrusive and recurring thoughts, impulses, and images, that are uncontrollable, irrational and elicit anxiety), and compulsions (irrational acts performed repeatedly in order to reduce anxiety) (Davison & Neale, 1996). Obsessions may take the form of doubts, endless chains of thoughts, impulses, fears, or persistent images. Common compulsive behaviours include excessive checking, cleaning, ordering, and counting. The DSM-IV (APA, 1994) diagnostic criteria for OCD are presented in Table 12.

Table 12. DSM-IV Diagnostic Criteria for Obsessive-Compulsive Disorder (adapted from American Psychiatric Association, 1994, pp. 422-423).

• Diagnostic criteria for 300.3 Obsessive-Compulsive Disorder
A. Either obsessions or compulsions: <i>Obsessions as defined by (1), (2), (3), and (4):</i>
(1) Recurrent and persistent thoughts, impulses or images that are experienced, at some time during the disturbance, as intrusive and inappropriate and that cause marked anxiety or distress
(2) The thoughts, impulses, or images are not simply excessive worries about real-life problems
(3) The person attempts to ignore or suppress such thoughts, impulses, or images, or to neutralise them with some other thought or action
(4) the person recognises that the obsessional thoughts, impulses, or images are a product of his or her own mind (not imposed from without as in thought insertion)
<i>Compulsions as defined by (1) and (2):</i>
(1) repetitive behaviours (e.g., hand washing, ordering, checking) or mental acts (e.g., praying, counting, repeating words silently) that the person feels driven to perform in response to an obsession, or according to rules that must be applied rigidly
(2) the behaviours or mental acts are aimed at preventing or reducing distress or preventing some dreaded event or situation; however, these behaviours or mental acts either are not connected in a realistic way with what they are designed to neutralise or prevent or are clearly excessive
B. At some point during the course of the disorder, the person has recognised that the obsessions or compulsions are excessive or unreasonable. Note: This does not apply to children.
C. The obsessions or compulsions cause marked distress, are time consuming (take more than 1 hour a day), or significantly interfere with the person's normal routine, occupational (or academic) functioning, or usual social activities or relationships.
D. If another Axis 1 disorder is present, the content of the obsessions or compulsions is not restricted to it (e.g., preoccupation with food in the presence of an Eating Disorder; hair pulling in the presence of Trichotillomania; concern with appearance in the presence of Body Dysmorphic Disorder; preoccupation with drugs in the presence of a Substance Use Disorder; preoccupation with having a serious illness in the presence of Hypochondriasis; preoccupation with sexual urges in the presence of a Paraphilia; or guilty ruminations in the presence of Major Depressive Disorder).
E. The disturbance is not due to the direct physiological effects of a substance (e.g., a drug of abuse, a medication) or a general medical condition.
<i>Specify if:</i> With Poor Insight: if, for most of the time during the current episode, the person does not recognise that the obsessions and compulsions are excessive or unreasonable

It can be seen from this that both obsessions and compulsions must be present for a clinical diagnosis of OCD, that obsessions must be intrusive, uncontrollable and anxiety-producing, and that compulsive behaviours must be rigidly defined, aimed at

reducing anxiety or distress, but irrational (in that they are excessive or not connected to the anxiety-producing obsessions in a realistic way).

Theories of aetiology of OCD

Theories of aetiology of obsessive-compulsive disorder can be grouped according to psychoanalytic, behavioural, cognitive and biological theories (Davison & Neale, 1996). Psychoanalytic theories regard obsessions and compulsions as resulting from instinctual sexual and aggressive forces that are not under control because of a fixation at the anal stage (Davison & Neale). Behavioural theories implicate learned behaviours that are reinforced by their consequences (e.g., counting may have helped to focus the mind away from anxiety-producing thoughts in the past, so the repetition of such behaviours is reinforced) (Davison & Neale). However, if the cause of OCD could be attributed purely to this, one would expect compulsive behaviours to be tied to the anxiety-eliciting obsessions in a more realistic way. Carr (1974), based on a cognitive model of OCD, proposed that those with OCD overestimate the likelihood of harmful outcomes, and their compulsions are a result of this. For example, a compulsive checker may continue to check if a door is locked properly, simply because his or her judgement of the likelihood that the door has been left unlocked and that the house will be burgled is impaired.

Biological theories for obsessive compulsive disorder have been widely reported, and are based on many precepts. Family studies have demonstrated a genetic component of obsessive-compulsive disorder. High rates of anxiety disorders were found in the close relatives of a sample of OCD sufferers (McKeon & Murray, 1987). Lenane et al. (1990, cited in Davison & Neale, 1996) found OCD in 30% of the first-degree relatives of their OCD sample. Monozygotic twins have a higher concordance rate for

OCD than do dizygotic twins (APA, 1994). These findings suggest that there is a real biological basis to the presence of obsessive-compulsive disorder. Theories have tended to place emphasis on frontal-striatal neural circuits; supported to a large extent by both studies of lesion location in TBI patients who develop obsessive-compulsive symptoms, and research on the brain structure and function and neuropsychological impairments characteristic of idiopathic OCD. A detailed review of both of these strands of research follows.

OCD following TBI

Psychiatric disorders are quite common following traumatic brain injury. Two common types are depression and anxiety. Newburn (1998) reported rates of 10-77% for depression following moderate to severe TBI, while a compilation of two studies involving 1,199 patients revealed approximately 29% were diagnosed with clinical anxiety (Epstein & Ursano, 1994). Much of the high prevalence of these disorders can be attributed to the psychosocial changes that often follow TBI (e.g., social isolation increased dependency on family, realisation of restrictions on the future), and to the stress that must naturally accompany such a traumatic and life-changing experience. Some may also be attributed to pre-morbid levels of psychiatric disturbance.

Amongst the group of anxiety disorders is obsessive-compulsive disorder (OCD). Epstein and Ursano (1994) reported that 3% of their sample displayed OCD, which, while not being particularly high, is slightly above that of the general population. Silver, Kramer, Greenwald and Weissman (2001) reported a lifetime prevalence rate for OCD following severe TBI at 4.7% (compared to 2.3% in a large non-head injured control group). It is highly likely however, that OCD-related symptoms feature in many more patients, but that the majority do not have all the symptoms required for a

clinical diagnosis (particularly that the obsessions or compulsions do not cause sufficient anxiety or distress, or that they are excluded due to their ‘general medical condition’).

Case studies of people who display obsessive-compulsive behaviours following a brain lesion of known location may be very beneficial to our understanding of obsessive-compulsive disorder as it appears in its idiopathic form (the majority of cases). Such studies are limited, but the major details and findings of some of these can be found in Table 13. While all suggest possible linkages between brain lesions (especially in the frontal lobes) and development of OCD symptoms, the exact nature and area of injury and the way the symptoms manifest vary widely. Of the 33 cases mentioned, all but 14 (studies [1], [2], case 2 in [6], cases 1 and 2 in [7], study [8], cases 3, 4, and 5 in [10], cases 1-4 in [11], and [12]) specifically implicate areas of the frontal cortex. Of these 14 cases, most were too mild to find any focal lesions, and some even suggested frontal damage based on neuropsychological testing. The other major area highlighted in these studies is the basal ganglia, particularly in [8], where small lacunar infarcts situated in the left basal ganglia were associated with the development of “needing to know” obsessions. No other lesions were discovered. Also of interest is the fact that the development of “need-to-know” form of obsessions tended to be associated with vascular lesions. While it is difficult to make conclusions about a link between this type of brain damage and categories of obsessions given the limited number of studies mentioned here, this is perhaps worthy of further research.

Neuropsychological and neurological findings amongst OCD patients

Various studies have considered the link between OCD and brain structures by looking at idiopathic OCD populations, rather than TBI populations. We may gain much

knowledge about the neural substrates involved in the development and manifestation of OCD by looking at populations of OCD patients. A distinction in the research can be made between those studies that focus on neuropsychological differences displayed by people with OCD, and studies that have investigated differences in neurological structure or function.

Details of the studies that have assessed neuropsychological function in people with idiopathic OCD can be found in Table 14. Research has tended to focus on impairments in frontal lobe functioning, using such assessment tools as the Wisconsin Card Sorting Task (WCST) and the Tower of London Task. Findings from those studies that have been more general in their assessment of cognitive functioning have suggested that this focus on frontal lobe tests is not without merit. The overwhelmingly conclusion that can be made from reviewing the literature on neuropsychological impairments in OCD patients is that frontal lobe functioning tends to be impaired. These frontal lobe impairments can be found in the form of difficulties in set-shifting, impaired attention, problems with planning (e.g., Complex Figure Test), perseveration, and in generating alternative solutions to problems. Other impaired areas of cognitive function that have been linked to OCD patients include visuo-spatial processing and immediate visual recall, which has led some researchers to suggest that the right hemisphere may be implicated in OCD.

Some of the researchers in Table 14 have been more specific in implicating frontal pathways and frontal-striatal circuitry based on neuropsychological findings in OCD samples. The investigation of performance on the WCST and the Object Alternation Test for OCD and schizophrenic patients in [1] suggested that while both psychiatric illnesses appear linked to frontal pathways, OCD can be associated more with

impairment in the orbital-frontal cortex, and schizophrenia with the dorso-lateral prefrontal cortex. In [4], the cognitive rigidity when needing to put aside a mistake and set a new sub-goal that was seen in OCD patients is discussed in terms of how this implicates the frontal-striatal pathways. Tallis (1997), in a review of the neuropsychology of obsessive compulsive disorder, suggested that investigations in this area can be divided into four broad areas: general intellectual functioning, an ‘under-inclusive’ thinking style, performance on frontal lobe tests and memory impairment. The research in these four areas can all be linked back to theories of impairment in frontal-striatal circuits, including the basal ganglia. Tallis specifically refers the *defect in knowing* or epistemological sense that may occur as a result of damage to frontal-striatal pathways, and how this doubting may manifest itself as obsessive checking or other such OCD behaviours. In any case, it is clear that such theories linking OCD with the frontal lobes and its striatal connections are supported by the research on neuropsychological assessment of OCD patients.

OCD patients have also been the focus of studies on their neurological differences from controls. These have included volume of neural structures, single photon emission tomography (SPECT) studies, and auditory event-related potentials (AERPs). A list of these studies can be found in Table 15. Overall, the research on the neurology of OCD patients has revealed enlarged ventricles, reduced caudate nucleus size, increased amplitude on auditory event-related potentials for irrelevant stimuli, and focal perfusion value abnormalities (both increases and decreases) in frontal and anterior temporal lobes on SPECT studies. Again, the research supports theories of altered fronto-striatal circuits in OCD, as well as more generalised damage (indicated by ventricle size). In a review by Stein (2002), it was argued that functional imaging has consistently shown that OCD is characterised by an increase in activity in

orbitofrontal cortex, cingulate, and striatum at rest. This increased activity is especially pronounced during exposure to feared stimuli. Some studies have indicated that serotonin may be the neurotransmitter most closely linked to the manifestation of obsessive-compulsive symptoms, with these over-active areas decreasing to normal levels of activity following treatment with selective serotonin reuptake inhibitors (SSRIs) (Tekin & Cummings, 2002). Pharmacological treatment of OCD, particularly with SSRIs (e.g., fluoxetine, citalopram), will be discussed in the following section, with a focus on how BP's medication may have influenced the presence of OCD-like symptoms.

It is clear that case studies of OCD secondary to TBI, neuropsychological assessment of those with idiopathic OCD, and neuroimaging of OCD patients all support the popular hypothesis that frontal-striatal pathways are in some way implicated in OCD. However, it is unknown whether abnormalities in this area are a principle cause of the development of the disorder, or if this may be an effect of OCD (although the case studies of OCD following TBI would suggest that it is at least partly a causal relationship). The exact nature of the relationship, specifics regarding the different neural substrates of the cognitive (i.e., obsessions) and behavioural (i.e., compulsions) aspects of OCD, and the causes for the varying subtypes of OCD (e.g., checking, counting, contamination) are still largely unknown. More research, with improved neuroimaging tools, may help to elucidate such brain-disorder relationships in the future.

Table 13. Case studies involving obsessive-compulsive symptoms following brain injury

Study	Case studies	Injury type	Injury severity	Pre-injury factors	Location of lesion(s)	Reported outcome
[1] Khanna, Narayanan, Sharma & Mukundan, 1985	1 Age: 18 Male	TBI, bus accident	Moderate (PTA= 4 days)	No family history of OCD or obsessional personality, and no past history of psychosomatic complaints, peptic ulcer, constipation or dyspepsia.	Left occipital depressed fracture, with bone fragments displaced intracranially. Later development of meningitis. Bulging of the cerebellum continued, and part of this was excised. Neuropsychological assessment suggested some frontal lobe dysfunction.	3 years post-trauma, he developed repeated behaviours, such as opening and closing books and picking up and placing down a pen. Developed excessive doubting and checking for things being done properly, and obsessed about various things entering his head physically. Impaired abstraction was also noted. Over two and half years of medical treatment he continued to show sustained improvement.
[2] Drummond & Gravestock, 1988	1 Age: 26 Male	TBI, blow to the back of the head resulting from a violent incident at work.	Mild (no reported PTA).	No previous psychiatric history.	CT scan normal. No neurological signs or symptoms. Unable to presence and/or whereabouts of damage.	Immediately following the accident, he reported mild headache and insomnia, irritability, difficulty in turning head and personality change (from happy and confident to miserable, apathetic and lacking in self-confidence). These symptoms were medically treated and he returned to work. After a few weeks, he became obsessed by thoughts of doing harm to himself and others if he didn't perform certain actions the perfect way. These included counting rituals while shaving, washing and combing his hair, and contamination obsessions that would result in stereotyped breathing compulsions. All written material and notices were memorised. Excessive repetition of compulsions was observed. Hence he was unable to work, highly anxious, and moderately depressed. After treatment, OCD symptoms subsided, but complaints of feeling miserable and suicidal remained.
[3] Seibyl, Krystal, Goodman & Price, 1989	1 Age: 54 Female	Dural-based lesion, consistent with Meningioma	N/A	No reported history of OCD, motor or verbal tics, panic attacks, phobic avoidance, depression, sleep or appetite disturbance, anhedonia, or impaired concentration. No family history of OCD, Tourette's	2.5-cm, enhancing, well-circumscribed, dural-based lesion in the anterior right frontal lobe.	Developed a compulsion to repeat words and phrases of conversation, and obsessions regarding the need to verify the validity and accuracy of spoken statements. She kept a notebook, which she would use to write down parts of people's conversations, and read back to them for verification. She was hospitalised 4 times over 3 years for these obsessions and compulsions. A CT scan during the 3 rd stay confirmed the presence of brain lesion. Many different medications were trialled over the 3 years, to little effect. After the fourth stay, a

[4] Max , Smith, Lindgren, Robin, Matteis, Stierwalt & Morrisey, 1995	1 Age: 12 Female	TBI, motor vehicle accident (MVA)	Severe (GCS=3; PTA= 15 days)	syndrome, major depression, panic disorder, substance abuse, psychiatric hospitalisation or suicide.	PTSD for 6 months at age 5 following sexual abuse; no history of obsessions or compulsions	Widespread damage, (bilateral areas 9, 10, 11, 12, 45, 46, 38, 22, 21, 20, 36, and 32; areas 37, 6, 24 and 47 in left hemisphere). Frontal and temporal lobe damage, no apparent striatal damage. Wedge-shaped infarct in right frontal region.	trial of a MAOI was begun, and compulsive questioning was shown to reduce over 3 weeks. This was later augmented with lithium, resulting in further symptom reduction. At 12-month follow-up, on the same medications, these improvements have been maintained, and no further hospitalisation has been required. Development of compulsive hand-washing; counting and ordering rituals. Compulsions disappeared approx. 8 months post-injury (prob. due to introduction of fluoxetine). Disinhibition, markedly impaired social judgement and aggressive outbursts also developed; at 1 yr disinhibition continued but aggression diminished. Development of OCD symptoms, extreme anxiety about "needing to know" things (e.g., an actor's name); extreme lengths to ease anxiety (e.g., ringing the network to ask, ringing friends in middle of the night). Depression (likely as a result of distress created by these symptoms). Recurrent paresthesias of left arm and face. Both developed compulsive cleaning rituals and hand washing behaviours post-injury.
[5] Swoboda & Jenike, 1995	1 Age: 70 Male	Occlusion of right internal carotid artery	N/A	High achieving worked more than 80 or 90 hours per week as CEO, forced into semi-retirement at age 57 following a retinal artery haemorrhage.	None specified.	Case 1: Primarily frontal lobe injury Case 2: Ruptured aneurysm in right medial area.	
[6] Todd, Levy, Kevorkian & Gouvier, 1996	2	Case 1: CHI, MVA Case 2: Ruptured aneurysm	Case 1: Severe Case 2: N/A	Case 1: None specified. Case 2:			

[7] Kant, Smith-Seemiller & Duffy, 1996	4	Cases 1 & 2: Ages: 43(M), 26(M), 16(M) & 45(F).	CHI, industrial accident. Case 3: CHI, assault. Case 4: CHI, MVA.	Case 1 and 3: Mild (LOC for a few minutes) Case 2: Mild (LOC for 20 minutes) Case 4: Mild to moderate	No psychiatric history.	Cases 1 and 2: Nothing revealed by brain imaging, but neuropsych assessment suggested possible frontal lobe dysfunction. Case 3: Small right frontal depressed fracture, but difficulty finding actual lesions (assessment complicated by depression and inability to complete entire evaluation). Case 4: Possible frontal lobe damage (based on neuropsych assessment), but no clear lesions found by brain imaging.	Case 1: Development of severe aggressive and sexual obsessions. Major depression, generalised anxiety disorder, possible delusional syndrome. Symptom reduction following clomipramine & sertraline. Case 2: Development of checking compulsions, resisting these produced extreme anxiety. Cog. difficulties (concentration, information-processing speed, visual memory). Depressed, irritable, argumentative. Case 3: Development of compulsions (checking, counting) 1 month post-injury. Severe depression. Case 4: Development of OCD symptoms (checking, counting ritualised finger tapping) within 2 months post-injury. Major depression; suicidality; paranoia; cognitive deficits (visuospatial, word fluency, information-processing speed, divided attention, learning and memory), prob. exacerbated by extreme psychological distress at the time.
[8] Geidd, Rapoport, Leonard, Richter & Swedo, 1996	1	Streptococcal throat infection	Age: 12 Male	N/A	2 yr history of OCD, made stable by drug regimen, exacerbated by streptococcal infection. Family history (grandfather w/ Sydenham's chorea, OCD in aunt & mother, Tourette's syndrome in brother). Plasmapheresis decreased caudate volume by 24%, putamen by 12% and globus pallidus by 28% from size 1 day prior treatment to 1 day post-treatment.	Increased size of basal ganglia, diminished by plasmapheresis treatment for the infection (and associated with corresponding decreases in OCD symptoms). Plasmapheresis decreased caudate volume by 24%, putamen by 12% and globus pallidus by 28% from size 1 day prior treatment to 1 day post-treatment.	Dramatic increase in previous OCD behaviours (checking and repeating behaviours, need for symmetry and order, excessive exercising). After discovery of streptococcal infection, plasmapheresis treatment began, and symptoms ameliorated. Relapses at 5 weeks and 6 months- pharmacological and behavioural treatment eased symptoms. Relapse at 10 months was associated with a reemergence of the infection, and plasmapheresis once again diminished symptoms.
[9] Childers, Holland, Ryan & Rupright, 1998	4	Case 1 and 4: Ages: 36(M), 37(M), 38(M), & 41(M).	All cases TBI, assault. Case 2: TBI, hit by train. Case 3: TBI, fall from moving vehicle.	severe. Case 2: Case 3: History of chronic alcohol addiction.	Cases 1-3: No prior injury or psychiatric illness mentioned. Case 4: Two TBIs prior to fall- one of these was severe. History of chronic alcohol addiction.	Case 1: Large left frontal intracerebral haematoma, collapsed left frontal horn, and small right intracerebral haemorrhage. Case 2: Open left frontal skull fracture, haematoma of the base of the interior horn and left frontal region. Case 3: Basilar skull fracture, frontal contusion and intracerebral haemorrhage. Case 4: Right frontal temporal	Case 1: Developed obsessions about work following PTA. Convinced someone was waiting to pick him up, causing anxiety, and refused to cooperate in therapy. Obsessions subsided once treated with Prozac. Case 2: Developed ritualistic behaviour and obsessions about object arrangement on his desk. Symptoms subsided after treatment of clomipramine. Case 3: During recovery from PTA he began to obsess about a former girlfriend, compulsively touching, handling and arranging a photograph and letters from her. Obsessions stabilised over a few days after taking

[10] Hugo, van Heerden, Zungu-Dirwayi & Stein, 1999	6	Case 1: acute haemorrhage of right anterior cerebral artery.	Cases 1 and 4: N/A	Case 1 and 2: Good health pre-injury.	contusion, right subdural haematoma, left skull fracture.	paroxetine.
	41 (M), 59 (M), 32 (M), 46 (F), 41 (M), & 29 (F).	Case 2: TBI, fall.	Case 2: no PTA or GCS specified, SPECT	Case 3: History of generalised seizures since childhood.	Case 1: Right frontal haematoma, extending into the ventricular system.	Case 4: Developed persistent cleaning rituals, and repeatedly attempted to aid cleaning staff in his facility. He obsessed about the details of many activities, such as arrival time of therapists (one minute late would be enough to elicit significant anxiety). OCD symptoms improved following clomipramine treatment.
		Case 3: carbon monoxide poisoning	Case 3: scan indicated a severe	Cases 4, 5 and 6: None specified	Case 2: Bilateral anterior temporal and anterior frontal atrophy.	Case 1: 3 months after haemorrhage he experienced persistent and intrusive thoughts regarding the need to check that past events had occurred, and to check his diary for verification of future dates. He began checking that doors were locked and counting his steps. He declined medication treatment.
		Case 4: aneurysm on left internal carotid artery.	Case 4: injury.	Case 4: Left internal carotid artery, although CT scan normal.	Case 3: Generalised atrophy, and a lacunar infarct in the right internal capsule.	Case 2: He had developed obsessional doubts and compulsive checking from the time of the TBI. He also demonstrated emotional lability and memory impairment post-injury, and had depressive symptoms. 1 year post-TBI he suffered a seizure. Fluoxetine treatment had resulted in moderate improvement after 4 months.
		Case 5: TBI, penetrating gunshot wound.	Case 5: LOC lasted for 2-3 days, indicating	Case 5: Right anterior-inferior temporal lobe spongiosis consistent with bullet wound.	Case 6: CT scan normal, although SPECT scans showed decreased perfusion in focal areas through the cortex, particularly bilaterally in frontal, anterior temporal, and occipital cortices.	Case 3: Developed intrusive aggressive thoughts (e.g., hurting his sister) and repetitive mental rituals (e.g., repeating nonsense words) immediately following the trauma (a suicide attempt). He continued to show depression following his attempted suicide, but also experienced new levels of emotional lability and memory impairment. Fluoxetine treatment resulted in much improvement after 4 months.
		Case 6: TBI, MVA.	Case 6: a severe injury.	Case 6: Severe (PTA = 1 month)	Case 4: Obsessions about numbers and counting compulsions developed following surgery from an aneurysm, as well as obsessions about having another aneurysm. On psychiatric interview 3 years later there was also mild cognitive impairment. Medical treatment had so far been unsuccessful, but a new treatment (citalopram) was about to be started.	Case 5: began having intrusive thoughts about possible harm to his children from time of injury. He also demonstrated personality changes with increased

[11] Berthier, Kulisevsky, Gironell & López, 2001	10. Mean Age: 30, sd= 9.3. 4 Male, 6 Female.	All TBI through MVA. CSF fistulas with intracranial air or otorrhea.	6 mild (GCS= 13-15), 2 moderate (GCS= 9-12), 2 severe (GCS= 3-8).	1 patient's aunt diagnosed with OCD following a cavernous angioma of the left temporal lobe; no other family history of OCD, tics or Tourette syndrome reported. 4 patients had a positive personal history of psychiatric symptoms pre-TBI: 2 had lifelong indecisiveness and perfectionism but not OC symptoms, and 2 had suffered from PTSD during adolescence as a result of sexual abuse. No reported cases of substance abuse.	Of the 6 mild cases (cases 1-6): MRIs normal, but SPECT studies in two cases showed decreased perfusion rates in the left frontotemporal region (case 5) or in the inferior parietal cortices bilaterally (case 4). Of the 2 moderate cases: One showed atrophic changes at the head of the left caudate nucleus, and laminar bifrontal hygromas (case 7). The other had multiple contusions in the left orbitalfrontal and mesial temporal cortices, as well as the white matter anterolateral to the head of the caudate nucleus (case 8). Of the 2 severe cases: Case 9 had contusions centred in the left anterior cingulate cortex, with atrophy in the frontotemporal cortices and in the left caudate nucleus. Case 10 had small contusions in the orbitofrontal cortex bilaterally.	apathy and withdrawal, memory impairment, and symptoms of depression. Treatment with lofepramine resulted in a decrease in both OCD and depressive symptoms.
[12] Carmin, Wiegartz, Yunus & Gillock, 2002	1 Age: 78 Male	Vascular disease, infarct	N/A	Self-reported anxiety attack at aged 29. Long history of hypertension and diabetes. Extremely avoidant to having	Two small lacunar infarcts, situated in the left basal ganglia.	Case 6: 6 months after the TBI, an intrusive musical tune started up, "playing" constantly in her mind (for at least the 1 month prior to the study.. She attempted to suppress the tune by occupying her mind with other thoughts. Personality changes also occurred, with aggressiveness and apathy, and she had little insight into these changes. Medication had been declined. All cases developed clinically significant OCD following their TBI. All reported multiple obsessions and compulsions. The mean number of obsessions and compulsions at the time of the study was 3.7 and 3.6 respectively. Common obsessions were those pertaining to aggression and contamination, symmetry and order, or somatic or sexual obsessions. The most common compulsions involved checking, cleaning/washing, and repeating behaviours. 5/10 had counting compulsions. Obsessional slowness was observed in cases 4, 9 and 10, while cases 7-9 demonstrated compulsive exercising behaviours. Comorbid psychopathology was very common—especially general anxiety (all cases), depression (9/10), PTSD (7/10) and an aggressive personality change (3/10). Overall, neuropsychological test scores were significantly lower for this group with TBI-related OCD, when compared to a control group, especially in the areas of executive function, memory and language.

[13] Stengler- Wenzke & Mueller, 2002	1	TBI, MVA	Severe	<p>blood pressure measured. Wife's breast cancer causing extreme distress.</p> <p>None specified.</p>	<p>Multiple lesions in right ventral-lateral prefrontal cortex, right anterior temporal lobe, corpus callosum, and adjacent white matter regions.</p>	<p>subsided, so that full resumption of his work and daily activities was reported at 6- and 12-month follow-up.</p> <p>Developed severe checking compulsions and obsessions following the injury, and reported greater impulsivity. He scored a total of 30 on the Yale-Brown Obsessive Compulsive Scale 2 years after injury. A course of fluoxetine for 90 days drastically reduced his compulsions (more than his obsessions), and re-testing at this time showed his Yale-Brown score had dropped to 10. He also reported a great improvement in his quality of life.</p>
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Table 14. Studies involving performance of obsessive-compulsive patients on selected neuropsychological assessment measures

Study	Participants	Exclusion factors?	Control group?	Neuropsychological tests used	Differences observed
[1] Behar, Rapoport, Berg, Denckla, Mann, Cox, Fedio, Zahn & Wolfman, 1984	16 adolescents with OCD Mean age: 13.7	Excluded if: <ul style="list-style-type: none"> • full-scale IQ<85 • definite neurological disease • they did not cooperate with testing • had psychotic symptoms • symptoms too mild to interfere significantly with academic and personal functioning. • depression is primary illness (secondary allowed) 	16 controls matched for age (\pm 1 yr), sex, race, handedness and IQ (within 15 points). No history of psychiatric illness or behavioural or learning difficulties in participants or their families. All in good health. No history of serious head injury.	Money's Road Map of Directional Sense, Stylus Maze Learning, RAVLT, Rey-Osterrieth CFT, tactual testing, reaction time.	Sig. deficits in maze learning, road map test, "immature" approach to complex figure copying. Spatial processing difficulties suggest right-hemisphere involvement; approach to complex figure suggests "neurodevelopmental immaturity" and/or frontal lobe problems.
[2] Harvey, 1987	19 OCD (DSM-III diagnosed) patients	Excluded if: <ul style="list-style-type: none"> ▪ neurological impairment ▪ psychosis ▪ alcoholism ▪ history of head injury 	A control group of patients with gross frontal lobe damage.	Nelson's Modified Wisconsin Card Sorting Test (MWCST), Alternating Category Verbal Fluency (ACVF), assorted subtests of WAIS.	MWCST: Based on normative data, OCD patients were found to perseverate significantly more than normals. Their mean percentage perseveration was also greater than frontal lobe damaged patients (50% and 42%, respectively). Perseveration correlated with degree of obsessionality on the Leyton Obsessional Inventory (Spearman's $r=0.50$). ACVF: This test also assesses cognitive set-shifting ability. Performance was negatively correlated with obsessionality ($r= -0.62$).
[3] Zielinski, Taylor, & Juzwin, 1991	21 OCD (DSM-III-R diagnosed) patients	Excluded if: <ul style="list-style-type: none"> ▪ pure obsessional (i.e., no compulsions) ▪ primary obsessional slowness ▪ current symptoms of major affective disorder ▪ previously diagnosed with schizophrenia ▪ history of alcohol or drug 	21 controls matched for age (\pm 5 yrs), sex, race, and education. No history of past or present mental or neurological illness, or history of psychiatric disorder in any	Raven's Progressive Matrices- short Form, CVLT, Corsi's Block Tapping Test, Recurring Figures Test (RFT), continuous performance test, WCST, Controlled Oral Word Association Test (COWA), Design	RFT: While no significant differences were observed between OCD patients and controls on number of figures recalled (either immediate or delayed), the OCD group made more false-positives (i.e., reported novel figures as existing in the original stimulus group) during both immediate and delayed trials. Corsi's blocks: OCD patients had smaller immediate recall span, and were less able to learn a recurring spatial pattern, than were controls. CVLT: The only difference observed was that the OCD

			<ul style="list-style-type: none"> ▪ abuse ▪ coarse neurological disease 	first-degree relative.	Fluency Test (DFT).	<p>group reported higher numbers of intrusions on the word lists.</p> <p>Raven's Matrices: The OCD group made significantly more errors than controls. This difference was largely restricted to the easier test items.</p> <p>No significant differences were found on the COWA, DFT, or WCST.</p> <p>Overall, OCD patients exhibited impairment on visuo-spatial measures, but not on verbal learning, frontal lobe, or attentional measures.</p> <p>Tower of London: OCD group spent more time generating alternative solutions once a mistake was made or checking that the next move would be correct. Suggestive of either difficulty in setting aside the main goal for planning necessary subgoals, or excessive monitoring of responses to ensure another mistake isn't made.</p> <p>Attention set-shifting. Steady decline in number of OCD that pass at each stage, indicating difficulty in shifting cognitive set and maintaining attention.</p> <p>Both impairments indicative of frontal lobe or fronto-striatal dysfunction.</p> <p>WCST: Schizophrenic patients performance significantly impaired; average performance for OCD and controls. suggests involvement of dorso-lateral prefrontal cortex in schizophrenia.</p> <p>OAT: OCD patients performance significantly impaired; average performance for schizophrenia and controls. Suggests involvement of orbito-frontal cortex in OCD.</p>
[4] Veale, Sahakian, Owen, & Marks, 1996	40	OCD (DSM-III-R diagnosed)	Excluded if:	22 healthy controls for Tower of London task, and 36 healthy controls for attention set-shifting task, all well-matched for age and verbal IQ.	NART, vocabulary and comprehension subtests of WAIS-R, Tower of London task, attention set-shifting task.	
[5] Abbruzzese, Ferri & Scarone, 1997	60	OCD and 60 schizophrenia patients from different hospital in-patient services.	Excluded if:	30 controls recruited from hospital employees and nursing staff with no history of psychiatric or neurological illness or alcohol abuse.	Wisconsin Card Sorting Task (WCST), Object Alternation Test (OAT).	

[6] Li, Chen, Yang, Chang, Liu, Shen & Chen, 2000	18 OCD and 12 Generalised Anxiety Disorder (GAD) patients from an outpatient clinic. Average ages: 28 (OCD) and 32 (GAD).	Excluded if: <ul style="list-style-type: none">▪ psychotic symptoms▪ symptoms and signs fulfilling criteria of major depressive disorder▪ major systemic illness▪ history of head injury▪ substance abuse (all subjects denied use of a substance other than tobacco or alcohol on a social basis)	18 normal controls, mostly made up of nurses, occupational therapy students, and other hospital staff. Average age = 25 years.	Perceptual alternation test (of Schröder's staircases).	OCD patients showed significantly higher alternation rates than normal controls. Alternation rates were also found to positively correlated with degree of obsessiveness. These findings supported the authors' hypotheses, suggesting that impaired inhibition of competing perspectives (due to impairment of the inhibitory function of cortico-striatal circuitry) may be the reason for higher alternation rates.
[6] Okasha, Rafaat, Mahallawy, El Nahas, El Seif, Sayed, and El Kholi, 2000.	30 OCD patients from an outpatient clinic. Ages: 16-40 ($\mu=24$)	Excluded if: <ul style="list-style-type: none">• comorbid current psychiatric illness• drug abuse• medical or neurological disorder (including tic or Tourette disorder)	30 controls, selected from hospital staff and volunteers, and matched for age, gender, and years of education. No history of psychiatric, medical or neurological conditions, or drug use during research period.	Selected WAIS subtests (incl. block design, object assembly, digit span, vocab), Wechsler Memory scale (WMS), Wisconsin Card Sorting task (WCST).	Object assembly: OCD scores significantly lower. Suggestive of defective visual recognition, pointing to possible right temporo-parietal dysfunction. WCST: OCD patients inferior to controls in trials administered, total errors, % of errors, % of perseverative errors % of conceptual level response and categories completed. This indicates possible impairments in set-shifting abilities, mental control, cognitive flexibility and abstraction, all associated with fronto-cortical dysfunction.
[7] Coetzer, Stein and Du Toit, 2001.	13 OCD patients, and 13 TBI patients, taken from clinical records (retrospective analysis),	Excluded if: <ul style="list-style-type: none">• Less than 10 years formal schooling• IQ < 99• Age <18 or >60	10 normal controls.	Shortened version of the SAWAIS (South African WAIS), the Austin Maze, Rey-Osterrieth Complex Figure Test.	TBI grouped performed significantly worse than the other groups on general intelligence. The TBI group were also more likely to use abnormal strategies on CFT. No differences were found between groups on the Austin maze. Overall, the OCD group performed as well as controls on all tests, failing to confirm the hypothesis that OCD patients and TBI sufferers exhibit similar executive impairments.

Table 15. Studies that detail neurological abnormalities found in obsessive-compulsive patients

Study	Participants	Exclusion factors?	Control group?	Measures/ types of scans used	Differences observed
[1] Behar, Rapoport, Berg, Denckla, Mann, Cox, Fedio, Zahn & Wolfman, 1984	16 adolescents with OCD Mean age: 13.7	Excluded if: <ul style="list-style-type: none">• full-scale IQ<85• definite neurological disease• they did not cooperate with testing• had psychotic symptoms• symptoms too mild to interfere significantly with academic and personal functioning.▪ depression is primary illness (secondary allowed)	16 controls matched for age (± 1 yr), sex, race, handedness and IQ (within 15 points). No history of psychiatric illness or behavioural or learning difficulties in participants or their families. All in good health. No history of serious head injury.	CAT scan, EEG	The OCD group had a significantly higher ventricular-brain ratio (VBR) than controls (7.4% compared to 3.4%), indicative of enlarged ventricles in the OCD patients. VBRs were not related to severity of obsessions, onset age, duration of illness, history of head trauma or neuropsychological scores. There was some indication that those OCD patients who had rituals without accompanying thoughts were more likely to have enlarged ventricles, although the sample size was too small for a significant finding.
[2] Machlin, Harris, Pearlson, Hoehn-Saric, Jeffrey & Camargo, 1991	10 OCD patients (DSM-III-R diagnosed) Mean age: 34.1	Excluded if: <ul style="list-style-type: none">• ever met criteria for major depression• history of central neurologic disorder (incl. TBI with LOC > 1 hr)	8 healthy controls matched for age, race and sex. The exclusion criteria also applied for controls, but also included any history of an axis I psychiatric disorder.	SPECT scan	OCD patients had a significantly greater medial-frontal perfusion value than did controls (109.7%, compared to 102.9%). This finding is consistent with the hypothesis that the frontal lobes are involved in OCD. The OCD group's medial-frontal perfusion values were negatively correlated with scores on the Hamilton Rating scale for Anxiety, but did not correlate with degree of obsessiveness. No differences in perfusion values were found for either the left or the right orbital-frontal cortex.
[3] Robinson, Wu, Munne, Ashtari, Alvir, Lerner, Koreen, Cole & Bogerts, 1995	26 OCD (DSM-III-R diagnosed) patients Mean age: 32.2	Excluded if: <ul style="list-style-type: none">▪ cardiac pacemaker, metallic clips, or other metallic implants or artifacts in the patient's body▪ significant medical illness▪ had psychosurgery▪ alcohol or psychoactive substance abuse or were considered	26 healthy controls recruited from advertisements and medical centre personnel. Controls had no history of psychiatric disorder or psychotropic medication use, and were considered	MRI scan (to determine volumes of prefrontal cortex, caudate nucleus, lateral and third ventricles and the whole brain)	No significant differences were found for size of the whole brain or the prefrontal cortex. The OCD patients had substantially larger lateral ventricle size, but this finding did not reach significance. There was, however, a significant difference in caudate nucleus volume, with OCD patients having much smaller caudate nuclei (the mean caudate volume for OCD patients was 750mm ³ less than the control group mean). This reduced caudate nucleus was significant for both left and right hemispheres. The authors argue that the findings provide additional

			<ul style="list-style-type: none"> ▪ dependence ▪ dementia, delirium, medically healthy. ▪ schizophrenia, ▪ schizoaffective, ▪ delusional, or psychotic disorder ▪ mental retardation ▪ pregnant 	Mean age: 29.8	evidence for the involvement of basal ganglia structures in OCD.	
[4] Hugo, van Heerden, Zungu-Dirwayi & Stein, 1999	6 patients who had developed OCD symptoms following brain trauma. Ages: 41 (M), 59 (M), 32 (M), 46 (F), 41 (M), & 29 (F).	None specified.	None.	CAT scan, SPECT scan	<p>The SPECT scans showed abnormalities in all 6 cases. In all cases there was decreased perfusion to the temporal lobes. Only the anterior part of the temporal lobes was involved in 5 of the 6 cases.</p> <p>All patients showed focal cortical perfusion abnormalities in the frontal lobes (either increases, decreases, or both).</p> <p>4 of the 6 cases also demonstrated decreased perfusion in the occipital cortices.</p> <p>Individual cases demonstrated perfusion abnormalities in a variety of other areas, including the parietal lobes, the right thalamus, and the left basal ganglia.</p>	
[5] Okasha, Rafaat, Mahallawy, El Nahas, El Seif, Sayed, and El Kholi, 2000.	30 OCD patients from an outpatient clinic. Ages: 16-40 ($\mu=24$)	Excluded if:	<ul style="list-style-type: none"> • comorbid current psychiatric illness • drug abuse • medical or neurological disorder (including tic or Tourette disorder) 	30 controls, selected from hospital staff and volunteers, and matched for age, gender, and years of education. No history of psychiatric, medical or neurological conditions, or drug use during research period.	MRI, Auditory event-related potentials (AERPs)	AERP results showed that OCD patients had higher P200 (Cz) amplitude to frequent stimuli, which the authors suggested could be caused by being overwhelmed by irrelevant stimuli that they can't disregard. The OCD group also showed lowered N200 amplitude (particularly for those with the most severe OCD symptoms), indicating inattention to task-relevant stimuli. The authors argue that this inattention is due to the inability to disregard irrelevant (frequent) stimuli, thus disturbing selective attention.
[6] Busatto, Buchpiguel, Zamignani, Garrido, Glabus, Rosario-Campos, Castro, Maia, Rocha, McGuire & Miguel, 2001	26 OCD patients (DSM-IV diagnosed). 13 were described as early-onset (<10 yrs at	Excluded if:	<ul style="list-style-type: none"> ▪ history of alcohol/ drug abuse ▪ history of neurological disorders ▪ use of psychotropic 	22 healthy controls, with no history of clinical or psychiatric disorders. Controls' gender, age and handedness	MRI (for initial exclusion), SPECT scan	Early-onset OCD cases showed decreased regional cerebral blood flow (rCBF) in the left anterior cingulate cortex, and right orbitofrontal cortex and increased rCBF in the right cerebellum compared to the control group. Late-onset OCD patients showed reduced rCBF in the orbitofrontal cortex and increased rCBF in the left precuneus.

onset), and 13 late-onset (>12 yrs). Mean current ages: early-onset-31.2 late-onset- 33.1	medication 3 weeks prior to the study (6 weeks if fluoxetine) ■ gross abnormalities on structural MRI scans (by a radiologist who was blind to psychiatric diagnosis)	were significantly different from the OCD group.	not	Early-onset and late-onset OCD patients differed in several ways also. The early-onset group showed decreased rCBF in the right thalamus, left anterior cingulate cortex, and bilateral inferior prefrontal cortex, compared to the late-onset group. Severity of OCD symptoms was also found to correlate positively with left orbitofrontal rCBF in early-onset patients, but no such effect existed for late-onset OCD patients. These results suggest that the brain mechanisms involved in OCD differ according to age at onset.
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BP's current situation regarding OCD-like symptoms

Frontal-striatal theories of OCD are consistent with the damage that BP experienced due to his TBI. His damage was very severe and widespread (i.e., DAI), but included the prefrontal cortex and basal ganglia, areas that one would expect to be implicated in the onset of obsessions or compulsions. BP's compulsive behaviours, his apparent need to do most things in multiples of three, have largely subsided over the last two years. In fact, the number three now appears to play a much smaller part in BP's everyday activities. Card games are still done in multiples of three, but this is probably mostly out of habit. Punching three times is now quite a rarity (probably also due to the fact that behaviour modification techniques have tried to eliminate punching and other aggressive behaviours altogether), and a preoccupation with exact times does not seem to include the number three any more than other numbers. There are some situations where multiples of the number three do still crop up, such as during Playstation games (as mentioned before), or if he is fatigued (when most problem behaviours are exacerbated). Nonetheless, there is no doubt that these compulsions have decreased to a great degree. Given that these behaviours did not cause anxiety, and that no rehabilitative effort was put into the specific treatment of them (there have always been much more important behaviours for rehabilitation to focus on, for both his safety and his independence), the question remains as to why this preoccupation with the number three subsided.

While it is true that different behaviours come and go throughout the course of recovery from TBI, the most likely reason for this decrease in OCD-like symptoms is the medications that BP currently takes. BP was prescribed a course of carbamazepine (Tegretol), an anti-convulsant, soon after his discharge from the private rehabilitation centre he had resided at for the 2 months following hospital discharge. This was in response to a grand mal seizure BP experienced one night in his bed. Since this time, occasional seizures have been suspected (usually in hindsight when associated with a rapid increase in fatigue and problem behaviours), but these have been few and far between. Approximately 1 year post-injury, BP was also experiencing mood fluctuations, with particular periods of dysphoria. To stabilise his mood, a course of citalopram (Cipramil), a selective serotonin reuptake inhibitor (SSRI), was begun. It was over the course of the following months that the OCD-like symptoms appeared to decrease. In order to provide support for the link between citalopram and a decrease in OCD-

like symptoms, the research into SSRIs and their usefulness in treating OCD is reviewed.

In a review of the pharmacotherapy of OCD, Stein (2002) argued that SSRIs have been shown to be the most effective line of treatment. While results of meta-analyses of OCD have suggested that less selective agents such as clomipramine have shown greater effect size, he explained that the methods of these meta-analyses had many limitations, and that all head to head studies have indicated that SSRIs are at least as effective. Agents from other drug classes (e.g., MAOIs, dopamine blockers, benzodiazepines) have not been shown to consistently lessen OCD symptoms (Stein, 2002). Given that SSRIs have a better safety and tolerability profile than clomipramine, Stein suggested that SSRIs be used as the first line of defense, with clomipramine to be tried only if SSRIs have been ineffective in lessening symptoms. This approach has also been suggested when dealing with the treatment of OCD secondary to TBI (Newburn, 1998).

One common SSRI used in the treatment of idiopathic OCD is fluoxetine. It has been shown that, like other SSRIs, OCD symptoms tend to diminish following treatment with fluoxetine. One such study investigated the effect of fluoxetine and a placebo on 103 children and adolescents with OCD (Geller et al., 2001). At one year, clinicians blind to the status of patients at baseline rated 55.0% of fluoxetine-treated patients as 'much improved' or 'very much improved' at the end of the 13-week treatment, compared to 18.8% of those who had been given a placebo. Scores on the Children's Yale-Brown Obsessive Compulsive Scale (CY-BOCS) were also found to be significantly lower for the fluoxetine group than for the placebo group. Other SSRIs, (including sertraline and fluvoxamine) are also mentioned as having effectively reduced OCD in other populations (Geller et al., 2001).

Citalopram is the most selective of the SSRIs (Thomsen, Ebbesen & Persson, 2001). A review by Joubert, Sánchez and Larsen (2000) described citalopram as being effective in treating major depression, as well as a variety of anxiety disorders (including OCD), with relatively minor side effects of transient nature (rarely continuing past the first two weeks of treatment). Thomsen, Ebbesen and Persson (2001) examined the long-term effects of a course of citalopram on 30 adolescents with idiopathic OCD. Dosage was between 20.8mg and 67.4mg per day (mean dose of 46.5mg) over the first year (this is compared to BP who has taken 20mg per day for the past two years). Results indicated that OCD symptoms decreased significantly over a two-year timeframe, with mean Yale-Brown Obsessive Compulsive Scale (Y-BOCS) scores dropping from 28.7 at baseline, to 23.3, 20.0, 18.4 and 17.9 at 10-week, 6-month, 1-year and 2-year follow-up. Overall, 70% showed a decrease of more than 35% in total Y-BOCS score from baseline to 1-year follow-up. However, 20% remained at clinical levels for OCD (Y-BOCS > 20) after a year of treatment, suggesting that citalopram (or SSRIs in general) may not be the best treatment for all OCD-sufferers. Also, while citalopram treatment was effective for the first year, no significant reduction in symptoms was found from 1-year to 2-year follow-up. A shortcoming of this study is that all patients received cognitive-behavioural therapy (CBT) in conjunction with citalopram, but the levels of CBT varied greatly for each patient, making it difficult to measure the exact effect that either treatment had on outcome. It would be wise for future research on the long-term effects of citalopram on an OCD sample to also factor in the effects of CBT. Results may support the use of only one of the treatment procedures, or uncover an interaction effect between the two interventions.

While research into the efficacy of SSRIs for the treatment of idiopathic OCD continues to grow, there is very little research specifically focusing on the treatment of posttraumatic OCD. The majority of the case studies in Table 13 described some form of treatment, with the most common being pharmacological treatment (sometimes in tandem with cognitive-behavioural therapy). SSRIs (e.g., fluoxetine, sertraline, citalopram, paroxetine) and tricyclic antidepressants (e.g., clomipramine, lofepramine) made up the majority of these, but other treatments included lithium and a monoamine oxidase inhibitor (MAOI). One study (Stengler-Wenzke & Müller, 2002) mentioned in Table 13 specifically focused on a 90-day trial of an SSRI (fluoxetine), based largely on the SPECT finding that their patient exhibited lower serotonin transport density in the midbrain and hypothalamus (as well the MRI findings of multiple prefrontal, orbitofrontal and temporal lesions). The patient's score on the Yale-Brown Obsessive Compulsive Scale decreased from 30 to 10 in this time, a significant improvement. The authors argued that based on their study, as well as other case reports, the same medications that have been found to be effective in the treatment of idiopathic OCD (namely, SSRIs) are also effective in treating secondary (organic) OCD. However, the use of such medication is often in a trial-and-error method, as some post-traumatic OCD cases have responded better to other forms of intervention (e.g., exposure and response prevention (ERP); Carmin et al., 2002).

The literature supports the theory that people with OCD have lower levels of serotonin, and that their symptoms can be treated effectively with SSRIs. This also appears to be the case for those who have acquired obsessions or compulsions as a result of a TBI. It is logical therefore, to assume that the near-elimination of BP's preoccupation with the number three is a direct result of the introduction of citalopram. While this medication is being used as a mood stabiliser, the observed

relationship between OCD-like behaviours and citalopram in BP provides further support for the serotonin-based theories of OCD, and for the use of treating those who actually have clinical OCD (idiopathic or otherwise) with SSRIs.

The boundaries between perseveration, rituals, and obsessions/compulsions

While it has been made clear that the behaviours exhibited by BP are in many ways quite different from classic symptoms of OCD, as defined by the American Psychiatric Association (1994), it is not so clear where the dividing line between compulsions and impulsive rituals characteristic of many sufferers of frontal lobe dysfunction is. Some researchers have suggested that there may be a significant overlap between executive function following TBI and OCD. For instance, Coetzer, Stein and Du Toit (2001) suggested that the repetitive nature of the symptomatology of OCD resembled the impaired capacity for many TBI patients to use and learn from mistakes. However, their research failed to support their hypothesis.

Perseveration, or the continuation or recurrence of experience or activity without the appropriate stimulus (Sandson & Albert, 1984), is commonly displayed by those who have suffered a TBI. Many of the studies in Table 14 note perseverative deficits amongst OCD patients during neuropsychological testing, and perseveration has been shown to be correlated with degree of obsessiveness on the Leyton Obsessional Inventory (Harvey, 1987). While both TBI and OCD populations display perseverative behaviours, each might have different reasons for these behaviours. Sandson and Albert (1984) propose that perseveration can be grouped into three different subtypes: (1) continuous perseveration of a current behaviour, (2) stuck-in-set perseveration (where a continuous and inappropriate set or framework is maintained), and (3) recurrent perseveration of a previous response to a subsequent

stimulus. The 'stuck-in set' type is associated with a breakdown in executive functioning, so that intention and actions become disconnected. The nature of this subtype is very close to one's idea of impulse disorders, much like that displayed by BP. Perseveration on tests that use attention and cognitive flexibility have tended to be associated with frontal lobe lesions, and interestingly, frontal lobe damaged samples have almost always demonstrated perseveration of the 'stuck-in-set' type (Sandson and Albert, 1984).

It is clear that while there are many similarities between the perseverative behaviours exhibited by frontal lobe damaged and impulse-disordered people, and the repetitive behaviours of OCD patients, one must also recognise the differences. OCD patients tend to have particular obsessions and behaviours, and these often fall into distinctive subtypes, such as 'contamination', 'counting' or 'checking'. While many behaviours displayed by people with impulse control disorders fit into such subtypes (e.g., BP's preoccupation with the number three is something one might expect from a person with a 'counting' form of OCD), it seems likely that most would not fit so neatly into these categories. In fact, most would involve the general impairment in being able to switch cognitive set to a different situation, set of circumstances, or stimuli. Also the impulse disorders often involve the presence of behaviours without any accompanying cognitive involvement. If associated with cognitive states, it is often a result of the initial impulsive reaction (e.g., anger, guilt), rather than a causal factor. This is certainly the case with BP, where his impulses do not seem to be triggered by cognitive processes, but occur in and of themselves (barring risk factors: e.g., fatigue, loud noise, big crowds). BP reports no accompanying anxiety to the multiples-of-three compulsions.

Of course, obsessive-compulsive disorder is by definition an anxiety disorder, and for a clinical diagnosis compulsions must be elicited with the intent in mind that they diminish anxiety caused by the initial obsessions. While it is true that the majority of reported cases of OCD following TBI have reported anxiety, and would qualify for a clinical diagnosis if it were not for the ‘caused by general medical condition’ clause, the fact that many frontal lobe patients show similar behaviours without anxiety suggests that the external boundaries of OCD are not so clear-cut.

Castle and Groves (2000) discussed the fuzziness of the external boundaries of OCD. They suggested that, due to the difficulties in defining the ‘caseness’ of OCD, it would be a useful approach to consider OCD to be a disorder with permeable external boundaries, and considerable overlap with a variety of other disorders. These include ‘impulse control’ disorders, neurological disorders associated with OCD features, and disorders regarding bodily appearance or sensation (e.g., anorexia nervosa). All of these disorders could be put along a spectrum according to their cognitive or behavioural component. Another difficulty with defining the external boundaries of OCD is the common comorbidity among OCD patients. It is more the rule than the exception that a person with OCD will have at least one comorbid psychiatric illness (often depression) (Castle & Groves, 2000). OCD symptoms might in fact be secondary to another psychiatric disorder.

There has been a call for the reconsideration of OCD being classed as an anxiety disorder. Summerfeldt and Endler (1998) reviewed the literature on the cognitive biases of OCD. Overall, their conclusion was that OCD of the ‘contamination’ variety was the only subtype to be associated with similar cognitive tendencies as the other anxiety disorders (i.e., associated with emotional tone or *content* at the attentional

level). The research on other forms of OCD are so varied and inconsistent (compared to the other anxiety disorders), that it is suggested that OCD is qualitatively different from the other anxiety disorders, and that anxiety may not be the key feature of OCD (Summerfeldt & Endler, 1998). In fact, it is argued that perhaps OCD symptoms trigger anxiety, rather than the other way round. Either way, there is plenty of support for the notion that in many ways OCD sits better alongside impulse control disorders, than it does with anxiety disorders (e.g.; Summerfeldt & Endler, 1998; Castle & Groves, 1999; Newburn, 1998).

Self-stimulation

The discussion of a spectrum of disorders on an impulsive-compulsive continuum warrants a particular focus on the specific stimulation-seeking behaviour that is observed in BP. BP's search for new stimulation is a constant. This may come in the form of attention from others, in the hit of nicotine from a cigarette, in drugs or alcohol, or the intake of excessive amounts of sugar. One of the greatest difficulties in the rehabilitation of BP has been in finding alternative activities that are as or more reinforcing for BP than these forms of stimulation (i.e., what could be more stimulating for a person with difficulty in creating long-term goals and a focus on short-term satisfaction, than getting intoxicated?).

Hollander (1998, cited in Castle & Groves, 2000), supporting the idea of a spectrum of OCD-like psychiatric disorders, proposed an impulsive-compulsive continuum based on 'risk avoidance'. At one end lie the 'impulsive' individuals (e.g., people with sexual compulsions or impulsive personality disorder), who are risk-takers trying to maximise pleasure, arousal or gratification. At the other end lie the 'compulsive'

individuals, whose primary aim is to avoid harm or reduce anxiety (e.g., anorexia nervosa). This heuristic is helpful in trying to explain the overlap among many disorders associated with impulsive or compulsive behaviour, and the inconsistencies in cases of those with these disorders. It is also supported by the literature that suggests different neurochemical substrates are associated with different ends of the spectrum. Namely, impulsive disorders have been shown to respond rapidly to serotonergic agents, but for their effectiveness to decrease over time, while disorders at the compulsive end of the spectrum tend to have a slower onset of response but longer-term gains (Hollander, 1998, cited in Castle and Groves, 2000). It appears that BP would be at the 'impulsive' end of the spectrum, given his constant need for stimulation.

Whether or not OCD can be considered an anxiety disorder, or is better suited to being placed along a spectrum of impulsive-compulsive disorders is still being debated. There is a general consensus, however, that given the considerable overlap of OCD and other related psychiatric disorders, such individuals need to be taken on a case-by-case basis. Keeping external boundaries of impulse and compulsive disorders blurry (while still using the rigid criteria for clinical diagnosis), may be very helpful when trying to understand and treat OCD and related disorders.

Conclusion

While BP does express several behaviours that are very characteristic of people with obsessive-compulsive disorder, there is no doubting that such a diagnosis would be wholly inappropriate on several counts. Firstly, his compulsions about the number three are in no way elicited by anxiety. Secondly, they are due primarily to a 'general medical condition', namely a severe frontal lobe injury. Thirdly, they may be better

assigned to symptoms of impaired impulse control (i.e., frontal lobe dysfunction), which would be in line with the rest of BP's symptoms. Nonetheless, the similarities warranted a review of the overlap between OCD and TBI of the sort BP experienced. The majority of current theories of OCD implicate the frontal-striatal pathways. Case studies of OCD following TBI, as well as neuropsychological and neuroimaging profiles of those with idiopathic OCD, largely support these theories. The effectiveness of SSRIs in the treatment of OCD (both idiopathic and secondary to TBI) has been demonstrated, which would explain the consistent decrease in his 'multiples-of-three' OCD-like behaviour following the introduction of citalopram for mood stabilising. The distinction between OCD and other impulsive-compulsive disorders is a blurry one, and the ill fit of OCD amongst other anxiety disorders suggests it may be better to consider OCD as lying on a spectrum of psychiatric disorders. One proposed spectrum based on risk seeking or risk avoidance appears to have merit.

Chapter 5: Caregiver burden following TBI

Caregiver burden refers to the stresses that are faced by the many people in our society that are faced with the role of caring for a sick or disabled person. These are often family members or spouses. Those who need care are a varied and diverse group, so a discussion of the psychological distress incurred by caregivers must always be wary of the heterogeneity of the population of people being cared for. Caregivers of those with traumatic brain injury, while facing many of the same stressors involved with caring for other populations, are likely to have stressors specific to TBI. For instance, the lengthy processes of rehabilitation, and the uncertainty regarding prognosis, are likely to be particularly stressful. Even within this population, the huge variety in outcome means that TBI caregivers may be dealing with completely different stress-inducing behaviours. For instance, a person with frontal lobe impairment may be more difficult to care for (for a variety of reasons mentioned throughout this chapter, particularly regarding personality change), than a person who has a focal lesion in the primary motor cortex.

BP exhibits many post-TBI behaviours that may cause significant distress in his caregivers. His personality change following the injury (e.g., becoming more outspoken and brash, wanting to socialise with anyone he comes across, but with a reduction in focus and drive) may be particularly difficult to deal with for those close to BP. His impaired motivation and initiative may be particularly distressing for caregivers with regard to participating in rehabilitation. His diminished interests and initiative may cause distress in that his parents may see an uncertain future for their son. Myself, as a caregiver, have found BP's emotional incontinence, or rapid fluctuations in expressed mood (often dissociated from his actual mood) particularly

distressing. For instance, going from an aggressive action or insult to the other extreme of inappropriate closeness (e.g., he says ‘I love you’ often throughout the day often within minutes or seconds of an incongruous behaviour). It is expected that those caring for TBI individuals that display such huge and rapid fluctuations in expressed mood (with extremes that are not in line with social norms of expressed emotion) would be more likely to be suffering from caregiver burden.

The hypothesis for this chapter, based on the caregiver burden literature, is that caregivers of TBI patients who exhibit such behaviours would tend to have higher levels of psychological distress than other caregivers. ‘Fluctuating extremes of expressed emotion’ (or “over-the-top” behaviour) is not a very well defined construct however, and thus has not been examined in the caregiver burden literature. To investigate the caregiver burden literature for this specific behaviour, this construct must be inferred from the investigation of other variables (e.g., the effects of specific behaviours rather than cognition/physical complaints on caregiver burden; social impairment; mood changes; emotional sequelae). Qualitative research on the specific TBI behaviours that cause the most distress in caregivers would be needed to fully support this hypothesis.

This chapter outlines the literature on caregiver burden. It begins by explaining the concept of caregiver burden, the ways in which it is measured and the epidemiology of caregivers. A particular focus on caregivers of people with traumatic brain injury follows, and the research literature regarding caregiver burden for this population is discussed. The next section details those factors found to increase burden in TBI caregivers, finishing with a detailed discussion of the literature regarding the effects of those specific behaviours elicited by people post-injury that have been associated

with higher levels of distress in caregivers. Those behaviours that BP displays, particularly those pertaining to the current hypothesis, are given particular attention.

Defining caregiver burden

Caregiver burden refers to the strain experienced as a result of the caring role. A distinction has been made in the literature between ‘objective’ burden (measuring the stressors themselves, e.g., problem behaviours in the TBI patient that are deemed to cause stress) and ‘subjective’ burden (self-report measures focusing on the level of stress actually experienced by the caregiver). Such a distinction seems logical, as what may be considered stressful by researchers may not actually be considered as such by some caregivers. Some researchers have suggested that ‘caregiver burden’ is too broad a construct to provide meaningful information about specific distress experienced by caregivers (Kreutzer, Gervasier & Camplair, 1994, cited in Morris, 2001). For this reason, the focus in this area of research has shifted towards measuring psychological distress, a construct that is both better defined and easier to replicate (Morris, 2001). The presence of other affective disorders that have been linked to stress (e.g., depression, anxiety) have also been investigated in caregivers (e.g., Harris, Godfrey, Partridge and Knight, 2001), as have such associated variables as quality of life and overall psychiatric morbidity (Moules & Chandler, 1999), family functioning (Douglas & Spellacy, 1996), family coping and structure (Curtiss, Klemz, & Vanderploeg, 2000), and degree of life change post-injury (Wallace et al., 1998).

Epidemiology of caregivers

Much of the responsibility for taking care of a brain-injured injured person in the long-term falls on informal caregivers (typically parents or spouses) (Knight, Devereux, & Godfrey, 1998). In one study investigating caregiver burden following

severe TBI (Knight, Devereux, & Godfrey, 1998), the participants were found by using the primary caregivers of 52 members of head injury societies. 43 were found to be women, while only 9 were men, and they had an average age of 47. All of the 52 caregivers were either relatives (29 were parents of the person being cared for, and 6 more distant relatives, such as grandparents or siblings) or spouses (17 participants). The majority (53%) had successfully completed a formal qualification at university (e.g., school certificate or university entrance), and 12% had attended university (not particularly low when you consider the average age of the caregivers in this sample). 60% were described as being in paid employment, 17% were retired, 10% “worked from home”, and the other 13% were either students or on a welfare benefit (it is important to keep in mind that the majority of the 40% not in paid employment were likely put in this position because of the caring role they had found themselves in). A study of stress in 60 caregivers by Ergh et al. (2002), found similar demographics, with 72% women, a mean age of 54 years, and years of education ranging from 5 to 18 years. More than half of the sample were parents of the TBI patient, with the remainder made up of spouses (20%), siblings (15%), children (3%), grandparents (3%), and 4 others (a nephew, uncle, sister-in-law and friend). Other studies have found similar demographic breakdowns (e.g., Blankfeld & Holahan, 1999; Marsh, Kersel, Havill & Sleigh, 1998a; Morris, 2001).

Manifestation of caregiver burden

Caregiver burden can manifest itself in many ways. Typically stress impacts on a person by increasing general anxiety levels, and perhaps less directly, by increasing levels of depression and dysphoria. Both anxiety and depression are particularly common among caregivers of both TBI and other illness-related caregiving. Other negative consequences that caregivers often face include psychosomatic disorders,

increased use of alcohol and/or drugs (prescription or otherwise), financial difficulties, changing roles, increasing social isolation, and poor psychosocial adjustment (Marsh, Kersel, Havill, & Sleigh, 1998a).

There is some evidence in the research literature on caregiver burden that women may be of particular risk, experiencing greater burden and impaired psychological adjustment than men (Blankfeld & Holahan, 1999). This is of interest given that the overwhelming majority of caregivers are women. It could be that women are more often shouldered with the responsibility of caring for a relative with TBI, regardless of whether they are prepared or skilled in the role. Interestingly, men have been found to report more burden for other forms of caregiving (e.g., caring for a person with dementia) (Martin, 2000), suggesting caring for a person with TBI presents unique stressors that may be more distressing for women (e.g., sexual inappropriateness). On the other hand, Sander et al. (1997) reported higher levels of emotional distress in male caregivers of TBI patients than female, suggesting that a particular risk for women may not exist after all. Male caregivers may report their distress in terms of anger and fatigue, rather than depression and anxiety (Perlesz, Kinsella & Crowe, 2000). Further research in the area may clarify any effect of gender on caregiver burden.

Studies examining the effect of race have tended to show that White caregivers are more likely to complain of burden than African American caregivers (Martin, 2000), or that levels of burden are comparable but that African American caregivers report less needs being met by care providers (Nabors, Seacat, & Rosenthal, 2002). Other studies have found no effect for race on caregiver burden (e.g., Harris, Godfrey, Partridge & Knight, 2001). Research into the relative caregiver burden experienced in

New Zealand by different ethnic minorities (particularly in examining the difference between Maori, Pacific Island and Pakeha carers) is needed.

Caregiver burden for TBI populations

The stresses for those providing care for people post-TBI is similar in many ways to other groups of caregivers. Allen et al. (1994, cited in Knight, Devereux, & Godfrey, 1998) found that the subjective burden for the relatives of 131 head-injured people was comparable to the relatives of other disabled groups (e.g., cystic fibrosis, muscular dystrophy). Some differences have been found between caregivers of TBI patients and other caregiver populations however. One such study (Pelletier, Alfano, & Fink, 1994) demonstrated that family members of people who had suffered a moderate to severe closed head injury reported significantly greater symptoms of depression, and greater perceived stress with regard to difficult personality characteristics of the patient and day-to-day problems with caregiving, than families caring for a person with spinal cord injury. Family members of closed head injury patients have also reported greater levels of stress in relation to lack of support and overcommitment (Alfano, Neilson, & Fink, 1994).

In a study involving 69 primary carers of severe TBI patients in a New Zealand sample, it was found that 39% of respondents reported clinically significant levels of anxiety, with 9% rated at severe, at 6-month post-injury (Marsh, Kersel, Havill & Sleigh, 1998a). Depression was also high amongst the caregivers in this sample, with 37% reporting clinically significant levels, and 10% experiencing severe depression. Interestingly, anxiety and depression levels in the same sample were found to be similar at 1-year post-injury as they were at 6-months (35% still experienced clinically significant anxiety, and 32% depression), although the severity of these

conditions appeared to be decreasing slightly over time (only 3% reported severe levels of anxiety at this time, while 8% still suffered from severe depression) (Marsh, Kersel, Havill & Sleigh, 1998b).

Social adjustment was also examined in the two studies by Marsh et al. (1998a; 1998b), and was found to be impaired in the caregiver sample in several areas. The biggest changes to caregivers' lives throughout the first year following the TBI appeared to be changes involving the family unit or with 'overall' change, followed by those involving social and leisure activities or the extended family. Disruption in marital, work and parental roles, while less prevalent, were mentioned consistently enough to also warrant attention. These studies indicated that impairment in social adjustment for primary caregivers' of TBI patients is widespread across different aspects of social functioning, and that while the effect is lessened over time, social adjustment continues to be a problem for caregivers' in the long-term (at least to 1 year post-injury).

Caring for a person that has suffered a TBI can be very costly, through lost work hours and opportunities, adaptations to the home, and the general cost of care and medical treatment. This added financial burden would likely contribute significantly to subjective stress levels of carers. Osberg et al. (1997) queried parents of children who had suffered a TBI about their financial and work stresses 1-month and 6-months post-discharge from hospital. Results indicated that TBI caused financial problems in many parents (30% of the sample 'agreed' or 'strongly agreed' at 1-month, and 24% at 6 months), but that financial pressures were of particular concern to parents of severe TBI children (79%, and 57% respectively). In fact, severity of injury was found to have a significant effect on all aspects of work and financial problems tested,

at both 1-month and 6-months post-injury. The persistence of more than four out of nine areas of impairment (vision, hearing, feeding dressing, eating, walking, bathing cognition and behaviour) was also significantly related to the experience of financial difficulties. Supporting the notion that financial problems can be a burden for caregivers, household income for families dealing with TBI has been found to be negatively correlated with affective/behavioural burden (i.e., poorer socio-economic households tend to suffer greater levels of burden) (Nabors, Seacat, & Rosenthal, 2002).

It is perhaps unfair to clump all caregivers in one group, as this is a diverse group with differing relationships to the brain-injured individual. Spouses and parents deal with caring for their loved one in a different way, as they often have different issues to deal with. Parents have been found to be more concerned about the future of their brain-injured child than spouses are about their partner's, whilst spouses express less positive feelings about their caregiving (e.g., 'caring has enriched our family life') (Allen et al., 1994, cited in Knight, Devereux, & Godfrey, 1998). Leathem, Heath and Woolley (1996) investigated role change, social support, perceived stress and health problems in a sample of New Zealand caregivers of TBI. Results demonstrated that partners tended to indicate higher levels of role change than parents post-TBI, particularly with regard to finances and the relationship (e.g., less close, less sharing of co-operation). Parents also tended to experience more health problems and report more stress, although neither of these differences reached statistical significance. Interestingly, differences in types of support utilised may have had a moderating effect on burden measures (in line with research that has demonstrated that partners often don't consider offered support to be for them (Morris, 2001)). Another study reported higher levels of anger and family satisfaction in wives of people with TBI

than in mothers (Perlesz, Kinsella, & Crowe, 2000). While a considerable number of studies do attribute higher burden to partners of TBI, the literature regarding the amount of stress suffered by different types of caregiver has been inconsistent. However, there is a general consensus that different relationships between caregiver and TBI person may result in different types of stress and burden, and that the nature of this relationship needs to be taken into account when examining TBI-related stress (Curtiss, Klemz & Vanderploeg, 2000).

Understandably (given that the large majority of primary caregivers of people with TBI are parents or caregivers), there is a dearth of research on caregiver burden in caregivers that are neither family members nor spouses. Psychological distress in primary, secondary and tertiary caregivers was investigated in one study however (Perlesz, Kinsella, & Crowe, 2000). Tertiary caregivers were mostly made up of siblings of the person with TBI. As expected, primary caregivers were found to have higher levels of distress than secondary or tertiary caregivers. Against the authors' hypotheses however, a higher proportion of tertiary caregivers reported clinical levels of depression, anxiety, anger and family dissatisfaction. Those caregivers and family members that play a lesser part in the caring role may not be receiving adequate assistance and support. Continued research on the distress suffered by caregivers other than parents and partners may uncover altogether different problems and stresses than those already found in the literature.

Factors affecting caregiver burden

Pre-injury factors

It is important to remember that caregiver functioning post-injury must be related to pre-injury functioning. The risk factors for TBI (see Chapter 2), such as alcohol and

drug abuse or low socio-economic status, may contribute to stress, depression and anxiety both before and after a head injury (i.e., being a caregiver of a TBI person is not the primary cause of stress in some families). Also, behaviour problems, another risk factor in suffering a TBI, may have been causing caregiver stress in some parents before the injury occurred. Some pre-injury factors appear to place families at greater risk of disruption, such as poorer psychological functioning, and psychiatric disturbance in parents (Wade, Drotar, Taylor, & Stancin, 1995). In any case, it is necessary to examine pre-injury functioning in order to determine to what extent caregiver burden is a result of the stresses of caring for a TBI person.

Social support

Social support refers to a person's emotional back-up, guidance and tangible assistance from family, friends, and other people in their broader social network (Blankfeld & Holahan, 1999). Social support available for caregivers of people with TBI, and the total types of social support used by caregivers, have both been linked to lower levels of stress (Leathem, Heath, & Woolley, 1996) and better psychological health (Pelletier, Alfano & Fink, 1994).

Douglas and Spellacy (1996) attempted to quantify the degree of association between 4 variables (demographic (e.g., age of TBI patient), injury-related, patient functioning and caregiving functioning) and the level of family functioning for 30 families living with a severe TBI relative. Post-hoc analysis showed that self-reported social support (composing of four factors: problems with excessive responsibilities and demands, problems with strong-tie relational support, lack of money, and lack of involvement) accounted for 43% of the variance in family functioning post-TBI. Of these factors, the problem of excessive responsibilities and demands was the only one to uniquely

contribute to the prediction of family functioning. When injury severity and the presence of residual deficits were added to social support in the regression analysis, the explained variance increased to 58% of the total variability for long-term family functioning after severe TBI. Sander et al. (1997) also reported that satisfaction with social support was a significant predictor of psychological distress in caregivers of people with severe TBI. Interestingly though, actual social support was not a significant predictor of distress.

A very in-depth study by Ergh, Rapport, Coleman and Hanks (2002) attempted to assess the moderating affect of social support on how stressors (e.g., neurobehavioural impairments in the TBI person, cognitive dysfunction, time since injury) affect the distress experienced by 60 primary caregivers of TBI patients. Level of social support (as measured by the Social Provision Scale (SPS), a 12-item self-report questionnaire that assesses perceived social support) was found to have a direct and linear relationship with family functioning (i.e., the less social support the more family dysfunction). Of particular interest is the fact that while awareness of deficits, cognitive dysfunction and time since injury were all found to significantly increase psychological distress in caregivers who have inadequate social support, these effects were not significant when adequate social support was provided (or at least was perceived to be adequate). This moderating influence of social support has important implications with regard to looking after the well being of primary caregivers of TBI patients. Those involved in the rehabilitation process should be aware of this influence and should not be afraid to ask carers if they feel they have sufficient support from their friends, family, and rehabilitation providers. This mediating role of social support has also been effectively demonstrated in other studies (e.g., Blankfeld & Holahan, 1999).

Social support for different types of caregivers is likely to be qualitatively different. It is likely that spouses would feel a social stigma attached to some of the behaviours their partner is displaying, and thus may feel uncomfortable talking about these issues with others. Some of the outcomes of TBI may be more in line with social norms for children (e.g., childishness, irresponsibility, mood changes), which may make it easier for parents and family members to talk about the child's behaviour with friends and rehabilitation professionals. Research has indicated that, despite both parents and partners indicating satisfaction with levels of support, parents use more of the social support available than do partners (Leathem, Heath, & Woolley, 1996).

Coping responses

The way caregivers react to stressors and cope in the face of stress is highly important regarding the subjective burden a carer may face. Moore, Stambrook, Peters and Lubusko (1991) demonstrated that wives of men with TBI who used coping resources (both external, such as church groups, or internal, such as reframing family problems) more frequently, reported greater levels of marital satisfaction. Other studies have shown that different families use different types of coping strategies (e.g., cognitive, behavioural) in the face of TBI-related caregiver stress, and that types of coping also change according to time since the injury (Kosciulek, 1999, cited in Curtiss, Klemz & Vanderploeg, 2000). A study of family structure and coping strategies following TBI (Curtiss, Klemz & Vanderploeg, 2000) found that the types of coping strategies used not only differ from pre- to post-TBI, but are also different for varying types of family structures. This is especially important given that caregivers that use emotion-focused coping have shown greater levels of emotional distress than caregivers that use a more problem-focused coping style (Sander, High, Hannay, & Sherer, 1997). It

is clear that while coping responses have a significant impact on the development of caregiver burden, this relationship is a very complex one.

Theories of caregiver burden have often employed interactional theories of stress (Knight, Devereux, & Godfrey, 1998). These models emphasise the mediating factors of social support and coping resources, so that a threat or stressor will exert strain and stress on a person once those coping resources (e.g., support from others) are exhausted (Brooks, Campsie, Symington, Beattie, & McKinlay, 1986). A stress-appraisal coping (SAC) model (Godfrey, Knight, & Partridge, 1996) goes one step further in proposing that emotional distress is experienced once an individual *perceives* that environmental demands exceed available coping resources. The inclusion of carer appraisal may be helpful in conceptualising the way coping resources mediate the development of caregiver burden following TBI. A study by Harris, Godfrey, Partridge and Knight (2001) supported the usefulness of this model. They demonstrated that adverse effects on family members following TBI was the only significant predictor of the development of depression in caregivers (out of seven predictor variables), but that carer appraisal of these adverse effects on family had a mediating effect on the relationship between stressors and caregiver depression. Future use of SAC models when investigating the effects of coping resources and stressors on caregiver burden is suggested.

Provision of information

It is widely regarded that the provision of information for caregivers about traumatic brain injury is invaluable. However, there is a dearth of research on the effects providing information has on caregiver burden (Morris, 2001). It seems logical that caregivers that are well informed will be less burdened by unexpected TBI outcomes,

and will have better knowledge and access to services for both the person with TBI and the carer themselves. A literature review of carers' needs showed that those needs described as important were predominantly about receiving adequate information (Sinnakaruppan & Williams, 2001). Morris (2001) attempted to measure the effect that providing written information (an information booklet about head injury, cognitive impairment, emotional and behavioural change, and 'looking after yourself') has on the development of burden in caregivers of TBI sufferers. Levels of distress were found to decrease slightly as a result of receiving written information, but these decreases did not reach statistical significance. This could be due to the TBI sample, which included mild, moderate and severe TBI. A sample consisting of just severe TBI caregivers may have reached significance. Moreover, caregivers in the study overwhelmingly responded that such written information would be beneficial at discharge from hospital. Given that the TBI sample consisted of people who had suffered a head injury 2 months or more ago, the lack of significant result could be due to the information not being supplied early enough post-injury. More research on the effects of providing information to carers is needed.

Specific behaviours in the TBI individual

Coping with changes in cognitive functioning has been found to heighten caregiver distress, and be an important predictor of life change among caregivers of a family member recovering from a TBI (Wallace et al., 1998). Social aggression and cognitive dysfunction have been reported by a group of parents of TBI children as being more stressful to cope with than physical deficits (Allen et al., 1994, cited in Knight, Devereux, & Godfrey, 1998). However, Ergh, Rapport, Coleman and Hanks (2002) argue that no consistent pattern between caregiver distress and specific cognitive impairments have been established. While not having the support of empirical

evidence it is important to note however that they also argue that it makes intuitive sense to expect those with executive function impairments to be particularly stressful and demanding to care for, given the suspected extra supervision required.

While the literature is inconclusive regarding the importance of cognitive problems on caregivers post-TBI, studies have repeatedly shown that personality and emotional changes have a more profound impact on the wellbeing of caregivers than physical changes do (Brooks, Campsie, Symington, Beattie, & McKinlay, 1986). Ergh et al. (2002) found that neurobehavioural and affective disturbance in a TBI sample, as measured by their total scores on the Neuropsychology Behaviour and Affect Profile (NBAP), was the strongest predictor of caregiver distress. Unfortunately the effect of specific behaviours was not sought as the focus was on investigating the moderating influence of perceived social support on family functioning and caregiver distress. A study by Oddy, Humphrey and Uttley (1978, cited in Knight, Devereux, & Godfrey, 1998) demonstrated that caregiver stress was more strongly related to the presence of behavioural problems and symptoms exhibited by the TBI-sufferer, than initial TBI severity.

Knight, Devereux, & Godfrey (1998) attempted to uncover which behaviours were particularly stressful to primary caregivers of those who had suffered a severe TBI (as well how the social support and perception of coping skills impacted on their feelings of caregiver burden). The participants (the primary carers of 52 TBI patients that were members of the Head Injury Societies of Canterbury, Otago and Southland) who completed the questionnaires did so at differing stages of the recovery process, with the average year since injury being approximately 6 years. A list of the symptoms asked about, their prevalence, and their subjective distress rating given by caregivers

(ranging from 0 (no distress) to 3 (severe distress)) can be found in Table 16. Of these, the behaviours that BP displays are highlighted.

Table 16. Percent frequency and subjective distress ratings for behavioural effects of TBI (adapted from Knight, Devereux, & Godfrey, 1998, p. 472)

Behaviour	Frequency	Distress Rating Mean (0-3) (and standard deviations)
Impatience	85%	1.82 (0.69)
Anger	77%	1.98 (0.70)
Rapid mood change	71%	2.05 (0.74)
Poor judgement	69%	1.64 (0.76)
Anxiety	67%	1.97 (0.75)
Easily upset	67%	1.91 (0.70)
Depression	65%	2.09 (0.79)
Impulsivity	65%	1.82 (0.80)
Argumentative	63%	1.70 (0.73)
Loss of motivation	58%	1.63 (0.76)
Irritability	56%	1.90 (0.77)
Loss of interest	52%	1.81 (0.83)
Loss of control	52%	1.59 (0.80)
Poor insight	50%	1.81 (0.85)
Childishness	48%	1.60 (0.82)
Querelousness	40%	1.38 (0.59)
Aggression	40%	1.95 (0.80)
Loss of initiative	37%	1.53 (0.70)
Irresponsibility	35%	1.94 (0.80)
Over dependence	29%	1.67 (0.72)

A New Zealand sample of 69 severe TBI patients were followed up 6 months post-injury to determine the levels of burden their caregivers faced, and what particular aspects of their situation tended to exacerbate this burden (Marsh et al., 1998a). One particular focus was to determine which behaviours exhibited by the brain-injured individual were particularly stressful to caregivers. These same authors also revisited the same sample 1-year post-injury, once again in order to determine which factors increased subjective and objective burden (Marsh et al., 1998b). Participants were asked to identify which of 20 brain injury-related behavioural symptoms were present in the individuals they cared for, and to rate how distressful they found they behaviour

(on a 4-point scale; 1 = no distress, 4 = severe distress). A list of the prevalence of the 20 behaviours and the subjective distress this caused caregivers, at both 6-months and 1-year post-injury, can be found in Table 17. Once again the problem behaviours particular to BP are highlighted.

While there was some variation in the behaviours chosen when compared to the research undertaken by Knight, Devereux, and Godfrey (1998), there is sufficient crossover to be able to compare the results. One particularly startling observation from Marsh et al.'s (1998a; 1998b) findings is that the prevalence rates for many behaviours actually increased from 6-months to 1-year post-injury (only 6 of the 20 behaviours measured decreased in prevalence over this time). Knight, Devereux and Godfrey's (1998) head-injured sample had an average time since injury of 5.7 years ($sd=4.55$), and prevalence rates for problem behaviours were found to be

Table 17. Percent frequency and subjective distress ratings for behavioural effects of TBI (adapted from Marsh et al., 1998a, p. 231; Marsh et al., 1998b, p. 1051)

Behaviour	Frequency at 6-months post injury	Frequency at 1-year post-injury	Distress Mean (1-4) at 6-months	Rating	Distress Mean (1-4) at 1-year post-injury	Rating
Impatient	65%	74%	2.57	2.61		
Depressed	63%	54%	2.44	2.60		
Poor decision making	56%	48%	2.63	2.39		
Lack of interest	52%	54%	2.60	2.65		
Overly sensitive	52%	61%	2.57	2.31		
Poor insight	52%	49%	2.51	2.50		
Impulsive	52%	61%	2.23	2.17		
Mood changes	50%	45%	2.74	2.65		
Anger	50%	55%	2.68	2.76		
Irritable	50%	48%	2.68	2.49		
Lack of Motivation	50%	54%	2.56	2.68		
Childish	50%	61%	2.38	2.26		
Anxious	49%	49%	2.39	2.44		
Argumentative	47%	58%	2.69	2.33		
Lack of initiative	44%	42%	2.30	2.24		
Social behaviour	37%	38%	2.68	2.54		
Complaining	35%	39%	2.63	2.33		
Dependent	29%	38%	2.60	2.69		
Aggression	28%	32%	2.79	2.50		
Irresponsible	28%	29%	2.58	2.55		

considerably higher still at this time. For example impatience, cited in by both samples as being the most common behaviour post-trauma, was reported by 65% of caregivers at 6-months, 74% at 1-year, and 85% in the longer-term (average of 6 years post-injury). Given that both samples involved caregivers for severe TBI individuals in a New Zealand population, it may be reasonable to assume that the higher prevalence rates are due primarily to the increased time since injury. However, one possible reason that prevalence rates for Knight et al's study may be higher, is that TBI patients that are still needing care in the very long-term (i.e., 6 years) are likely to have been more severely injured than the average TBI patient being cared for at 1-year (i.e., many of Marsh et al's TBI sample may have been on the verge of independent living at 1-year). Regardless, the general increase in head injury-related symptoms is alarming and suggests that support for caregivers must continue years after the initial injury. It may be argued that the increase in behaviours can be attributed at least partly to a drop-off in the rehabilitation services provided, or to increasing psychosocial problems like social isolation and drug abuse. It is also interesting to note that some behaviours showed significant decreases from 6-months to 1-year post-injury, particularly depression (63% and 54%, respectively), poor decision making (56% and 48%), and mood changes (50% and 45%). Given that these symptoms have all been found to be particularly stressful for caregivers, these exceptions are important ones.

Important to note is that while the majority of behaviours measured by Marsh et al. (1998a; 1998b) increased in prevalence from 6-months to 1-year, the subjective distress levels did not. Some behaviours appeared to impact more on caregiver burden at 1-year, particularly depression (2.44 and 2.60 at 6-months and 1-year, respectively),

lack of motivation (2.56 and 2.68), dependence (2.60 and 2.69), and anger (2.68 and 2.76). However most behaviours (13 of 20) were reported as being less stressful at 1-year. Argumentativeness (2.69 and 2.33, at 6-months and 1-year, respectively), complaining (2.63 and 2.33), aggression (2.79 and 2.50), being overly sensitive (2.57 and 2.31), poor decision making (2.63 and 2.39), social behaviour (2.68 and 2.54) and childishness (2.38 to 2.26) showed the biggest drop in associated caregiver distress. These findings suggest that some caregivers learn over time to cope with many problem behaviours that are initially found to be stressful. No consistent relationship between behaviour prevalence and subjective distress was found. Many behaviours that were reported as being more prevalent at 1-year, were actually found to be less stress-inducing at this time (e.g., being overly sensitive), while other behaviours showed the opposite pattern (e.g., depression). Future studies in this area would be wise to use a rating scale for measuring behaviour prevalence as well, as the current literature does not reflect potential frequency changes (i.e., so that the effect of a change from ‘displays the behaviour almost constantly’ to ‘occurs once a week’ could be measured).

Over the two years of providing care for BP, it has become apparent that some behaviours provoke more stress in myself than others. In particular, BP’s “over-the-top” behaviour, which can be frustrating and annoying. This is a term used frequently in BP’s home to refer to being overly emotional, or manic, in a particular situation. Often this is displayed in situations where BP feels uncomfortable in a situation, or when he is trying to gain people’s attention (i.e., in an unfamiliar social setting).

This “over-the-top” label could also be applied to the excessive levels of emotion that BP tends to express at various times throughout the day. BP tends to tell people “he loves them” often. Often this expression is inappropriate in either the people it is expressed to, the situation where it is being expressed, or (and this is often the case) the frequency of such expression. One rehabilitation goal with BP

has been to guide his expressions of emotion into more socially appropriate ones. For instance, a goal may be for BP to only say it to me a maximum of once each day. Alternative things to say are also practiced, so that he may replace "I love you" with something more specific, such as "I appreciate the work you are doing for me", or "I like your company". While this over-emotional behaviour is endearing, it is often in drastic contrast to emotions and behaviours being exhibited only minutes or seconds prior. A common example is for BP to call me a derogatory name, pretend to be angry (including clenching his fist), and then putting his arms out as if to hug. These fluctuations in expressions of closeness and inappropriateness can be very taxing, and one would assume such fluctuating behaviours are particular stressful for other caregivers.

My hypothesis regarding which behaviours may be particularly stressful for caregivers are somewhat supported by the results from the studies by Knight, Devereux and Godfrey (1998). While depression was found to cause more distress than any other symptom (2.09 distress rating on a 0-3 scale, 3 indicating severe distress), rapid mood change was the second most distressing symptom (2.05). While BP's constant fluctuations between different emotions is best represented by the construct 'rapid mood change', BP's excessive (and inappropriate) expression of emotions is not really focused on in Knight et al's study. Of the constructs used, 'anger' and 'easily upset' would probably be the closest representations to inappropriate expressions of emotions (although in many cases such expressions of emotion are probably entirely appropriate given the situation that people find themselves in post-TBI). Both of these symptoms were found to be significantly distressing to caregivers (1.98 and 1.91 respectively). Other symptoms of TBI that BP displays appeared to cause moderate distress, including irresponsibility (distress rating of 1.92), impatience (1.82), impulsivity (1.82), loss of interest (1.81), poor insight (1.81), argumentativeness (1.70), over dependence (1.67), poor judgement (1.64), loss of motivation (1.63), childishness (1.60), loss of control (1.59), and loss of initiative (1.53).

The results for the two studies by Marsh et al. (1998a; 1998b) also show some support. Once again, 'mood changes' probably best represents BP's constant fluctuation between expressed emotions. This behaviour was once again one of the most distressful for caregivers, with a subjective distress rating of 2.74 (on a 1-4 scale) at 6-months (only aggression was considered more stressful), and 2.65 at 1-year post-injury (one of the five most distressing symptoms). The addition of

two constructs not investigated by Knight, Devereux and Godfrey (1998) is particularly relevant to the distressing behaviours that BP exhibits. ‘Social behaviour’, certainly relevant given BP’s disregard of social norms when it comes to expression of emotion and social contact, was found to be quite distressing for caregivers at both 6-months (2.68, 6th most stressful behaviour) and 1-year post-injury (2.54, 9th most stressful). The other construct that is best related to the current hypothesis is ‘overly sensitive’. While it is unsure what precisely this construct would entail, it would apply to BP’s over-emotional behaviours when responding to compliments or criticism (e.g., a typical response from BP to praise may be to say ‘I love you’, or ‘f**k you’, or both). Being ‘overly sensitive’, was a common observation in this TBI sample (52% at 6-months and 61% at 1-year post-injury). While caregivers considered this symptom to be reasonably distressing in the short-term (2.57 at 6-months), it became the third-least distressing behaviour at 1-year. It is likely that caregivers become accustomed to the overly sensitive nature of those they are caring for, and become more careful over time the way things are worded around them. It may also be attributed to caregivers learning over time that the heightened sensitivity of the TBI individual is a symptom of their brain injury, and to not take their over-reactions to heart. Either way, being ‘overly sensitive’ does not appear to be a particularly distressing behaviour to deal with in the context of other post-traumatic behavioural symptoms. ‘Anger’, while not being a behaviour that is particularly characteristic of BP, is a construct that might tap into the over-expression of emotion being investigated in this chapter (BP does show moments of anger, but they are always very fleeting). In line with the findings of Knight, Devereux and Godfrey (1998), anger was found to be significantly distressing for caregivers at 6 months post-injury (2.68, one of the 6th highest subjective distress ratings), but was particularly distressful at 1-year (2.76), at which point it was considered to cause more distress than any other TBI-related behaviour. This supports the hypothesis that extreme displays of emotion are a major cause of caregiver burden. Most of the other TBI-related behaviours that BP displays caused moderate distress at both times measured, including dependence (2.60 and 2.69, at 6-months and 1-year post-injury, respectively), lack of interest (2.60 and 2.65), lack of motivation (2.56 and 2.68), impatience (2.57 and 2.61), irresponsibility (2.58 and 2.59), poor insight (2.51 and 2.50), argumentativeness (2.69 and 2.63), poor decision-making (2.63 and 2.39), and complaining (2.63 and 2.33). Childishness (2.38 and 2.26) and lack of initiative (2.30 and 2.24) were not considered as difficult to deal with, while impulsivity was reported as the least distressing TBI symptom of all. This last finding is particularly surprising,

given that Knight, Devereux and Godfrey (1998) found that impulsivity caused much more distress in caregivers.

Finding solutions to caregiver burden

While it is clear that caregiver burden following TBI is a major concern, it is less clear what steps can be taken to lessen the load for caregivers. Given the consistent findings that caregivers may suffer from great stress and burden years on from the injury (and in fact may increase with time), rehabilitation professionals must continue to offer support many years after the initial phase of recovery. This support should be tailored to the specific needs of caregivers, so an open line of communication and a good working relationship should be present in order for carers to feel completely comfortable expressing these particular needs. A study by Moules and Chandler (1999) indicated that different caregivers express different unmet needs with regard to TBI support, and that basing individual caregivers' support needs on ongoing personal needs assessments is probably the most effective means of combating caregiver burden.

Caregivers should be provided with information on the various ways caregivers of people with TBI cope (Nabors, Seacat, & Rosenthal, 2002). Caregivers report having less access to resources for the person with TBI, with the problem increasing as time since injury increases, yet treatment team members report that caregivers are less likely to accept offers of assistance (e.g., monthly support meetings) (Nabors, Seacat & Rosenthal, 2002). This indicates that rehabilitation providers need to be especially persistent in the offering of such assistance, with extra concern placed on the way these services are offered (e.g., so as not to make the caregivers feel they are being pitied, or that the support is unusual, or that the caregivers are being a burden to the health professionals). The finding that many partners don't consider themselves as

'carers', but just as doing their 'job' as a partner, and that they often feel that services for caregivers aren't catering for them, suggests that the rehabilitation services must make it clear that they are also targeting partners (Morris, 2001).

It is apparent that breaks from the caregiver role would be beneficial to a carer's wellbeing. The presence of respite care is crucial, and should be present years on from the injury, in the event that a caregiver feels it is necessary. Not only would it be helpful for a parent or spouse under strain, but rest periods can allow caregivers and loved ones to 'replenish their batteries', and thus provide better quality of care for the person with TBI. While respite care can be a very expensive service, it is an invaluable one, to caregivers, families, rehabilitation providers, and the TBI persons themselves.

There are no easy solutions to the easing of caregiver burden. While it appears that there are many services that can be offered in the long-term for both caregivers and people recovering for TBI, most of these cost considerable amounts of money (a source that is somewhat limited in health fields). While much research has focused on which caregivers face more stress from their role (important in determining what are the main causes of caregiver stress), a new focus of research on what strategies and services have been found to alleviate strain in a high-stress population would be extremely beneficial for rehabilitation professionals.

Resilience

While considerable attention has been given to the adverse effects of TBI on caregivers, it is important to also recognise the resilience of some. For instance, many families do not experience deterioration in functioning. Some families, as a result of

the crisis, appear to draw closer together (Wade, Drotar, Taylor, & Stancin, 1995). A study by Perlesz, Kinsella and Crowe (2000) reported that while many TBI caregivers suffered from caregiver burden of varying degrees several years post-injury, between 51% and 80% of primary caregivers were not psychologically distressed. The percentage of other caregivers and family members to report distress was much lower. More research is needed in the area of resilience to caregiver burden. Identification of the behaviours and characteristics of caregivers who do not become 'stressed out' in the caregiver role, not to mention the characteristics of the TBI patient being cared for by these people, would be invaluable in guiding interventions for those who are suffering from caregiver distress.

A note must be made regarding the resilience of BP's parents. They are BP's main caregivers (primary and secondary by definitions in the literature), and while also finding particular behaviours annoying and frustrating, they cope very well. While much of the research on psychological distress uses clinical levels as a threshold, burden that is at a sub-clinical level may be as relevant to our knowledge of caregiver burden in TBI populations. BP's parents also have many of the factors mentioned above that have been linked to lower levels of burden, including regular respite care, large social networks, reasonably high socio-economic status, and a supportive family environment.

Interestingly, BP's father, when given the Head Injury Behaviour Rating Scale (Smith & Godfrey, 1995) to complete, signalled somewhat different aspects of BP's behaviour as being particularly distressing. In fact, he rated BP's lack of control over his behaviour and social inappropriateness as being the most distressing (a rating of 4 = 'causes severe distress'), followed by a rating of 3 (moderate distress) for impulsivity, lack of interest, lack of initiative, irresponsibility and lack of motivation. Being overly dependent, poor decision making and childishness were all regarded as causing mild distress, while poor insight was regarded as causing distress for BP but causing no distress in BP's father himself. Surprisingly, and inconsistent with my perception of BP's behaviour, rapid mood change was not rated as a being a problem. Discussion with BP's father about this clarified his position: while fluctuating expressions of

mood are a problem and can be distressing, he did not relate this expression as being indicative of changes in underlying mood. This is a position that I concur with. It is possible that looking at the effect of rapid mood change on caregiver burden (consistently found to be one of the most distressing of TBI symptoms) was not an accurate indication of the effect of rapid fluctuations in *expressions of mood*, or *emotional incontinence*. This finding supports the need for inclusion of emotional incontinence as a separate construct in such burden questionnaires. Nevertheless, until it is, levels of distress caused by 'rapid mood change' may be the closest construct we have to infer the effects of rapid 'expressions of mood' change. The differing pattern of distress between myself and BP's father supports the notion that caregiver burden will be experienced differently depending on the relationship of a caregiver with the TBI person. A parent may be more likely to find those aspects of the person's behaviour regarding their future as more distressing (i.e., identified by BP's father's high distress ratings for those behaviours dealing with initiative, interest and motivation). The notion that those behaviours that are found distressing may change over time is also supported (as exemplified by BP's father's comments regarding the severe distress aggression caused at earlier stages of recovery, whereas initiative, motivation, interest and socially inappropriate behaviour were much more distressful in the long term. In fact he did not consider aggression to be a current problem).

Overall, while BP's father reported distress regarding certain behaviour symptoms exhibited by BP, it seems unlikely that any of BP's family would qualify for a clinical diagnosis related to caregiver burden (i.e., major depression or general anxiety disorder). This is based on my subjective viewpoint, and not by clinical measures of subjective distress. I too, have not been measured for clinical levels of distress, but my own viewpoint is that while some behaviours tend to be more stress-inducing than others, my own caregiver burden would fall well below that of clinical significance. While some reasons as to why such resilience may exist in carers are suggested above, the factors that influence resilience (including resilience of other types, e.g., some severe TBI patients recover quickly; some children of psychiatrically disturbed parents display no disturbance) are still largely a mystery.

Conclusion

The literature on caregiver burden tends to give support to the hypothesis mentioned at the start of the present chapter. Behaviour problems post-TBI are frequently cited as being more stressful for caregivers than other changes in the TBI individual (e.g., physical changes, cognitive changes). Social aggression, a behaviour that BP displays (albeit in a joking manner), has also been found to be particular stressful. Of specific behaviours pertaining to the current hypothesis, rapid mood change appears to be a particularly difficult behaviour for caregivers to cope with. This is probably the best indication that fluctuations in extremes of expressed emotion in people following TBI would heighten levels of caregiver burden in these carer populations. Impaired social behaviour (also a characteristic of BP's so-called "stressful behaviours") also results in higher psychological distress in caregivers. While the literature does not sit well with the construct of "over-the-top" behaviour, it at least may be said that caregiver burden research gives moderate support for the current hypothesis. As expressed earlier, qualitative research into caregiver burden may yet demonstrate that these specific behaviours in BP found most stressful, are particularly distressing in other caregivers. Further research into the resilience shown by the majority of caregivers of people with TBI may help to gain an understanding into the characteristics that are necessary to ward off stress, and aid psychologists and health professionals in the easing of caregiver burden in others.

Chapter 6: Conclusion

The current thesis describes several aspects of the life of a person recovering from a severe frontal lobe traumatic brain injury. Several lessons are to be learned from this thesis.

Firstly, rehabilitation for TBI is extremely difficult. It must involve many people from different disciplines with different skills, and with altogether different relationships with the TBI person. Despite having the best possible skills, tools and strategies, and despite maximum input from an array of different rehabilitation professionals, some people do not respond well to rehabilitation. The issue is in fact more complex than this. People with TBI may respond to different types of rehabilitation but not others. Rehabilitation may be successful at stages along the recovery timeline, but not at other times. While some people may demonstrate great recovery in a certain faculty or situation, this improvement may not generalise to other areas of function or to other situations. The hope of this thesis is that it may encourage rehabilitation professionals to keep in mind that varying success is inherent in the nature of TBI rehabilitation, and is not necessarily the result of incorrect or inappropriate implementation of aspects of a rehabilitation plan. It is possible to always find something that could have been done differently in hindsight, but the truth is that we cannot know if this would have made a difference to the TBI outcome. The closest we can come to knowing this is by adjusting the rehabilitation plan, and implementing new strategies.

However, while rehabilitation of different forms is likely to have varying success, this is by no means a reason to disregard the theories and research into TBI rehabilitation that has come before. TBI rehabilitation should be based in theory, and be consistent. But it must also be flexible to changing circumstances and to demonstrated lack of

success. Demonstrated lack of success can only be determined by accurate monitoring of the effectiveness of interventions. For instance, while the behaviours of some people who have suffered a severe TBI may make accurate monitoring difficult, one must always try to gauge as close to accurate an account of problem behaviour frequency and severity as possible. It is only through this monitoring that one can be sure how successful a rehabilitation intervention has been. But rehabilitation, due to the complexities inherent in dealing with complex human beings, must use a somewhat trial-and-error approach. What works for some people may not for others, so once it is clear that an intervention is not working, it is probably best to change tack and try something else. Rehabilitation is a lengthy process, and often must continue many years post-injury to regain maximum independence and/or recovery of function. This is especially true for those with severe head injury. Throughout this lengthy process, many ups and downs are likely. They are a necessary part of the job for a TBI rehabilitation professional.

So what can be done to aid our understanding of what interventions work and for whom? The greatest help will be the proliferation of research into rehabilitation efficacy. Rehabilitation research remains a relatively young field, and is likely to grow rather rapidly in the next decade. Rehabilitation professionals should take note of the latest research, as well as use their own experiences to add to the rehabilitation literature. A focus on which interventions work for whom, and a need for models that focus on functional levels of community integration and behaviour in everyday situations is needed.

This thesis has focused on a few issues pertinent to the life of one person dealing with a severe traumatic brain injury. However, there are a multitude of issues that could

have been discussed instead. For instance, the problem of dealing with alcohol and drug use in young people who have suffered a TBI could have been discussed in much greater depth with reference to BP's experiences. There is a great conflict between social norms (i.e., the behaviours of peers) and the inability for a person with TBI to handle these substances appropriately, and there is much reason for debate about the practical and ethical ways of dealing with this conflict. The problems that BP faces with regard to sexuality (or indeed the problem that any person with a disability may face with regard to not having a sexual partner or an appropriate outlet to channel their sexual energy) is an area that is rich for discussion. The nature of dealing with identity following a severe TBI, particularly of the sort BP has suffered (i.e., with such drastic changes in personality) is also an area that may be useful to explore in order to aid those struggling with identity issues. Every person is a complex human being, so to breaking down a person's behaviours and issues into distinct discrete units will always result in a lot of other (also important) issues being left behind. It is also true that rehabilitation is fluid, and that new problems and difficulties crop up every day. Some issues that may be greatly affecting BP's daily functioning at one point in his recovery process may not be so pertinent at other stages. Rehabilitation providers need to be prepared for the emergence of new problems and obstacles that the future may bring.

It is crucial to realise that while talking about one human being is helpful in that it can give some sort of human face to psychological literature, and hence some functional real world relevance, every person is more than the sum of the parts being discussed. BP is more than some compulsions regarding the number three, or the burden that may be suffered by his caregivers, or the extensive rehabilitative interventions that have been a part of his life over the last three years. He is more than a combination of

these issues, and more than the interaction of these issues. He is a complex human being unable to be deconstructed. While discussion about aspects of a case study will always be beneficial to rehabilitation professionals, it must always be remembered that by taking them out of the context of their situations and the rest of their lives, some essential ‘human-ness’ will inherently be lost in the discussion.

It is also important to keep in mind that BP is not just a cluster of deficits. There are many strengths that BP has, such as the ability to cheer people up and make them laugh, or his incredible memory capacity, particularly regarding remote memory. These must not only be used in the compensation of impaired function, but also cherished for what they are. One must also recognise that while severe traumatic brain injury can be devastating in so many ways and for so many people, some good can be taken from the experience. For instance BP has often talked of gaining ‘a great love for all people on the planet Earth’ since his injury. Many also discover spirituality and faith, a silver lining of hope amidst the myriad of terrible outcomes that are associated with severe TBI.

Much more research is needed in the area of provided functional and everyday accounts of the difficulties for people with TBI and their caregivers. Much can be gained from this real-world individual perspective, and it is hoped that together with large-sampled empirical studies, in-depth case studies can help rehabilitation professionals and TBI sufferers themselves gain a better understanding of their experiences and their road to maximum recovery.

Appendix

The following is the consent form that was used for accessing BP's medical records, signed by BP, BP's father, and myself.

25th November, 2002

I hereby consent that Dion James Blackler may access, read and/or photocopy the medical records of _____, and use them towards his Master of Arts thesis at Massey University (Wellington).

BP (real name used)

signature.....

BP's father (real name used)

signature.....

Dion Blackler

signature.....

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