8. Mental Health Issues Post ABI

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<tr>
<td>ABI</td>
<td>Acquired Brain Injury</td>
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<tr>
<td>BAC</td>
<td>Blood Alcohol Concentration</td>
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<td>BAL</td>
<td>Blood Alcohol Level</td>
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<tr>
<td>CBT</td>
<td>Cognitive Behavioural Therapy</td>
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<td>CSG</td>
<td>Coping Skills Group</td>
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<td>GCS</td>
<td>Glasgow Coma Score</td>
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<tr>
<td>OCD</td>
<td>Obsessive Compulsive Disorder</td>
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<tr>
<td>PEDro</td>
<td>Physiotherapy Evidence Database rating scale</td>
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<tr>
<td>PTSD</td>
<td>Post-Traumatic Stress Disorder</td>
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<tr>
<td>RCT</td>
<td>Randomized Control Trial</td>
</tr>
<tr>
<td>TBI</td>
<td>Traumatic Brain Injury</td>
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</table>
### Key Points

**The effectiveness of sertraline in treating depression post-traumatic brain injury is unclear.**

Citalopram and carbamazepine may be effective in the treatment of mood disorders.

Desipramine may be effective in reducing depression.

Results of various surveys indicate that those who have sustained an acquired brain injury have a higher incidence of depression post injury.

Music therapy may be more efficacious in improving anxiety and depression than standard rehabilitation alone.

Systematic Motivational Counselling may reduce negative affect.

Teaching coping skills to those who have sustained a traumatic brain injury helps to reduce anxiety and depression.

Compassion Focused Therapy may reduce depression and anxiety while improving self-compassion.

Exercise is associated with feeling less depressed and an improved quality of life post-traumatic brain injury.

A mindfulness-based stress reduction programme may be efficacious in reducing depressed mood.

Cognitive Behavioural Therapy and supportive psychotherapy may decrease patient symptoms associated with depression.

There is no difference in reduction of depressive symptoms between Cognitive Behavioural Therapy delivered over the phone or in person.

Positive psychology, involving patients writing down things they enjoy in life, may increase patient happiness.

Cognitive Behavioural Therapy does reduce anxiety and depression post-acquired brain injury.

Although Obsessive Compulsive Disorder has been identified post-acquired brain injury there does not appear to be one method of intervention that works for all, but rather interventions remain individualized.

Little research has been conducted looking at the effects of various interventions on Obsessive Compulsive Disorder post-acquired brain injury.

Little research has been conducted regarding Post-Traumatic Stress Disorder in patients with moderate-severe traumatic brain injury, additional research is needed.
Little research has been conducted regarding treatments for suicide in individuals with moderate or severe traumatic brain injury; further research is warranted.

Amantadine requires further research before conclusions can be drawn on its effects on aggression.

Carbamazepine may decrease agitated behaviour post-traumatic brain injury.

Lamotrigine may be successful in reducing pathologic laughing post-traumatic brain injury. More research is needed, with a greater number of subjects, to validate these findings.

Valproic acid may assist in the reduction of aggressive behaviours; however more research is needed.

Anticonvulsants may be used to decrease the incidence of agitated behaviour; however, more research is needed.

Sertraline HCl may be useful in reducing aggressive and irritable behaviours.

Amitriptyline may be used to decrease agitation.

Pindolol can decrease aggressive behaviour following brain injury.

Propranolol may reduce the intensity of aggressive and agitated symptoms following brain injury.

Although there is evidence to suggest that quetiapine does help reduce aggressive behaviour, more research is needed.

Ziprasidone in one small study has been shown to assist in the controlling of agitation; however more research is needed.

Lithium may reduce behavioural problems but is associated with a high risk of neurotoxicity.

Medroxyprogesterone intramuscularly may reduce sexual aggression.

Methylphenidate may be safe for controlling agitation following an acquired brain injury.

Methylphenidate is effective in reducing anger following a brain injury.

Droperidol may be an effective agent for calming agitated patients.

Haloperidol appears to have little negative effect on recovery following traumatic brain injuries.

There is limited evidence that pharmacological interventions can reduce verbal, physical and/or sexual aggressive behaviours. Rigorous, randomized controlled trials are needed.

Anger self-management training is effective in teaching those with a traumatic brain injury identify anger signals and develop more appropriate ways of dealing with anger and frustration.
Cognitive Behavioural Therapy with focus on anger and aggression management may be effective at reducing aggressive behaviours.

Antecedent management and/or feedback of consequences may reduce undesirable behaviour.

Anger management and social skills training reduce aggressive behaviour.

Music therapy may reduce psychomotor agitation post coma and improve mood following severe traumatic brain injury.

Substance abuse and intoxication at time of injury is a frequent phenomenon in the traumatic brain injury population.

Substance addiction pre injury is predictive of substance addiction post injury.

The impact that blood alcohol levels have on Glasgow Coma Scale, Injury Severity Score, mortality, and long term outcomes has yet to be determined.

Although alcohol and elevated blood alcohol levels have been linked to an increase risk of sustaining a TBI, there is evidence to suggest that elevated blood alcohol levels are not linked to an increase risk of mortality post injury.

The possible neuroprotective role acute alcohol intoxication plays in TBI warrants further investigation.

Earlier studies indicated that elevated blood alcohol levels are associated with poorer performance on a variety of cognitive communication tasks; however, these finding have generally not been supported in most recent studies.

Recent research has found age at injury to be negatively associated with cognitive outcome. More research needs to be conducted investigating the impact of alcohol on cognitive outcomes post injury.

Education and motivational interviewing do not appear to have a strong impact on excessive alcohol consumption post-traumatic brain injury.

Providing financial incentives does encourage those with a traumatic brain injury and a substance addiction to attend treatment more so than offering solutions to other barriers.

Despite their use, there is no evidence to support the use of restraints in those who have sustained an acquired brain injury/traumatic brain injury.

Staff education programs to reduce the use of physical restraints, without increasing the risk of falls, have been shown to be somewhat successful with staff in nursing homes. Further research needs to be completed looking at the impact these education programs would have on those staff working in rehabilitation hospitals.
8. Mental Health Issues Post ABI

8.1 Introduction
Although mood is an internal subjective state, it is often inferred from our posture, behaviours, and the way we choose to express ourselves. Mood disorders such as agitation, major depression, and various anxiety disorders including post-traumatic stress disorder (PTSD) and obsessive compulsive disorder (OCD) may occur following an acquired brain injury (ABI) and are associated with suffering, worsening of other ABI sequela, and poorer outcomes (Bedard et al. 2003; Berthier et al. 2001; Jorge 2005; Jorge & Starkstein 2005). Post ABI, depression is often seen once the implications of the injury become apparent. This may be a reaction to the injury or the result of the neurological changes that have taken place. For some, depression will develop within months of the injury but for others it will be a few years before clinical symptoms are diagnosed (Deb et al. 1999).

Silver et al. (2001) conducted 5034 interviews with individuals who had been diagnosed with a psychiatric disorder; 361 had a severe brain injury. Analysis of their data indicated the most prevalent issues were major alcohol and drug abuse or dependence (34%) and depression (11.1%). These findings are similar to those reported by other researchers (Deb et al. 1999; Hibbard et al. 1998; van Reekum et al. 1996). Individuals who experience depression post ABI may report feeling tired, helpless, hopeless, socially withdrawn, and having difficulty concentrating.

Depression is often accompanied by anxiety and aggressive behaviours. Of note, those who develop aggression early in their recovery are at a higher risk for developing depression, which has been found to impact their length of stay in rehabilitation and their overall recovery (Jean-Bay 2000). Depression can exaggerate the effects of ABI and interfere with progress made during rehabilitation.

PTSD has been largely studied in those who have sustained a mild traumatic brain injury (TBI), particularly in military personnel; however, more recent work has also considered PTSD in the context of moderate and severe injuries. Population-based TBI samples report nearly 18% of individuals post TBI met criteria for PTSD (Barker-Collo et al. 2013). Individuals with co-morbid PTSD and TBI may experience cognitive impairment and sleep disruptions, along with anxiety and depressive symptoms (Barker-Collo et al. 2013).

Suicidal ideation and attempts are also more frequent among the TBI population. Rates of suicidal ideation (23-28%) (Mackelprang et al. 2014; Simpson & Tate 2002; Tsaousides et al. 2011) and attempts (26%) (Simpson & Tate 2005) are high post TBI, but can be further augmented through the presence of emotional disturbance and substance abuse (Simpson & Tate 2005).

Challenging behaviour following a brain injury occurs with a relatively high frequency (25-50%) (Baguley et al. 2006). Challenging behaviours include: non-compliance with treatment, anger, agitation, verbal and/or physical aggression, difficulties with emotional regulation and depression. The emergence of these behaviours likely arises from injury to the frontal lobes (and more specifically the orbitofrontal areas) resulting in disinhibited behaviour and lack of recognition of the consequences of one’s behaviour. Behavioural management and pharmacological techniques are often used to address these challenges. Each has been used with varying levels of success.

Few investigators have examined predictors of aggressive symptoms following brain injury, although it has been suggested that disinhibition and depression may result in aggressive behaviour in some
individuals with brain injury (Backhaus et al. 2010; Kim 2002; Seel et al. 2010). In a sample of 228 patients with moderate to severe brain injury, Baguley et al. (2006) found depression and younger age to be predictors of aggression following brain injury at 6, 24, and 60 months. Severe levels of aggression may be more evident than previously reported, but due to the lack of consistency in how aggression is measured comparing study results may be difficult (Baguley et al. 2006).

Addictive behaviours (alcohol and narcotics abuse and gambling) have been shown to be a serious problem for some individuals both pre and post ABI. Various studies have looked at the incidence of these behaviours and have found that 30 to 60% of individuals who sustain an ABI have a dependence issues (Jorge & Starkstein 2005). Many individuals relapse post injury, often within the first or second year. Alcohol abuse has also been linked to major depression both pre (Dikmen et al. 2004; Seel et al. 2010) and post injury (Jorge & Starkstein 2005), although it remains unclear as to which problem evolved first, the alcohol abuse or the depression. Affective symptoms such as depression and anxiety along with aggression, agitation and addictive behaviours appear to be important determinants of functional and quality of life outcomes. They frequently cause significant distress for individuals with brain injury, their family members, and may result in diminished access to services. This module will review the evidence for both pharmacological and non-pharmacological treatments of depression, anxiety, suicidal ideation, OCD, PTSD aggression and agitation, difficulties in emotional regulation and addictive behaviours post ABI.

8.2 Depression

In Canada, it is estimated that approximately 11% of men and 16% of women will suffer from depression in their life-time (Health Canada 2009). For those who sustain an ABI, depression is the most common mood disorder diagnosed (Jean-Bay 2000; Jorge & Starkstein 2005; Seel et al. 2010; Underhill et al. 2003). It is however, very difficult to diagnose due to the complexities of the brain injury itself (Underhill et al. 2003). Studies have suggested the development of depression may be related to the location of injury, a pre-existing condition, personality type, family support, social support post injury and/or neurochemical imbalances (Jorge & Starkstein 2005; Ownsworth & Oei 1998; Rosenthal et al. 1998). Psychological stressors, being employed pre injury but not post, and older age are also predictors of depression among the ABI population (Sigurdardottir et al. 2013). Complicating the diagnosis is the lack of consistency in the tools used to measure depression post injury (Jorge & Starkstein 2005).

8.2.1 Lesion Location and Depression

Research has investigated the link between the area of brain that has been damaged and the occurrence of depression. Results indicate that those found to have left anterior (dorsolateral frontal or basal ganglia), parietal-occipital, or right hemisphere lesions were more likely to be diagnosed with depression (Fedoroff et al. 1992; Jorge et al. 2004).

8.2.2 Incidence and Prevalence

Studies looking at depression following ABI have noted that depression or depressive symptoms can begin within the first 3 months of injury but may also become evident much later. Depression occurring within the first year has been noted in 18 to 39% of those injured (McKinlay et al. 1981). However, in studies looking at depression in individuals who were one or more years post injury, prevalence rates ranged from 13 to 61% (Fleminger et al. 2003; Gordon et al. 1998; Osborn et al. 2014; Sigurdardottir et al. 2013). The risk for depression is high post ABI and remains this way for decades post injury (Hoffman et al. 2010). More specifically, a meta-analysis conducted by Osborn et al. (2014) report 21-43% of individuals have depression within the first 5 years of TBI and then after 5 years rates stabilized around
22%, which is still high compared to the general population. Further, distinguishing between depression and the behaviours resulting from the injury can prove to be challenging as there is overlap between symptoms. For example, the gradual decline in one’s ability to perform everyday tasks, the ability to cope with everyday stressors, and an increase in irritability and behavioural issues (e.g., anger, frustration, agitation) may be symptoms of depression or brain injury (Fleminger et al. 2003). Additional observational studies looking at depression can be found in the table below.

**Table 8.1 Non-Intervention Studies Examining Depression**

<table>
<thead>
<tr>
<th>Author/Year/Country/Study Design/PEDro/N</th>
<th>Methods</th>
<th>Outcomes</th>
</tr>
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<tbody>
<tr>
<td><strong>Anke et al.</strong> (2015) Norway Observational N=278, N=163</td>
<td>Population: TBI=163; Mean Age=40.1yr; Gender: Male=127, Female=36; Mean GCS=5.7. Intervention: Questionnaires administered at admission, 3mo and 12mo post-injury. Outcome Measure: Hospital Anxiety and Depression Scale (HADS), Life Satisfaction Questionnaire.</td>
<td>1. 20% of participants showed signs of anxiety whereas 19% had depressive symptoms 12mo post-TBI. 2. At 12mo, lack of functional recovery is associated with increased anxiety (p=0.006) and depression (p=0.001) compared to individuals who recovered, according to HADS. 3. Individuals had lower life satisfaction at 12mo when they had higher levels of anxiety (p=0.014) or depression (p&lt;0.001).</td>
</tr>
<tr>
<td><strong>Juengst et al.</strong> (2014) USA Observational N=64</td>
<td>Population: TBI; Mean Age=46yr; Gender: Male=51, Female=13; Mean Time Post Injury=50mo. Intervention: Questionnaires administered. Outcome Measure: Mayo Portland Adaptability Inventory, Primary Care Evaluation of Mental Disorders, Positive and Negative Affect Schedule.</td>
<td>1. Participants with no depression reported higher positive affect than those with depression (p&lt;0.05). 2. Participants with no depression reported significantly lower negative affect than either those with a history of depressive episode or those with a current depressive episode (p&lt;0.001).</td>
</tr>
<tr>
<td><strong>Sigurdardottir et al.</strong> (2013) Norway Observational N=118</td>
<td>Population: TBI; Mean Age=32.5yr; Gender: Male=84, Female=34. Intervention: Questionnaires administered over time (3mo, 1yr, 5yr post injury). Outcome Measure: Semi-structured psychological interview, Hospital Anxiety and Depression Scale (HADS), Symptom Checklist 90-Revised (SCL-90-R), Fatigue Severity Scale (FSS), Visual Analogue Scale for Pain (VAS-P).</td>
<td>1. The prevalence of depressive symptoms was 18% at 3mo, 13% at 1yr and 18% at 5yr after injury. 2. 4% of patients had persistent depressive symptoms at all time-points. 3. Association was found between HADS-Depression and SCL-90-R Anxiety scores (p&lt;0.001). 4. A high number of ongoing psychological stressors, older age, being employed pre injury and being unemployed post injury were main predictors of depression and accounted for 43% of variance (p&lt;0.001). 5. Individuals with high levels of depressive symptoms at 1yr had significantly more pre-existing psychological stressors than those with low levels (p&lt;0.001). 6. FSS and VAS-pain scores did not significantly differ over time.</td>
</tr>
<tr>
<td><strong>Spitz et al.</strong> (2013) Australia Observational N=97</td>
<td>Population: TBI; Mean Age=35.8yr; Injury Severity: Mild=6, Moderate=17, Severe=69; Mean Time Post Injury=19mo. Intervention: Questionnaires administered. Outcome Measure: BIRT Memory and Information Processing Battery- List Learning</td>
<td>1. 73% of patients scored in the normal range for the HADS-Depression subscale while 65% scored normal for the HADS-Anxiety subscale. 2. Less frequent use of adaptive coping strategies (p&lt;0.01) and greater use of...</td>
</tr>
<tr>
<td>Study</td>
<td>Country</td>
<td>Study Design</td>
</tr>
<tr>
<td>-------</td>
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</tr>
<tr>
<td>Bay et al. (2012)</td>
<td>USA</td>
<td>Observational</td>
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<tr>
<td>Hudak et al. (2012)</td>
<td>USA</td>
<td>Observational</td>
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<tr>
<td>Arango-Lasprilla et al. (2012)</td>
<td>USA</td>
<td>Observational</td>
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<tr>
<td>Alway et al. (2012)</td>
<td>Australia</td>
<td>Case Control</td>
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### Intervention: Questionnaires administered to patients and a family member.

**Outcome Measure:** Hospital Anxiety and Depression Scale (HADS), The Family Questionnaire (FQ): Criticism Subscale (CS) and Emotional Over-involvement subscale (EOI), and the Illness Perception Questionnaire Revised- Patient control subscale.

1. For family members, based on the HADS, 28.5% scored mild for anxiety, 19.1% moderate and 2.4% severe. 19% reported mild and 2.4% moderate depressive symptoms.
2. On the FQ-EOI, 83.7% of family members reported low levels of emotional involvement while the remaining had emotional over-involvement.
3. There was a moderate positive relationship between family member FQ-CS, and patient HADS-A and HADS-D (p<0.05).
4. There was a moderate positive correlation between family member FQ-EOI and patient HADS-D (p<0.01) and HADS-A (p<0.05).
5. Family member HADS-A was moderately positively associated with FQ-CS and strongly positively correlated with FQ-EOI (p<0.05).
6. Family member HADS-D was moderately positively associated with FQ-CS (p<0.05) and strongly positively associated with FQ-EOI (p<0.01).

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### Diaz et al. (2012)
Brazil
Observational
N<sub>Initial</sub>=48, N<sub>Final</sub>=33

Population: TBI; Mean Age=32.3yr; Gender: Male=42, Female=6; Mean Time Post Injury=18mo.

**Intervention:** Assessments completed.

**Outcome Measure:** Structured Clinical Interview for DSM-IV, Hospital Anxiety and Depression Scale (HADS), Brief Psychiatric Rating Scale (BPRS), Apathy Evaluation Scale and Short Form Health Survey (SF-36).

1. Significant increase in the rate of major depressive disorder (MDD; p=0.02) and generalized anxiety disorder (p=0.02) after severe TBI occurred.
2. Significant decrease in rates of alcohol and cannabinoid abuse were observed after injury (p=0.001).
3. Comparing those with and without MDD, apathy scores did not differ between groups (p>0.05); however, patients with MDD scored lower on the HADS and BPRS (p<0.01).
4. Patients with current MDD showed impairment in all SF-36 domains (p<0.02).

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### Malec et al. (2007)
USA
Observational
N=135

Population: Gender: Male=77, Female=58; Mean Time Post Injury=47.1d. Mild TBI Group (n=42): Mean Age=35.8yr. Mod/Severe TBI Group (n=51): Mean Age=35.7yr. Ortho Injury Group (n=42): Mean Age=42.5yr.

**Intervention:** Questionnaires completed.

**Outcome Measure:** Neurobehavioural functioning inventory (NFI), Multidimensional scale of perceived social support, and Family assessment device.

1. No significant differences between injury groups were found on the NFI-depression subscale (p>0.05).
2. For the TBI sample only, patients discharge NFI-Impairment was strongly associated with their discharge NFI-dep (72% of variance, p<0.0001).

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### Deb & Burns (2007)
United Kingdom
Observational
N=165

Population: TBI; Gender: Male=111, Female=54. Group A (n=120): Mean Age=35.5yr. Group B (n=45): Mean Age=79.2yr.

**Intervention:** Questionnaire/interview 1yr post injury. Patients divided into two groups by age (Group A=18-65yr, Group B= >65yr).

**Outcome Measure:** Clinical Interview Schedule-Revised (CIS-R), General Health Questionnaire

1. Those in the younger group were found to have a higher incidence of psychiatric morbidity and depressive symptoms than those in the older age group based on GHQ-28 and CIS-R (p<0.01).
2. Low MMSE scores were associated with abnormal ERSS scores in younger but not older patients (p<0.001).

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**http://www.abiebr.com**
Updated August 2016
### Hibbard et al. (2004) USA Survey-Interviews N=189

**Population:** TBI; Gender: Male=100, Female=89.  
No Depression Group (n=91): Mean Age=43.8yr; Mean Time Post Injury=2.51yr.  
Resolved Depression Group (n=52): Mean Age=43.1yr; Mean Time Post Injury=2.63yr.  
Late-Onset Depression Group (n=19): Mean Age=38.5yr; Mean Time Post Injury=2.52yr.  
Chronic Depression Group (n=27): Mean Age=44.8yr; Mean Time Post Injury=2.16yr.  
**Intervention:** Two interviews were conducted 12mo apart.  
**Outcome Measure:** Structured Clinical Interview for DSM-IV, Beck Depression Inventory (BDI), Living life after TBI, Unmet important needs, Impact of TBI on Roles and Responsibilities Scale, Life-3.

1. 56% of sample had a DSM-IV diagnosis prior to their TBI.  
2. 17% of those defined as having a mood disorder were diagnosed with major depression post injury; 35% of those without a previous mood disorder were diagnosed with major depression after injury.  
3. Individuals in the chronic- depression group showed no improvement on the various psychosocial scales between time periods.

### Kreutzer et al. (2001) USA Survey N=722

**Population:** TBI; Mean Age=36.0yr; Gender: Male=462, Female=261; Mean Time Post Injury=2.5yr.  
**Intervention:** Patients completed an assessment; missing information was gathered via interviews.  
**Outcome Measure:** Diagnostic and Statistical Manual of Mental Disorders (DMS-IV), Neurobehavioural Functioning Inventory.

1. 42% met the prerequisite for a Major Depressive Episode diagnosis.  
2. Results using DSM-IV classification indicate that 85% of the individuals reported feeling frustrated, with 41% feeling frustrated quite often or all of the time.  
3. Problems such as increased feelings of hopelessness, sadness, worthlessness, increased tiredness, aggression and the inability to concentrate were experienced by 19% to 60% of participants.

### 8.2.3 Pharmacological Interventions for Depressions

Post ABI, depression is often treated pharmacologically. Included among these Interventions are various antidepressants: serotonin selective re-uptake inhibitors such as paroxetine, sertraline, or citalopram; serotonin norepinephrine reuptake inhibitors such as duloxetine; and tricyclic antidepressants such as amitriptyline. The use of tricyclic antidepressants is however, often restricted to the treatment of headaches in those who have sustained a mild TBI because their side effects (memory impairment, sedation, etc.) have proven to be problematic in individuals who have sustained more a moderate or severe brain injury (Bajo et al. 1999). Anticonvulsants such as carbamazepine have also been used to treat depression post ABI.
### Table 8.2 Pharmacological Interventions used to Treat Depression Post ABI

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<thead>
<tr>
<th>Author/Year/Country/Study Design/PEDro Score</th>
<th>Methods</th>
<th>Outcomes</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Rapoport et al. (2010)</strong> Canada RCT PEDro=9 N&lt;sub&gt;initial&lt;/sub&gt;=21, N&lt;sub&gt;final&lt;/sub&gt;=18</td>
<td>Population: TBI; Mean Age=47.67yr; Gender: Male=11, Female=10; Injury Severity: Mild=16, Moderate/Severe=5. Treatment Group: Mean Time Post Injury=105d. Control Group: Mean Time Post Injury=107d. Intervention: Individuals who had a DSM-IV diagnosis of major depression but met the criteria for remission were assigned to either the treatment group (n=10) who were given citalopram (~40mg/d) or the control group (n=11) which received a placebo for 40wk. Outcome Measure: Cumulative Illness Rating Scale, Hamilton Depression Rating Scale (HDRS), Mini Mental State Examination and the Rivermead Post Concussion Symptoms Questionnaire.</td>
<td>1. Comparing the treatment and control groups, relapse rates (p=0.835) and time to relapse (24.8 versus 22.3wk, respectively, p=0.700) were not significantly different. 2. All participants experienced adverse events regardless of the group they were placed in (e.g. headache, muscle/joint pain, and dizziness). 3. On the HDRS, patients with “more than mild agitation” relapsed sooner than those without that level of agitation (8.0 versus 27.18wk, p=0.013). 4. On the HDRS, those with “more than mild psychic anxiety” relapsed at a mean of 19.7wk compared to those with “none to mild” who did not relapse (p=0.046).</td>
</tr>
<tr>
<td><strong>Ashman et al. (2009)</strong> USA RCT PEDro=10 N=41</td>
<td>Population: TBI; Mean Age=49.1yr; Gender: Male=24, Female=17; Mean Time Post Injury=17.7mo; Injury Severity: Mild=15, Moderate=16, Severe=10. Intervention: The treatment group (n=22) was given sertraline (25mg adjusted every 2wk, range 25-100mg) and the control (n=19) received a placebo for 10wk. Outcome Measure: Structured Clinical Interview for DSM-IV Axis I Disorders, Hamilton Rating Scale for Depression (HAM-D), Beck Anxiety Inventory (BAI), and Life-3 scale (QOL).</td>
<td>1. Treatment responders, based on HAM-D (score &lt; 10 or decreased by 50%) were 59% in the treatment group and 32% in the control (p=0.08). 2. Changes in scores on the HAM-D, the BAI and the QOL scales did show improvement (p&lt;0.001) but no group effects were found.</td>
</tr>
<tr>
<td><strong>Rapoport et al. (2008)</strong> Canada Prospective Controlled Trial N&lt;sub&gt;initial&lt;/sub&gt;=65, N&lt;sub&gt;final&lt;/sub&gt;=54</td>
<td>Population: TBI; Mean Age=39.7yr; Gender: Male=38, Female=27; Injury Severity: Mild=33, Moderate to Severe=32. Intervention: Group A (n=29) received 20mg/d of citalopram for 6wk whereas group B (n=36) received 20mg titrated to 50mg/d for 10wk. Outcome Measure: The Hamilton Rating Scale for Depression (HAM-D), Clinical Global Impression, and the Rivermead Post Concussion Symptoms Questionnaire (RPQ).</td>
<td>1. Mean HAM-D scores decreased from baseline to 6wk (23.66 versus 16.30, p&lt;0.0001). Scores also decreased significantly from baseline to 10wk (12.96, p&lt;0.001). 2. 84.6% reported ≥1 adverse event; most often, dry mouth. 3. Of the 54 subjects who started the study, 24.1% were in remission at 6wk. Of the 26 assessed, 26.9% were in remission at 10wk. 4. The somatic score on the RPQ decreased significantly from 15.38 to 11.35 (p=0.0003) at 6wk; but not at 10wk (10.82, p=0.0632).</td>
</tr>
<tr>
<td><strong>Lee et al. (2005)</strong> Korea RCT PEDro=8 N=30</td>
<td>Population: TBI; Gender: Male=24, Female=6. Group A (n=10): Mean Age=35.3yr; Mean Time Post Injury=34.8d. Group B (n=10): Mean Age=33.6yr; Mean Time Post Injury=31.9d. Group C (n=10): Mean Age=35.5yr; Time Post Injury=30d.</td>
<td>1. In all 3 groups scores on the HAM-D and BDI improved from the baseline and week 4 (Group A, p&lt;0.001 on both measures; Group B, p&lt;0.01, for both; Group C, p&lt;0.05 BDI and p&lt;0.01 for HAM-D).</td>
</tr>
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</table>
Intervention: Patients assigned to one of three groups: Group A: methylphenidate (5mg/d increased to 20mg/d); Group B: sertraline group (25mg/d increased to 100mg/d); or Group C: placebo.

Outcome Measure: Beck Depression Inventory (BDI) and the Hamilton Rating Scale for Depression (HAM-D).

2. Groups A (p=0.005) and B (p=0.05) were significantly superior to Group C on the HAM-D.

3. The number of adverse events was higher in Group B than Group A (13 versus 6, p=0.010).

Perino et al. (2001)

Population: TBI; Gender: Male=11, Female=9.
Group A (n=11): Mean Age=26.9yr; Mean GCS Score=5.5; Mean Time Post Injury=4.7mo.
Group B (n=9): Mean Age=31.3yr; Mean GCS Score=6.1; Mean Time Post Injury=34.6mo.

Intervention: Patients received citalopram (20mg/d) and carbamazepine (600mg/d), and were divided into subgroups based on time post injury (Group A, <6mo; Group B, 24-36mo).

Outcome Measure: Psychiatric Rating Scale (BPRS) and the Clinical Global Impression (CGI).

1. The total sample significantly improved from baseline to 12 weeks on the BPRS (62.3±17.6 versus 51.7±12.8, p≤0.05) and CGI (4.4±1.1 versus 3.4±0.8, p≤0.005).

Wroblewski et al. (1996)

Population: TBI; Mean Age=32.2yr; Gender: Male=7, Female=3; Mean Time Post Injury=1.5yr; Injury Severity=Severe.

Intervention: The treatment group (n=6) received desipramine (150mg/d for 30d, 150-300mg/d after) and the control group (n=4) received a placebo. The control group crossed over and received desipramine after day 30.

Outcome Measure: Diagnostic and Statistical Manual of Mental Disorders checklist and Affect/Mood Scale.

1. 3 individuals from each group had nearly complete resolution of depression on desipramine.
2. 70% of subjects showed improvement over time on the affect/mood scale (p=0.001).
3. There were different rates of improvement over time in those started on the desipramine rather than placebo; with the treatment group making more rapid and greater improvements.

PEDro=Physiotherapy Evidence Database rating scale score (Moseley et al. 2002).

Discussion

Some research has been conducted exploring the effects of sertraline, citalopram, carbamazepine, desipramine and methylphenidate on the treatment of depression or depressive symptoms post ABI. A single, small sample Randomized Control Trial (RCT) found that desipramine was effective in treating long-standing depression (Wroblewski et al. 1996). Three of those in the treatment group and three in the control group had near complete resolution of depression; however, additional studies are necessary before firm conclusions are drawn on this medication.

Two RCTs looked at the effects of sertraline on depression post ABI (Ashman et al. 2009; Lee et al. 2005). Ashman et al. (2009) compared sertraline and a placebo and found improvements over time for both groups on all three outcomes (the Hamilton Rating Scale for Depression, the Beck Anxiety Inventory (BDI), and the Life-3 quality of Life scales). No statistically significant differences were shown between the two groups; therefore the changes may not have been related to sertraline. The second RCT added a third arm to their trial. The authors randomized individuals with mild or moderate TBI to a sertraline, methylphenidate or placebo group (Lee et al. 2005). Similar to the first study, all participants improved on the depression measures (BDI and Hamilton Rating Scale for Depression); however, the study results indicated that those assigned to the sertraline and the methylphenidate groups reported significantly less depressive symptoms on these measures than the placebo group at the end of the study (Lee et al. 2005). Further, fewer adverse events were reported for individuals receiving, methylphenidate than those administered sertraline.
The remaining three studies looked at citalopram. Rapoport and colleagues (2008) administered 20 mg/day of citalopram for 6 weeks to one group while the second group began with 20 mg/day which was titrated to a maximum of 50 mg/day. The second group was studied for 10 weeks. For participants in both groups, their depression scores significantly decreased compared to baseline. In another study participants were randomly assigned to receive citalopram or placebo (Rapoport et al. 2010). Post-treatment relapse rates were calculated for each group and there were no significant differences noted between the groups with individuals relapsing (meeting criteria for major depressive disorder) 22 to 24 weeks post treatment; relapse occurred in 52.4% of patients. In both studies, adverse events were common (Rapoport et al. 2008; Rapoport et al. 2010). While citalopram on its own has shown potential to aid with depression, a study by (Perino et al. 2001) found that when citalopram and carbamazepine were given to patients post TBI diagnosed with depression, after 12 weeks, scores on the Brief Psychiatric Rating Scale and the Clinical Global Impression were significantly improved.

Conclusions

- There is conflicting evidence that sertraline is effective in the treatment of major depression post-traumatic brain injury.

- There is Level 2 evidence that citalopram aids in the reduction of depression post-acquired brain injury.

- There is Level 4 evidence that citalopram and carbamazepine may be efficacious in the treatment of depression, anxiety and mood disorders.

- There is Level 2 evidence to suggest that the administration of desipramine assists in improving mood and reducing depression.

8.2.4 Non-Pharmacological Interventions for Depression

Several non-pharmacological interventions have been used to treat depression post ABI including: exercise, team sports, providing them with supports through an interdisciplinary team, or counselling (Knottnerus et al. 2007). Various interventions are summarized in this section.
### Individual Studies

#### Table 8.3 Non-Pharmacological Interventions used to Treat Depression Post ABI

<table>
<thead>
<tr>
<th>Author/Year/ Country/Study Design/PEDro/N</th>
<th>Population</th>
<th>Methods</th>
<th>Outcomes</th>
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<tbody>
<tr>
<td><strong>Bedard et al.</strong> (2014) Canada RCT Pedro=6 N=76</td>
<td>TBI; Gender: Male=42, Female=34; Mean Age=46.5yr. Treatment Group (n=38); Mean Time Post Injury=4.5yr. Control Group (n=38); Mean Time Post Injury=4yr.</td>
<td>Intervention: The treatment group received 10wk of mindfulness-based cognitive therapy with 1.5hr weekly sessions. The control group received regular care. Outcome Measure: Beck Depression Inventory-II (BDI-II), Patient Health Questionnaire-9 (PHQ-9), Symptom Checklist-90-Revised (SCL-90-R). Philadelphia Mindfulness Scale (PHLMS) and Toronto Mindfulness Scale (TMS) completed by intervention group only.</td>
<td>1. Following treatment, those in the treatment group showed significantly greater reduction in BDI-II scores than the control group (p=0.029) which was maintained at the 3mo follow-up.</td>
</tr>
<tr>
<td><strong>Bedard et al.</strong> (2012) Canada Pre-Post Test N=20</td>
<td>TBI; Mean Age=47.1yr; Gender: Male=9, Female=11; Time Post Injury ≥1yr. Treatment: A 90min session of mindfulness-based stress reduction therapy weekly for 8wk. Sessions included topics such as staying in the present, acceptance, and improving awareness of thoughts and feelings. Homework assignments were given after each session. Outcome Measure: Beck Depression Inventory-II (BDI-II), Hospital Anxiety and Depression Scale (HADS), Patient Health Questionnaire-9 (PHQ-9).</td>
<td></td>
<td>2. No significant improvements in PHQ-9 scores and SCL-90-R were found (p&gt;0.05). 3. Neither the PHLMS nor TMS reached statistical significance in demonstrating an increase in mindfulness for the intervention group (p&gt;0.05).</td>
</tr>
<tr>
<td><strong>Bedard et al.</strong> (2003) Canada Pre-Post N=13</td>
<td>TBI; Treatment Group (n=10): Mean Age=43yr; Gender: Male=3, Female=7. Control Group (n=3); Mean Age=39yr; Gender: Male=3, Female=0. Intervention: 12 weekly group sessions of a mindfulness-based stress reduction for the treatment group (n=10). Drop-outs served as the control group (n=3). Outcome Measure: Beck Depression Inventory (BDI-II), Positive Symptom Distress Index (PSDI), Short Form Health Survey (SF-36).</td>
<td></td>
<td>1. Following the intervention, a significant reduction in depression and anxiety symptoms was shown based on the change in scores on the BDI (p=0.001), PHQ-9 (p=0.003) and HADS-Depression subscale (p=0.023). 2. The anxiety subscale of the HADS scale did not change significantly pre to post intervention (11.1 versus 9.8, p=0.116).</td>
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</table>

#### Exercise Programs

<table>
<thead>
<tr>
<th>Author/Year/ Country/Study Design/PEDro/N</th>
<th>Population</th>
<th>Methods</th>
<th>Outcomes</th>
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<tbody>
<tr>
<td><strong>Damiano et al.</strong> (2016) USA Prospective Controlled Trial Ninitial=31, Nfinal=24</td>
<td>TBI=12, Healthy Volunteers (HV)=12. TBI Group (n=12): Mean Age=31.3yr; Gender: Male=7, Female=5. HV (n=12): Mean Age=32.5yr; Gender: Male=7, Female=5. Intervention: All participants completed an 8wk aerobic home based exercise program for 5d/wk. Training included exercise of moderate intensity for 30min on the elliptical. Compliance was monitored by a sensor. Assessment of motor and emotional function at baseline, post-treatment, and 8wk follow-up.</td>
<td></td>
<td>1. No significant changes in depression or anxiety scores from baseline to post treatment or follow-up were found. 2. Walking on the elliptical at a slower speed was associated with higher depression scores on HAM-D (p=0.03) whereas large excursion movements to the right was associated with less depressive symptoms (p=0.04).</td>
</tr>
<tr>
<td>Evidence-Based Review of Moderate to Severe Acquired Brain Injury</td>
<td>2017</td>
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</table>

**Outcome Measure:** Limits of Stability (LOS) test, Hamilton Depression Inventory (HAM-D), Beck Anxiety Inventory (BAI), Pittsburg Sleep Quality Index (PSQI).

**Weinstein et al.** (2016)

USA
Pre-Post
N<sub>Initial</sub>=12, N<sub>Final</sub>=10

**Population:** TBI; Mean Age=32.9yr; Gender: Male=4; Female=6; Mean Time Post Injury=6.6yr; Severity: Mild=5, Moderate=4, Severe=1.

**Intervention:** Participants completed one-on-one supervised aerobic exercise sessions (3d/wk for 12wk) where they reached 70-80% of maximum heart rate. Total Mood Disturbance (TMD) assessed before and after sessions at baseline, 4, 8 and 12wk.

**Outcome Measure:** Profile of Mood States-Short Form (POMS-SF).

1. Significant improvement in TMD from baseline to 12 weeks, as 80% of participants reported less mood disturbances on POMS-SF (p=0.04).
2. Marked 19% reduction in TMD from baseline to 12 weeks (p=0.04).
3. Significant short-term changes in TMD in response to singular exercise sessions as measured by POMS-SF, with the most substantial change in fatigue inertia (p=0.01) and anger hostility (p=0.09).

**Bellon et al.** (2015)

USA
RCT-Crossover
PEDro=6
N<sub>Initial</sub>=123, N<sub>Final</sub>=69

**Population:** TBI; Mean Age=43.7yr; Gender: Male=41, Female=28; Mean Time Post Injury=100.5mo; Severity: Mild=10, Moderate=10, Severe=35.

**Intervention:** Participants were randomized into a walking group (treatment) or nutrition group (control). The home-based walking group was administered a pedometer to track steps taken per wk for 12wk, with a coaching call 3d/wk to encourage increase in weekly step average. The home-based nutrition group learned about healthy eating habits through coaching calls 3d/wk for 12wk. After 12wk participants crossed over. Depression and anxiety measures assessed at baseline, after first 12wk, and post-treatment.

**Outcome Measure:** Centre for Epidemiological Studies-Depression (CES-D), Perceived Stress Scale (PSS).

1. Depression rates decreased from baseline to post-treatment for all participants, with a significant reduction noted at 12wk for those with a diagnosis of mild depression, according to CES-D (p=0.007).
2. Order of treatment did not make a difference for symptoms of depression by CES-D.
3. Stress decreased overall from baseline to post-treatment as measured by PSS (p=0.006), with a greater decrease in stress when participants were in the walking group (p=0.006).

**Wise et al.** (2012)

USA
Follow-up to Hoffman et al. 2010
RCT
N=40

**Population:** TBI; Mean Age=39.7yr; Gender: Male=15, Female=25; Mean Time Post Injury=2.1yr.

**Intervention:** 10wk exercise program, consisting of 1hr sessions in the gym. In addition, 4-30min sessions per week at home.

**Outcome Measure:** Beck Depression Inventory (BDI), Perceived Quality of Life Scale (PQOL), Short Form Health Survey (SF-12).

1. At the 6mo follow-up there was a reduction in the number of participants who were able to exercise >90 minutes per week (77% versus 52%).
2. Those who exercised more than 90 minutes per week, compared to those doing less than 90 minutes, had lower BDI scores (p=0.037), and felt they had a higher quality of life (PQOL; p=0.014) and better mental health (SF-12; p=0.014).

**Hoffman et al.** (2010)

USA
RCT
PEDro=5
N=80

**Population:** TBI; Treatment Group (n=40): Mean Age=39.7yr; Gender: Male=15, Females=25. Control Group (n=40): Mean Age=37.1yr; Gender: Male=20, Female=20.

**Intervention:** 10wk exercise program with 30min of aerobic exercises, 30min of warm up exercises, 15min of warm up exercises, 15min of cool down exercises. Each participant was asked to perform 30min sessions 4×/week while in the program. The control group was given the

1. There were no significant differences between the exercise and control groups post intervention on the BDI (p=0.250).
2. The treatment group reported greater improvements on the BPI compared to controls (p=0.030).
3. No differences in sleep quality, general health status, heart rate or ability to walk were noted between the groups.
<table>
<thead>
<tr>
<th>Source</th>
<th>Country</th>
<th>Study Design</th>
<th>PEDro</th>
<th>N</th>
<th>Population</th>
<th>Intervention</th>
<th>Outcome Measure</th>
<th>Results</th>
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<tbody>
<tr>
<td>Blake &amp; Batson (2009)</td>
<td>UK RCT</td>
<td>PEDro=6</td>
<td>20</td>
<td></td>
<td>TBI; Gender: Male=15, Female=5; Injury Severity: Mild=7, Moderate=8, Severe=5.</td>
<td>Treatment Group (n=10): Treatment Group (n=10): Mean Age=44.5yr; Mean Time Post Injury=16.4yr. Control Group (n=10): Mean Age=46.2yr; Mean Time Post Injury=13.62yr.</td>
<td>Beck Depression Inventory (BDI), Brief Pain Inventory (BPI).</td>
<td>1. At 8wk follow-up, results of the GHQ-12 showed a significant decrease in mood scores for those in the TBI group compared to the control group (p=0.026). 2. Physical self-esteem was found to improve significantly from baseline to follow-up for those in the Qigong group (p=0.017).</td>
</tr>
<tr>
<td>Driver &amp; Ede (2009)</td>
<td>USA RCT</td>
<td>PEDro=5</td>
<td>16</td>
<td></td>
<td>TBI; Treatment Group (n=8): Mean Age=38.78yr; Mean Time Post Injury=40.75mo.</td>
<td>Intervention: Participants randomly assigned to the treatment group received Qigong instruction for an hour a week. The control group attended non-exercise social and leisure activities for 1hr/wk for 8wk.</td>
<td>General Health Questionnaire-12 (GHQ-12), Physical Self-Description Questionnaire, Social Support for Exercise Habits Scale.</td>
<td>1. Significant differences in total POMS scores were noted between the groups post intervention (p&lt;0.05) in favour of the treatment group. 2. Within group scores for the treatment group, pre to post, showed significant differences on each of the sub-scales of the POMS (depression, anger, vigour, fatigue, confusion and friendliness; p&lt;0.05). 3. No significant differences were noted on each of the sub-scales for the control group.</td>
</tr>
<tr>
<td>Gemmell &amp; Leathem (2006)</td>
<td>New Zealand RCT</td>
<td>PEDro=6</td>
<td>18</td>
<td></td>
<td>TBI; Treatment Group (n=9): Mean Age=51.2yr, Females=40.2yr; Gender: Male=9, Female=9; Mean Time Post Injury=8.7yr.</td>
<td>Intervention: Participants randomly assigned to aquatic physical activity intervention (24 sessions for 8wk) or the control group which attended a vocational rehabilitation class for the same number of sessions.</td>
<td>Profile of Mood States (POMS).</td>
<td>1. Participants exercising &gt;90min/wk were found to have lower depression scores than those exercising &lt;90min/wk (p=0.033).</td>
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<tr>
<td>Gordon et al. (1998)</td>
<td>USA Case Series</td>
<td></td>
<td>379</td>
<td></td>
<td>Population: TBI=240; Healthy Adults=139; Gender: Male=245; Female=134. TBI Non-Exercise Group (n=176): Mean Age=37.1yr; Mean Time Post Injury=9.1yr. TBI Exercise Group (n=64): Mean Age=37.8yr; Mean Time Post Injury=11.2yr. Without TBI Non-Exercise Group (n=73) and without TBI Exercise Group (n=66).</td>
<td>Intervention: Participants randomly assigned to the treatment group received Qigong instruction for an hour a week. The control group attended non-exercise social and leisure activities for 1hr/wk for 8wk.</td>
<td>Beck Depression Inventory, The Institute for Rehabilitation Research (TIRR) Symptom Checklist, SF-36 Health Survey,</td>
<td>1. Results of TBI groups only. Patients with TBI who exercised reported significantly fewer symptoms on the TIRR checklist compared to patients with TBI who did not exercise (p&lt;0.0004). 2. Participants with TBI who exercised had less depressed mood than participants with TBI who did not exercise (p&lt;0.01). 3. Exercise intervention had no effect on community participation or handicap.</td>
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</table>
### Teaching of Coping Skills

<table>
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<tr>
<th>Study</th>
<th>Population</th>
<th>Intervention</th>
<th>Outcome Measure</th>
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<tbody>
<tr>
<td>Anson &amp; Ponsford (2006b)</td>
<td>TBI; Mean Age=36.7yr; Gender: Male=27, Female=6; Mean GCS Score=9; Mean Time Post Injury=516.8d. Group A (n=17), receiving 10wk of Coping Skills Group (CSG) intervention or Group B (n=16), receiving 5wk of the same intervention (90min sessions, 2×/wk).</td>
<td>1. Those who had a greater self-awareness following their injuries had demonstrated better outcomes post CSG intervention, noted in the decrease in depression scores. 2. Those who had poorer self-awareness had higher scores on the depression scales and did not perform as well following CSG intervention.</td>
<td>Community Integration Questionnaire, Craig Handicap Assessment Capacity Technique. Teaching of Coping Skills</td>
</tr>
<tr>
<td>Anson &amp; Ponsford (2006a)</td>
<td>TBI; Gender: Male=26, Female=5. Group A (n=15): Mean Age=38.9yr; Mean Time Post Injury=755.8d. Group B (n=16): Mean Age=37.8yr; Mean Time Post Injury=340.8d. Intervention: For Group A (n=15), baseline phase was 5wk, followed by 5wk of intervention, and a 5wk follow-up phase. For Group B (n=16), baseline was 10wk, followed by 5wk of intervention and a 10wk follow-up phase. The CSG consisted of 10 group sessions and ran for 900min 2×/wk.</td>
<td>1. No significant changes in anxiety or self-esteem scores were noted following the CSG (p&gt;0.05). 2. Although levels of depression and psychosocial dysfunction were significantly different between the two groups (p&lt;0.05) participation in the CSG did not have an effect on their scores. 3. Both groups significantly increased their adaptive coping skills following the CSG (p&lt;0.01).</td>
<td>Coping Scale for Adults, Hospital Anxiety and Depression Scale, Rosenberg Self Esteem scale.</td>
</tr>
<tr>
<td>Ruff &amp; Niemann (1990)</td>
<td>TBI; Gender: Male=17, Female=7. Experimental Group (n=12): Mean Age=28.3yr; Mean Time Post Injury=44.3mo. Control Group (n=12): Mean Age=31.1yr; Mean Time Post Injury=52.2mo. Intervention: Those in the experimental group participated in a cognitive retraining program. The program was divided into 4 modules and ran for 12wk. The control group participated in the same group therapy but the focus was not cognitive but psychosocial functioning and activities of daily living.</td>
<td>1. Individuals in both groups experienced a decrease in depressed mood, as measured by the KAS.</td>
<td>Katz Adjustment Scale (KAS).</td>
</tr>
<tr>
<td>Ashworth et al. (2015)</td>
<td>TBI=7, Stroke=3, non-TBI=2; Mean Age=40.9yr; Gender: Male=7, Female=5. Intervention: Participants received two phases of compassion focused therapy (CFT) 1d/wk for 18wk. Mood groups focused on identifying emotions that encompass ABI and strategies to manage them. Individual sessions addressed content from mood groups and in-depth development of CFT skills. Inter-session homework was encouraged. Assessment performed at baseline, post-treatment, and 3mo follow-up.</td>
<td>1. Significant decrease in depression and anxiety from baseline to post-treatment and baseline to follow-up (p&lt;0.05), measured by the HADS. 2. Increase in reassured self and reduction in hated and inadequate self, according to FSCRS from baseline to post-treatment and baseline to follow up (p&lt;0.05).</td>
<td>Compassion Focused Therapy</td>
</tr>
</tbody>
</table>
### Music Therapy

**Guetin et al. (2009)**  
France  
Pre-Post  
N=13

**Population:** TBI; Mean Age=31yr; Gender: Male=3, Female=10; Mean Time Post Injury=8yr.  
**Treatment:** Patients received music therapy (1hr/wk for 20wk). Each session was divided into two segments: receptive music therapy (e.g. listening) and active music therapy (e.g. playing an instrument).  
**Outcome Measure:** Hospital Anxiety and Depression Scale (HADS).

1. Following each music therapy session, significant improvements in mood were noted on the HADS (p<0.05).  
2. Anxiety scores also decreased following the sessions and could be seen when looking at the scores of Session 1 and Sessions 10, 15 and 20 (p=0.05).  
3. Depression scores also improved but significant improvement was only noted when looking at the scores of Session 1 and 10.

**Thaut et al. (2009)**  
USA  
Case-Control  
N=54

**Population:** Injury Type: TBI=24, CVA=5, Other=4; Mean Age=31yr; Gender: Male=3, Female=10.  
**Intervention:** The treatment group (n=31) received four different sessions of neurologic music therapy (30min) focused on emotional adjustment, executive function, attention and memory. The control group (n=23) were assessed and sent to a quiet room to rest for 30min.  
**Outcome Measure:** Brief Symptom Inventory-18 (BSI-18).

1. On the BSI-18 both groups showed significant improvement (treatment group, p<0.01; control, p=0.01).  
2. Depression and anxiety improved significantly in the treatment group (p=0.02 and p=0.04, respectively) but not the control (p=0.50 and p=0.29).  
3. Hostility significantly improved in the control group (p=0.02) but not the treatment group (p=0.06).

**Nayak et al. (2000)**  
USA  
Prospective Controlled Trial  
N=18

**Population:** TBI and Stroke; Mean Age=59.89yr; Gender: Male=6, Female=12.  
**Intervention:** Participants were assigned to either a control condition (standard rehabilitation alone; n=8) or a treatment condition (standard rehabilitation plus music therapy; n=10). Those in the treatment group received 2-3 treatments per week for up to 10wk.  
**Outcome Measure:** Faces Scale, Social interaction scale, Therapist assessment.

1. The more impaired a participant’s social behaviour at baseline, the more they benefit from the therapy.  
2. The treatment group was more motivated to participate (p=0.06) and more involved in therapy (p<0.01) than the control.  
3. The treatment group was associated with greater improvements in mood (patients’, families’, and therapists’ ratings) than the control group, but not on staffs’ ratings.

### Motivational Counselling

**Cox et al. (2003)**  
USA  
Pre-Post  
N=94

**Population:** TBI; Treatment Group (n=40): Mean Age=32.5yr; Gender: Male=33, Female=7; Mean Time Post Injury=4.4yr.  
**Comparison Group (n=54):** Mean Age=35.6yr; Gender: Male=38, Female=16; Mean Time Post Injury=4.4yr.  
**Intervention:** The treatment group received 12 individual systematic motivational counselling (SMC) sessions for their substance abuse whereas the control group received no counselling.  
**Outcome Measure:** Motivational Structure Questionnaire (MSQ), Positive Affect Negative Affect Scale (PANAS).

1. Within the treatment group substance abuse decreased across all time points (p=0.02).  
2. PANAS and MSQ showed participants in treatment group experienced a significant reduction in negative affect and improvement in motivational structure (p<0.05).

### Cognitive Behavioural Therapy

**Fann et al. (2015)**  
USA

**Population:** TBI=100; Mean Age=45.8yr; Gender: Male=63, Female=37; Mean Time Post

1. Non-significant trend towards improvement in depressive symptoms for
<table>
<thead>
<tr>
<th>Study</th>
<th>Population</th>
<th>Intervention</th>
<th>Outcome Measure</th>
</tr>
</thead>
</table>
| **Prospective Controlled Trial**<br>
N<sub>initial</sub>=100, N<sub>final</sub>=72 | Injury=3.33y; Severity: Moderate=69, Sever=31. **Cognitive Behavioural Therapy (CBT)** on telephone (CBT-T), CBT in person (CBT-IP), or usual care (UC). Both CBT treatments consisted of 30-60min weekly sessions for 12wk. In-session and inter-session homework was assigned. UC group received a phone call with their depressive status, encouraged to continue rehabilitation and directed towards community resources. Depressive symptoms assessed at baseline and 8wk, 16wk and 24wk (follow-up). **Cognitive Behavioural Therapy (CBT)**, **Supportive Psychotherapy (SPT)**<br>Participants received cognitive restructuring and reshaping automatic thoughts. The SPT group received client-centered treatment to improve ability to deal with daily problems effectively. Both groups had 90min sessions 2d/wk for the first week, followed by 50min 1d/wk for 3mo. All measures assessed before and after each treatment session. **Participate in CBT group**<br>Participants in CBT group received treatment based on standard CBT techniques with focus on cognitive restructuring and reshaping automatic thoughts. The SPT group received client-centered treatment to improve ability to deal with daily problems effectively. Both groups had 90min sessions 2d/wk for the first week, followed by 50min 1d/wk for 3mo. All measures assessed before and after each treatment session. **CBT compared to UC at 16wk as reported by patients on SCL-20 (p=0.074), but not on HAMD-17 (p>0.37).**<br>2. **Participants that completed 8 or more sessions of CBT-T had a greater improvement in reported depression by SCL-20 than UC (p=0.043) but not CBT-IP (p=0.170).**<br>3. 62% of CBT participants reported improvement on PGI compared to 39% of UC (p=0.010), however CBT-IP ratings fell to UC levels at 24wk. **Individuals without depression prior to TBI had a significantly larger decrease in depressive symptoms when randomized to CBT than UC (p=0.036), whereas individuals with prior depression did not show a significant difference.**<br>4. | **Outcome Measure**: Hamilton Depression Rating Scale (HAMD-17), Symptom Checklist-20 (SCL-20), Patient Global Impression (PGI). |
| **Ashman et al. (2014)**<br>USA<br>RCT<br>PEDro=7 | **Population**: TBI=54; **Cognitive Behavioural Therapy (CBT)** (n=28): Mean Age=47.5yr; Gender: Male=10, Female=18; Mean Time Post Injury=7.8yr; Severity: Mild=10, Moderate/Severe=17. **Supportive Psychotherapy (SPT)** (n=26): Mean Age=47.1yr; Gender: Male=12, Female=14; Mean Time Post Injury=13.2yr; Severity: Mild=9, Moderate/Severe=12. **Intervention**: Participants in CBT group received treatment based on standard CBT techniques with focus on cognitive restructuring and reshaping automatic thoughts. The SPT group received client-centered treatment to improve ability to deal with daily problems effectively. Both groups had 90min sessions 2d/wk for the first week, followed by 50min 1d/wk for 3mo. All measures assessed before and after each treatment session. **Outcome Measure**: Beck Depression Inventory-Second Edition (BDI-II), State-Trait Anxiety Inventory (STAI), Life-3. | **Cognitive Behavioural Therapy (CBT)**<br>Participants in CBT group reported significant improvement on BDI-II compared to SPT at end of treatment (p<0.05).**<br>2. **Within groups, there was a significant improvement on BDI-II scores (CBT group, p=0.03; SPT, p=0.06).**<br>3. **No significant differences in anxiety between groups were found at the end of treatment (p=0.12).**<br>4. **No significant differences in quality of life as measured by Life-3 (p>0.05) were found.** |
| **D’Antonio et al. (2013)**<br>USA<br>RCT<br>Pedro=6 | **Population**: TBI; Mean Age=48.8yr; Gender: Male=19, Female=25; Mean Time Post Injury=7.7yr. **Treatment**: Participants were randomly assigned to receive 16 sessions over 3mo of cognitive behavioural therapy (CBT) or supportive psychotherapy (SPT). For both groups, the first session lasted 90min; each subsequent session was 50min. **Outcome Measure**: Beck Depression Inventory-II (BDI-II).<br>1. **The CBT group reported significant improvements in sadness, loss of interest and loss of interest in sex (p<0.05).**<br>2. **The SPT reported improvements in agitation (p<0.05), irritability (p<0.01) and the somatic factor of the BDI-II (p<0.05).**<br>3. **Overall BDI-II scores significantly decreased compared to baseline for both groups (p<0.05).**<br>4. **No significant differences were found for individual items or total score of the BDI-II between groups.** | |
| **Andrewes et al. (2014)**<br>UK | **Population**: TBI; Mean Age=42.2yr; Gender: Male=9, Female=1; **Positive Psychology** | **The “three good things” intervention group scored significantly higher on the** |
### Discussion

Several studies were found that specifically evaluated non-pharmacological interventions for depression post ABI. Among these studies the efficacy of exercise and its role in reducing the levels of depression was investigated (Bellon et al. 2015; Blake & Batson 2009; Damiano et al. 2016; Driver & Ede 2009; Gemmell & Leatham 2006; Gordon et al. 1998; Hoffman et al. 2010; Mackelprang et al. 2014; Weinstein et al. 2016). Overall, although improvements in mood were seen with exercise (Bellon et al. 2015; Driver & Ede 2009; Gordon et al. 1998; Weinstein et al. 2016), it was not always significantly better than the controls (Damiano et al. 2016; Gemmell & Leatham 2006). A major difference noted was for individuals who exercised more than 90 minutes per week and those who exercised less. Those in the first group (>90 minutes) showed significantly lower depression scores, better mental health and a higher perceived quality of life (Hoffman et al. 2010; Wise et al. 2012). Moreover, individuals who performed home-based aerobic exercise for 30 minutes a day did not have a significant reduction in depression; however, a decrease in depressive mood was associated with better sleep quality after working out (Damiano et al. 2016). Interestingly, there was a marked reduction in mood disturbance after only one supervised aerobic exercise session found in another study (Weinstein et al. 2016). Two

<table>
<thead>
<tr>
<th>Study</th>
<th>Outcome Measure</th>
<th>Intervention</th>
<th>Population</th>
<th>Mean Time Post Injury</th>
<th>AHI measurement of happiness than the “signature strengths” (p=0.02).</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pedo=5 N=10</td>
<td></td>
<td>Participants were randomly assigned to the 12wk Positive Psychology interventions: “Three Good Things” group or the “Signature Strengths” intervention group. All participants also received weekly individual therapy sessions for substance misuse.</td>
<td>TBI; Mean Age=32yr; Gender: Male=37, Female=5; Mean Time Post Injury=81d.</td>
<td>Interventions: “Three Good Things” group or the “Signature Strengths” intervention group.</td>
<td>1. Based on RIDI, no significant differences between the start and end of therapy occurred.</td>
</tr>
<tr>
<td>Schonberger et al. (2014)</td>
<td>Hospital Anxiety and Depression Scale and Brief Strengths Test</td>
<td>Community Based Rehabilitation Program with a multi-disciplinary team (3-4x/wk).</td>
<td>TBI; Mean Age=32yr; Gender: Male=37, Female=5; Mean Time Post Injury=81d.</td>
<td>Outcome Measure: Hospital Anxiety and Depression Scale (HADS), Self-Awareness of Deficits Interview (SADI), Sydney Psychosocial Reintegration Scale-2 (SPRS) and Reactions to Impairment and Disability Inventory (RIDI).</td>
<td>1. Significant improvement of affective disorders was found; anxiety (p&lt;0.001), depressive mood (p&lt;0.001) and hostility (p&lt;0.01).</td>
</tr>
<tr>
<td>Wiart et al. (2012)</td>
<td></td>
<td>Retrospective review of patients referred to a single physician for at least 1yr of neuro-systemic psychotherapy.</td>
<td>TBI; Mean Age=33.4yr; Gender: Male=35, Female=12; Mean Time Post Injury=11.1yr.</td>
<td>Outcome Measure: Hospital Anxiety and Depression Scale (HADS), Self-Awareness of Deficits Interview (SADI), Sydney Psychosocial Reintegration Scale-2 (SPRS) and Reactions to Impairment and Disability Inventory (RIDI).</td>
<td>1. Significant improvement of affective disorders was found; anxiety (p&lt;0.001), depressive mood (p&lt;0.001) and hostility (p&lt;0.01).</td>
</tr>
<tr>
<td>N=42</td>
<td>Population: TBI; Mean Age=32yr; Gender: Male=37, Female=5; Mean Time Post Injury=81d.</td>
<td>Based on RIDI, no significant differences between the start and end of therapy occurred.</td>
<td>1. Based on RIDI, no significant differences between the start and end of therapy occurred.</td>
<td>2. Good RIDI adjustment was predicted by a good functional status as rated by SPRS total and positive SADI score.</td>
<td></td>
</tr>
<tr>
<td>N=47</td>
<td>Population: TBI; Mean Age=33.4yr; Gender: Male=35, Female=12; Mean Time Post Injury=11.1yr.</td>
<td>Retrospective review of patients referred to a single physician for at least 1yr of neuro-systemic psychotherapy.</td>
<td>Population: TBI; Mean Age=32yr; Gender: Male=37, Female=5; Mean Time Post Injury=81d.</td>
<td>Outcome Measure: Hospital Anxiety and Depression Scale and Brief Strengths Test.</td>
<td>3. RIDI adjustment was predicted by: SPRS-Therapist, SADI, and SPRS-Therapist interaction (p&lt;0.05).</td>
</tr>
</tbody>
</table>

PEDro=Physiotherapy Evidence Database rating scale score (Moseley et al. 2002).
studies investigated the benefits of the Chinese exercise methods Tai Chi Qigong (Blake & Batson 2009) and Tai Chi Chaun (Gemmell & Leathem 2006) on those who had sustained a TBI. Results from both these studies found an improvement in mood. However, due to the small sample sizes in each study limited conclusions can be drawn on their effectiveness in reducing depression.

Other non-pharmacological interventions are teaching coping skills, compassion focused therapy and CBT. In two RCTs conducted by Anson and Ponsford (2006a, 2006b), individuals who participated in the CSG, increased their adaptive coping skills. However, no significant changes in patients’ anxiety, self-esteem, depression or psychosocial scores were noted following the CSG. In the second study, those who had a greater self-awareness following their injuries demonstrated better outcomes post CSG intervention (Anson & Ponsford 2006b). Ruff and Niemann (1990) compared subjects who participated in an eight week cognitive remediation programme with subjects attending a treatment day program. As measured by the Katz Adjustment Scale, both groups experienced a decrease in depressed mood. In regards to compassion focused therapy, Ashworth et al. (2015) found a reduction in depression and anxiety, but also that individuals learned to be more positive and compassionate with themselves.

Two RCTs compared patients who received CBT for 12-16 weeks to patients who received supportive psychotherapy (Ashman et al. 2014; D’Antonio et al. 2013). Overall depression scores decreased compared to baseline, yet no between group differences were found (Ashman et al. 2014; D’Antonio et al. 2013). A third study compared CBT delivery methods and found no significant difference between treatment in person and treatment over the phone. However, individuals who underwent CBT had a larger decrease in depressive symptoms if they did not have a history of depression prior to TBI (Fann et al. 2015). Stalder-Lüthy et al. (2013) conducted a systematic review and report that CBT is effective for the treatment of depression for individuals who have experienced a TBI.

Three studies looking at the efficacy of mindfulness-based stress reduction (MBSR) programs on depression post ABI were conducted by Bedard and colleagues (Bedard et al. 2003; Bedard et al. 2012; Bedard et al. 2014). After a small pilot study (Bedard et al. 2003) and a pre-post study (Bedard et al. 2012) with positive results in favour of mindfulness-based stress reduction interventions reducing depression, Bedard (2014) investigated this therapy through an RCT. The programme consisted of 10 weeks of therapy designed to encourage a new way of thinking about life and disability. Results found that those in the intervention group showed a significantly greater reduction in Beck Depression Inventory scores compared to the control group with this reduction maintained at three month follow-up. In a recent pilot RCT, patients with TBI were randomly assigned to two variations of positive psychology interventions (“Three Good Things” or “Signature Strengths”) (Andrewes et al. 2014). No significant differences were found from pre-test to post test on the Authentic Happiness Scale or the Head Injury Semantic Differential Scale, although participants in the “Three Good Things” intervention, scored significantly higher on the happiness measure than patients in the “Signature Strengths” intervention.

Studies investigating music as an intervention have demonstrated positive results related to patient mood. In an early repeated measures design, patients who received standard rehabilitation plus music had greater improvements in reported mood in comparison to patients who received standard rehabilitation only (Nayak et al. 2000). More recently, both listening to music and active music therapy (e.g. playing a musical instrument, singing or writing a song) improved mood (symptoms of anxiety and depression) from initial assessments to final assessments (Guétin et al. 2009). Another study had participants in a treatment group participate in 4 sessions focussing on attention, memory, executive function and emotional adjustment followed by a 30 minute neurologic music therapy program while a
control group completed various assessments and sat quietly for 30 minutes after the assessments (Thaut et al. 2009). Although there were no improvements on cognitive measures, participants in the intervention group improved on depression and anxiety subscales and both groups improved on the BSI-18 (Thaut et al. 2009).

Although there is preliminary evidence for a number of non-pharmacological interventions for mood, in particular the treatment of depression, given the limited evidence, non-pharmacological interventions cannot be considered as alternatives to pharmacological interventions. However, non-pharmacological treatments may help augment the action of antidepressants and should therefore be part of the treatment of depression post ABI.

Conclusions

*There is Level 1a evidence that individuals with a traumatic brain injury who participate in exercise programs report feeling less depressed and report experiencing greater quality of life post injury.*

*There is Level 1b evidence that mindfulness-based stress reduction programmes may be efficacious in reducing depressed mood.*

*There is Level 3 evidence that music therapy does improve depression and anxiety post-acquired brain injury.*

*There is Level 4 evidence that Systematic Motivational Counselling may reduce negative affect.*

*There is Level 1b evidence that both Cognitive Behavioural Therapy and supportive psychotherapy may decrease symptoms associated with depression.*

*There is Level 2 evidence that positive psychology, involving patients writing down things they enjoy, is beneficial in improving happiness scores.*

*There is Level 4 evidence that rehabilitation decreases self-reported depression scores.*

| Music therapy may be more efficacious in improving anxiety and depression than standard rehabilitation alone. |
| Systematic Motivational Counselling may reduce negative affect. |
| Teaching coping skills to those who have sustained a traumatic brain injury helps to reduce anxiety and depression. |
| Compassion Focused Therapy may reduce depression and anxiety while improving self-compassion. |
| Exercise is associated with feeling less depressed and an improved quality of life post-traumatic brain injury. |
| A mindfulness-based stress reduction programme may be efficacious in reducing depressed mood. |
Cognitive Behavioural Therapy and supportive psychotherapy may decrease patient symptoms associated with depression.

There is no difference in reduction of depressive symptoms between Cognitive Behavioural Therapy delivered over the phone or in person.

Positive psychology, involving patients writing down things they enjoy in life, may increase patient happiness.

Individualized rehabilitation may decreases feelings of depression.

8.3 Anxiety Related Disorders

Anxiety is a subjective sensation of apprehension of danger and dread that may be accompanied by signs of restlessness, tension, tachycardia and shortness of breath that are part of the fight or flight response. Anxiety can be quite disabling whether it is generalized or includes a specific phobia to a certain stimulus. Anxiety disorders (e.g., Generalized Anxiety Disorder, PTSD, etc.) are common following ABI. Anxiety can be related to confusion and cognitive impairment or may be specifically related to the psychological trauma of the injury itself. It may also be a common symptom post ABI (e.g., associated with depression, related to stress, etc.). In the non-brain injured population, a cognitive-behavioural program directed at managing and reducing the disabling symptoms that cause avoidance of the stimulus may effectively treat anxiety. However treatment of anxiety post ABI may not be as effective because of cognitive impairments in this population.

8.3.1 Incidence and Prevalence post ABI

Post ABI, anxiety or anxiety disorders have been reported to occur in 4 to 28% of those who have been injured (Deb et al. 1999; Fann et al. 1995; O’Donnell et al. 2008; Osborn et al. 2015; van Reekum et al. 1996). A meta-analysis including 32 studies discovered that self-reported rates of anxiety in the TBI population is 37% (Osborn et al. 2015). In a study conducted by Hibbard et al. (1998) looking at various anxiety disorders post ABI, 19% of the study population was diagnosed with PTSD, 15% with OCD, and 14% with panic disorder. These findings were confirmed in a study where the most frequently reported disorders post TBI were anxiety disorders otherwise not specified, followed by PTSD (Gould et al. 2014). These results also reported that patients with TBI who experienced post injury anxiety were generally older than patients without post injury anxiety (Gould et al. 2014).

Table 8.4 Non-Treatment Studies of Anxiety Post ABI

<table>
<thead>
<tr>
<th>Author/Year/Country/Study Design</th>
<th>Population</th>
<th>Methods</th>
<th>Outcomes</th>
</tr>
</thead>
<tbody>
<tr>
<td>Gould et al. (2014) Australia Pre-Post N=66</td>
<td>TBI; Mean Age=32.9yr; Gender: Male=52, Female=14.</td>
<td>Examined associations between cognitive functioning and anxiety post TBI.</td>
<td>1. Prevalence of anxiety disorder post injury was 27.3%.</td>
</tr>
<tr>
<td></td>
<td>Intervention: Examined associations between cognitive functioning and anxiety post TBI.</td>
<td>Outcomes Measure: Structured Clinical Interview (SCID) from the DSM-IV, Controlled Oral Word Association Test, Hayling Sentence Completion Test, Trail Making Test part A (TMT-A), Digit Span subtest of the Wechsler Adult Intelligence Test-Third Edition (WAIS-III), Hayling Sentence Completion Test Part 1, Symbol Digit Modalities</td>
<td>2. Individuals with post injury anxiety were on average 9yr older than those without.</td>
</tr>
<tr>
<td></td>
<td>Outcome Measure:</td>
<td>3. The most frequent disorders were anxiety disorders otherwise not specified and PTSD.</td>
<td>4. More than half of the sample had a pre-injury psychiatric disorder but most patients with post-injury anxiety did not have psychiatric history.</td>
</tr>
</tbody>
</table>
Test Oral Version, SDMT, BIRT Memory and Information Processing Battery (B-MIPD), Doors and People Test.

5. The speed of information processing based on Hayling part 1, TMT-A and Symbol Digit Modalities test most strongly differentiated patients with and without anxiety (p<0.05).

6. The speed of information processing model accounted for more than a third of the variance associated with anxiety disorder status.

8.3.2 Non-Pharmacological Interventions for Anxiety

Although anxiety disorders appear to be well recognized post ABI there is little in the literature regarding use of non-pharmacological Interventions.

Individual Studies

<table>
<thead>
<tr>
<th>Author/Year/Country/Study Design/PEDro/N</th>
<th>Population</th>
<th>Methods</th>
<th>Outcomes</th>
</tr>
</thead>
<tbody>
<tr>
<td>Bertisch et al. (2013) USA Case Series N=54</td>
<td>TBI=17, ABI=8, Stroke=19, Other=2; Mean Age=54.7yr; Gender: Male=33, Female=21; Mean Time Post Injury=7.35mo.</td>
<td>1. Only BAI scores significantly predicted impairment on the Affective/Behavioural Scale (p&lt;0.05). 2. No cognitive variables were significant predictors on any scale of the Hifi PCL.</td>
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</table>

| Hsieh et al. (2012) Australia RCT PEDro=6 N=27 | TBI; Mean Age=38yr; Gender: Male=21, Female=6; Mean Time Post Injury=37.9mo. | Experimental Group 1 (n=9): received 3 weekly sessions of motivational interviewing (MI), then CBT. Experimental Group 2 (n=10): received 3 sessions of non-directive counselling followed by CBT. Control group (n=8) received customary care. | 1. The positive correlation between posttraumatic amnesia (PTA) and HADS change score suggests that greater reduction in anxiety is associated with lower injury severity. 2. Following treatment those in the experimental groups each showed significant reductions on the HADS anxiety but not on the DASS-anxiety. 3. Those in Experimental Group 1 showed a greater response to CBT than those in Experimental Group 2, in regards to a reduction in anxiety, stress and non-productive coping. |

| Arundine et al. (2012) Canada Cohort N=17 | TBI=9, ABI=8; Mean Age=42.94yr; Gender: Male=9, Female=7; Mean Time Post Injury=9.65yr. | Patients received the first cognitive behaviour therapy (CBT) session face to face. The remaining 10 sessions were conducted either face-to-face in a group format (G-CBT; n=10), or individually by phone (T-CBT; n=7). | 1. Results of the SCL-90-R-GSI and the DASS-21 indicate emotional distress was significantly decreased following the intervention (p<0.01) for all participants. 2. Results from the CIQ also improved significantly (p<0.01). 3. The two groups showed no significant difference between them following the intervention. 4. All participants showed greater improvement at the 6mo follow-up |
Evidence-Based Review of Moderate to Severe Acquired Brain Injury

Module 8 - Mental Health Issues Post ABI

**Hodgson et al. (2005)**
Australia
RCT
PEDro=5
N=12

<table>
<thead>
<tr>
<th>Population:</th>
<th>ABI; Gender: Male=7, Female=5. Treatment Group (n=6): Mean Age=44.2y; Mean Time Post Injury=96.7mo. Waitlist Group (n=6): Mean Age=33.8y; Mean Time Post Injury=150.5mo. Intervention: Participants were randomly assigned to either the CBT group or the wait list control group. The CBT treatment program consisted of relaxation training, cognitive strategies, graded exposure and assertiveness skills training. Those in the treatment group were seen weekly for hourly sessions, for a total of 9 to 14wk. Outcome Measure: The Hospital Anxiety and Depression Scale (HADS), Coppersmith Self Esteem Inventory, and the Social Phobia and Anxiety Inventory (SPAI).</th>
</tr>
</thead>
<tbody>
<tr>
<td>1.</td>
<td>Post treatment the SPAI scores had decreased for the treatment group, but not for the wait list control group.</td>
</tr>
<tr>
<td>2.</td>
<td>At the one month follow up a significant difference (p&lt;0.006) was found when comparing the two groups.</td>
</tr>
<tr>
<td>3.</td>
<td>Results from the HADS showed that prior to treatment, there were in total 7 individuals who displayed probable – definite levels on the depression subscale, and 11 who displayed probable – definite levels on the anxiety scale.</td>
</tr>
<tr>
<td>4.</td>
<td>Post intervention those in the wait list control group scored higher on the depression and anxiety subscales of the HADS, compared to the treatment group whose scores decreased significantly on both subscales.</td>
</tr>
</tbody>
</table>

**Discussion**

Several studies have investigated the benefits of CBT to reduce anxiety levels in those who sustained a TBI (Arundine et al. 2012; Hodgson et al. 2005; Hsieh et al. 2012). Hodgson et al. (2005) found that anxiety scores on the Hospital Anxiety and Depression Scale (HADS) and the Social Phobia and Anxiety Inventory decreased significantly more following 9 to 12 weeks of CBT treatment than for the wait list control group, indicating that CBT training can reduce anxiety and depression in a TBI sample. Hsieh and colleagues (2012) examined CBT in combination with other therapies; specifically, motivational interviewing and CBT (group 1), the non-directive counselling group and CBT (group 2), or the treatment as usual (TAU) group (group 3). Those in groups 1 and 2 showed a significant reduction in anxiety following treatment as compared to group 3. Those in group 1 showed a greater response to the CBT compared to group 2 (Hsieh et al. 2012). In terms of the way in which CBT is delivered, Arundine et al. (2012) found that both face-to-face group CBT and one-to-one telephone CBT were effective in improving community integration and mood, with no significant differences between groups. Study authors suggest teletherapy may be just as beneficial to patients post TBI as group therapy (Arundine et al. 2012).

**Conclusions**

There is Level 1b evidence that Cognitive Behavioural Therapy does reduce anxiety post-acquired brain injury.

Cognitive Behavioural Therapy does reduce anxiety and depression post-acquired brain injury.

**8.3.3 Obsessive Compulsive Disorder**

Following a TBI, anxiety disorders such as OCD, panic attacks and stress disorders are common both within the adult and paediatric populations. OCD is believed to be present in less than 10% of the brain...
injury population (Berthier et al. 2001), it is rarely reported in the literature (Drummond & Gravestock 1988). Studies conducted by McKeon et al. (1984) and Kant et al. (1996) have found OCD symptoms appearing shortly after injury, within the first few hours to the first week. Some patients were found to develop symptoms within the first 6 months of sustaining their injury. Several authors have suggested the location of the brain lesion may predict OCD in patients (Bilgic et al. 2004; Donovan & Barry 1994; Jenike & Brandon 1988). To date, although several theories have been put forth (lesion location, age of the individual) there is still no conclusive evidence. Grados (2003) noted that OCD has been treated successfully with serotonin selective re-uptake inhibitors such as fluoxetine, paroxetine, fluvoxamine or sertraline. Other supportive therapies have also been reported to be successful although there were no clinical trials found in the literature.

Interventions

Table 8.6 Interventions Used for Obsessive Compulsive Disorder Post ABI

<table>
<thead>
<tr>
<th>Author/Year/Country/Study design</th>
<th>Methods</th>
<th>Outcomes</th>
</tr>
</thead>
<tbody>
<tr>
<td>Childers et al. (1998) USA Case Study N=4 Population: TBI; Mean Age=38yr; Gender: Male=4, Female=0. Intervention: Patient 1: given 10mg of prozac daily; Patient 2: given 25mg of clomipramine hydrochloride at bedtime; Patient 3: given 20mg of paroxetine hydrochloride at bedtime; Patient 4: given 25mg clomipramine hydrochloride at bedtime. Outcome Measure: Diagnostic and Statistical Manual of Mental Disorders, 4th ed (DSM-IV).</td>
<td>1. Individuals would become upset, anxious, or disruptive if routines were disrupted for any reason. 2. Clinical improvement was noted for each patient.</td>
<td></td>
</tr>
</tbody>
</table>

Discussion

In each of these case studies, individuals were treated with a variety of medications to reduce or extinguish the frequency of OCD post ABI. In each case the individualized drug treatment therapy chosen was shown to be effective. No conclusions can be drawn from this study.

Although Obsessive Compulsive Disorder has been identified post-acquired brain injury there does not appear to be one method of intervention that works for all, but rather interventions remain individualized.

Little research has been conducted looking at the effects of various interventions on Obsessive Compulsive Disorder post-acquired brain injury.

8.3.4 Post-Traumatic Stress Disorder

Earlier literature of PTSD following TBI focussed on patients with mild brain injury. This was based on the belief that PTSD could not develop in the presence of amnesia for the traumatic event (Bryant et al. 2001; Mayou et al. 1993; Warden et al. 1997). Since then, research has found the PTSD can occur with mild, moderate and severe TBI (Al-Ozairi et al. 2014; Bryant et al. 2001). A recent population-based TBI sample reported nearly 18% of patients met criteria for PTSD, a sample which included patients with moderate and severe TBI (Barker-Collo et al. 2013). However, research within the severe TBI population is needed, as PTSD in this group is less studied than many other psychological disorders post TBI.
Table 8.7 Non-Intervention Studies Exploring Post-Traumatic Stress Disorder

<table>
<thead>
<tr>
<th>Author/Year/Country/Study design</th>
<th>Methods</th>
<th>Outcomes</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Al-Ozairi et al. (2014)</strong></td>
<td>Population: TBI; Mean Age=34.4yr; Gender: Male=702, Female=412; Mean Time Post Injury=69d. Intervention: Questionnaires administered. Outcome Measure: Clinical interview, Medical record review, GCS, 15-Item Impact of Event Scale, General Health Questionnaire-28 (GHQ-28), Rivermead Post concussion Disorder Questionnaire.</td>
<td>1. Increased PTA duration was related to reduced frequency of return to work, worse GCS scores (p&lt;0.001), increased duration of loss of consciousness (p&lt;0.001) and frequency of CT scan abnormalities (p&lt;0.001). 2. The PTA group with a duration of less than an hour had the most intrusive post-traumatic stress and anxiety symptoms (p&lt;0.001). 3. Predictors of post-traumatic stress avoidance symptoms included: all GHQ subscales, GHQ total and fatigue (p&lt;0.05). 4. Predictors of post-traumatic stress intrusive symptoms included: light sensitivity, noise sensitivity and PTA (p&lt;0.05).</td>
</tr>
<tr>
<td><strong>Bryant et al. (2001)</strong></td>
<td>Population: Severe TBI; Mean Age=34.6yr; Gender: Male=77, Female=19; Mean Time Post Injury=6.3mo. Intervention: Questionnaires administered. Outcome Measure: Westmead PTA Scale, GCS, PTSD Interview (PTSD-I), Beck Depression Inventory (BDI), General Health Questionnaire (GHQ), Functional Assessment Measure (FAM), Functional Independence Measure (FIM), Community Integration Questionnaire (CIQ), Overt Aggression Scale (OAS) and Satisfaction with Life Scale (SWLS).</td>
<td>1. PTSD was diagnosed in 27% of the patient sample. 2. 27% of patients reported minimal depression, 11% moderate and 8% severe. 3. Patients with PTSD reported higher PTSD-I, BDI, and GHQ scores and lower scores on the FAM, CIQ-total/Productivity, OAS-Total/Verbal aggression, and SWLS as compared to patients without PTSD (p&lt;0.05). 4. Significant PTSD re-experiencing symptoms, PTSD avoidance, and PTSD arousal symptoms were each positively correlated with BDI, GHQ, each aggression scale and were negatively correlated with satisfaction with life (p&lt;0.05). 5. PTSD avoidance was also negatively correlated with CIQ-social; and PTSD arousal with CIQ-productivity and social.</td>
</tr>
<tr>
<td><strong>Bryant et al. (2000)</strong></td>
<td>Population: Severe TBI; Mean Age=34.6yr; Gender: Male=77, Female=19; Mean Time Post Injury=6.3mo. Intervention: Questionnaires administered. Outcome Measure: GCS and PTSD Interview.</td>
<td>1. PTSD was diagnosed in 27% of patients. 2. Most patients who met criteria for PTSD endorsed each PTSD symptom. 3. The symptom that had the highest positive predictive power was intrusive memories, nightmares and emotional reactivity.</td>
</tr>
</tbody>
</table>

Discussion

A growing body of literature suggests that PTSD symptoms can develop after mild, moderate, and severe TBI. However, it has frequently been suggested that severe brain injury may be associated with a diminished risk of PTSD and research with a TBI sample compared to a control sample has also supported this claim (Zatzick et al. 2010). Additional research is necessary to confirm factors that may
Little research has been conducted regarding Post-Traumatic Stress Disorder in patients with moderate-severe traumatic brain injury, additional research is needed.

8.4 Suicidal Ideation
Suicidal ideations are the thoughts or considerations of suicide that when left unattended can lead to distress and attempted suicide. Risk factors for suicide overlap with characteristics present after a TBI; therefore, it is not surprising that there is an increased risk of suicide following a TBI (Bahraini et al. 2013; Simpson & Tate 2007). Unfortunately, the risk for suicidal ideation and attempt remains high at 20 years after ABI (Fisher et al. 2016).

8.4.1 Incidence and Prevalence Post ABI
Within the TBI population, 23-28% of individuals report suicidal ideation after sustaining a TBI (Mackelprang et al. 2014; Simpson & Tate 2002; Tsaousides et al. 2011). Male veterans are more likely to have suicidal ideation compared to females (Wisco et al. 2014); however, no such relationship was found for age at time of injury (Mackelprang et al. 2014; Simpson & Tate 2002). Risk of suicidal ideation can be further augmented with co-morbid diagnosis of depression, anxiety or PTSD (Tsaousides et al. 2011) and the number of sustained TBIs (Wisco et al. 2014). Furthermore, elevated suicidal ideation at 1 year post-TBI is associated with continual elevation of ideation at 5 years (Fisher et al. 2016), demonstrating the necessity for therapies targeting such ideations.

If suicide ideation is not minimized, the risk of suicide attempts is prevalent (Simpson & Tate 2007); this risk is further increased when emotional distress, a common TBI characteristic, is present (Gutierrez et al. 2008; Simpson & Tate 2002). Within their lifetime, 26% of individuals post TBI attempt suicide, with half making more than one attempt (Simpson & Tate 2002, 2005). Men are at a higher risk of attempting suicide post TBI compared to the general population, where women are more likely (Simpson & Tate 2002). Furthermore, emotional disturbance and substance abuse history increase the risk for attempted suicide by a factor of 21, compared to individuals with no history (Simpson & Tate 2005).

Table 8.8 Non-Pharmacological Intervention for Suicide Prevention

<table>
<thead>
<tr>
<th>Author/Year/Country/Study Design/N</th>
<th>Methods</th>
<th>Outcomes</th>
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<tbody>
<tr>
<td><strong>Simpson et al. (2011)</strong> Australia RCT PEDro=8 N=17</td>
<td><strong>Population</strong>: TBI=17; Severity: Severe=17; <strong>Treatment Group</strong> (n=8): Mean Age=39.4yr; Mean Time Post Injury=6.3yr. <strong>Control Group</strong> (n=9): Mean Age=44.1yr; Mean Time Post Injury=7.6yr. <strong>Intervention</strong>: Participants were randomized to receive a group-based therapy (2hr/wk for 10wk) focused on alleviating current problems with hopelessness. The control group received standard therapy. At 10wk, participants</td>
<td>1. Significant group-by-time interaction on BHS (p=0.002) but no significant main effects for either group or time. 2. Individuals receiving treatment had larger improvements in hopelessness (BHS scores) at 10wk; 75% reduced hopelessness by 1 severity band and 50% maintained/reduced severity by 20wk. 3. Hopelessness on BHS improved significantly from pre to post treatment for all</td>
</tr>
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</table>
Evidence-Based Review of Moderate to Severe Acquired Brain Injury

Module 8 - Mental Health Issues Post ABI

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Updated August 2016

<table>
<thead>
<tr>
<th>Author/Year/ Country/ Study Design/N</th>
<th>Methods</th>
<th>Outcomes</th>
</tr>
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<tbody>
<tr>
<td>crossed over. Assessments occurred at baseline, 10wk, and at study end (20wk).</td>
<td>participants (p=0.008), a clinical improvement from moderate to mild severity.</td>
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<tr>
<td>Outcome Measure: Beck Hopelessness Scale (BHS), Beck Scale for Suicide Ideation (BSS), Hospital Anxiety and Depression Scale (HADS), Herth Hope Index (HHI), Rosenberg Self-Esteem (RSE), Social Problem Solving Inventory-Revised (SPSI-R).</td>
<td>4. Suicidal ideation according to mean BSS scores was stable in the treatment group, but increased in the control group.</td>
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<tr>
<td>5. No significant change on HADS, HHI, RSE, and SPSI-R were found.</td>
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</table>

Discussion

Hopelessness is a precursor to suicidal ideation, which in turn increases the risk of suicide. One RCT found that feelings of hopelessness after severe TBI may be reduced through group-based therapy that targets associated psychological problems. Hopelessness decreased in the treatment group, but suicidal ideation increased in the control group who did not receive treatment, underlining the risk of leaving suicidal distress untreated (Simpson et al. 2011). It is important to continue therapy after the primary intervention, as only half of the individuals were able to maintain the reduction in hopelessness by study end (Simpson et al. 2011). Simpson et al. (2011) argue that hopelessness and depression can manifest independently; hopelessness decreased in the treatment group whereas depression levels remained the same. It is therefore important to develop therapies focused on the precursors of suicidal ideation, such as hopelessness, and not solely depression (Simpson et al. 2011). Therapies may target the prevention/amelioration of psychological symptoms of suicide to improve outcomes post-TBI, but further research is warranted, as only one high quality low powered study is presented.

Conclusions

There is Level 1b evidence that reducing hopelessness post-traumatic brain injury may be effective at decreasing suicidal ideation.

Little research has been conducted regarding treatments for suicide in individuals with moderate or severe traumatic brain injury; further research is warranted.

8.5 Challenging Behaviours

Behaviour can be defined as any interaction between an organism and their environment. This encompasses almost everything that humans do; however, most people tend to think of behavioural problems in a more restricted sense of antisocial, uncooperative or negative interactions associated with interpersonal problems. Challenging behaviour following a brain injury occurs with a relatively high frequency (25-50%). Challenging behaviour can include, but is not limited to, the following: non-compliance with treatment, anger, agitation, verbal and/or physical aggression and depression. The emergence of these behaviours likely arises from injury to the frontal lobes and results in disinhibited behaviour and a lack of recognition for the consequences of one’s behaviour (Kim 2002). Typically behavioural management techniques and pharmacological interventions are used to minimize and/or alleviate these challenges with varying degrees of success.
Few investigators have examined predictors of aggressive symptoms following brain injury, although it has been suggested that disinhibition and depression may result in aggressive behaviour in some individuals after injury (Bakchine et al. 1989; Kim & Humaran 2002). In a sample of 228 patients with moderate to severe brain injury, Baguley et al. (2006) found depression and younger age to be a main predictors of aggression following brain injury at 6, 24, and 60 months. Similarly, Wolffbrandt et al. (2013) found younger age to be associated with agitation in patients with TBI along with lower FIM scores. Due to small sample sizes in previous studies and the inconsistency in tools used to measure aggression, making comparisons between studies is difficult (Baguley et al. 2006).

8.5.1 Agitation and Aggression

Agitation is generally defined as wandering, edginess, distractibility, non-compliance, and/or impulsiveness, while aggression is defined as physical or verbal violence that may put the individual and others at risk for injury (Eisenberg et al. 2009). Aggressive behaviours post TBI are associated with the presence of depression, frontal lobe lesions, and a history of substance or alcohol abuse (Singh et al. 2014; Tateno et al. 2003). TBI injuries that can lead to aggressive or agitated behaviour may result from a diffuse injury, lesions in the frontal lobe (Warriner & Velikonja 2006) and/or injuries to the left hemisphere (Tateno et al. 2003). Agitation is more common among younger individuals and those with lower FIM scores on admission (Wolffbrandt et al. 2013). Individuals found to have poorer social functioning often engage in a variety of aggressive or agitated behaviours including: hitting, kicking, refusing to participate in activities, memory deficits and slowness, decreased attention span, impulsivity, wondering off the unit, throwing objects, verbal aggression and engaging in self-abusive behaviours (McNett et al. 2012; Rao et al. 2009).

Following an ABI, studies have suggested that aggressive behaviour is linked to the level of serotonin in the brain. An ABI often results in serotonergic dysfunction thus increasing the risk of aggressive behaviours (Jorge & Starkstein 2005).

8.5.1.2. Prevalence and Predictors of Agitation

Table 8.9 Non-Intervention Studies of Agitation Post ABI

<table>
<thead>
<tr>
<th>Author/Year/ Country/Study design</th>
<th>Methods</th>
<th>Outcomes</th>
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<tbody>
<tr>
<td>Singh et al. (2014) UK Observational N=146</td>
<td>Population: TBI; Mean Age=45.4yr. Intervention: Assessments completed. Outcome Measure: Agitation Behaviour Score (ABS), CT Scan, Glasgow Outcome Scale, and medication use.</td>
<td>1. 36.3% of patients were agitated; average duration of agitation was 39d. 2. In 49% of cases there was a history of alcohol excess. 3. 24.5% of patients received no behavioural medication, 32.1% received one agent and 43.4% required change of medication to an alternative agent. 4. Association between outcome and type of pathology (p&lt;0.002), with excess alcohol intake (p&lt;0.003), with the severity of the agitation behaviour as graded on ABS (p&lt;0.001) and with the type of treatment used (p&lt;0.001). 5. A number of factors were associated with agitation including, type of lesion on CT scan, severity of agitation and duration of behaviour.</td>
</tr>
</tbody>
</table>
8.5.2 Pharmacological Interventions for Agitation and Aggression

Agitation occurs in approximately 33-55% of patients with TBI (Singh et al. 2014; Tateno et al. 2003). The term agitation encompasses a wide variety of behaviours including restlessness, wandering, shouting, etc. This diversity of behaviours is typical of the agitation seen post ABI, but creates problems in terms of research regarding treatment efficacy (e.g., targeting interventions to particular types of agitation). Agitation is often a recovery-limiting factor as it creates both a disruptive and unsafe environment for rehabilitation (Rosati 2002). Pharmacological interventions are often used to treat this problem and include a variety of medications such as: anti-epileptics, dopaminergic agents, anti-depressants, beta-blockers, and anti-psychotics, as well as others. This section will look at each in detail.

8.5.2.1 Dopaminergic Medications

Dopamine is a fundamental neurotransmitter that plays a role in frontal lobe stimulation through the activation of dopaminergic receptors, affecting a range of functions such as behaviour, mood, motor
ability, and arousal, to name a few (Sawyer et al. 2008). Dopaminergic medications have been previously used in the treatment of Parkinson’s disease and for remediation of cognitive impairments post TBI.

8.5.2.2 Amantadine

Amantadine is a non-competitive N-methyl-D-aspartate receptor antagonist that decreases glutamate levels, which may improve learning, memory, and behaviour deficits (Hammond et al. 2014). Additionally, Amantadine has dopaminergic function; it can indirectly facilitate dopamine release presynaptically and directly inhibit dopamine reuptake at the post-synapse (Hammond et al. 2014). Originally, Amantadine was used as an anti-viral medication for influenza A, but later gained popularity as an anti-Parkinsonian treatment. However, the effects of amantadine on reducing irritability and aggression have yet to be established among the TBI population.

Table 8.10 Effects of Amantadine on Reducing Aggression

<table>
<thead>
<tr>
<th>Author/Year/Country/Study Design/N</th>
<th>Methods</th>
<th>Outcomes</th>
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<tbody>
<tr>
<td>Hammond et al. (2015) USA RCT PEDro=10 N_initial=168, N_final=157</td>
<td>Population: TBI=168; Amantadine (n=82): Mean Age=40.2yr; Gender: Male=66, Female=16; Severity: Mild=20, Moderate=3, Severe=59. Placebo (n=86): Mean Age=38.2yr; Gender: Male=64, Female=22; Severity: Mild=22, Moderate=1, Severe=63. Intervention: Participants were randomized to receive either 100mg of amantadine or a placebo every morning and at 12pm for 60d. Assessments to determine state of irritability were conducted at baseline, 28d, and 60d. Outcome Measure: Neuropsychiatric Inventory Irritability (NPI-I) Most Problematic, NPI-I Most Aberrant, NPI-I Distress.</td>
<td>1. No significant differences in irritability between groups on observer NPI-I ratings at 28d or 60d, but both groups showed improvement in irritability. 2. Participant-rated NPI-I Most Problematic (p=.0353) and Distress (p=.0362) scores were significantly different between amantadine and placebo at 60d, however after adjustment multiple comparisons revealed no significant difference.</td>
</tr>
<tr>
<td>Hammond et al. (2014) USA RCT PEDro=9 N_initial=76, N_final=72</td>
<td>Population: TBI=76; Amantadine Group (n=38): Mean Age=34.7yr; Gender: Male=25, Female=13; Mean Time Post Injury=5.3yr; Mean GCS=9.5. Placebo Group (n=38): Mean Age=42.1yr; Gender: Male=22, Female=16; Mean Time Post Injury=4.7yr; Mean GCS=7.5. Intervention: Participants were randomized to receive placebo or 100mg of amantadine hydrochloride in the morning and at 12pm every day for 28d. Participants were assessed for effects of amantadine on irritability and aggression at baseline and post-treatment. Outcome Measure: Neuropsychiatric Inventory Irritability (NPI-I) and NPI Agitation/Aggression (NPI-A), NPI Distress (NPI-D), Beck Depression Inventory-II (BDI-II), Brief Symptom Inventory (BSI), Global Mental Health Scale (GMHS).</td>
<td>1. 81% of patients with a TBI who took amantadine had improved irritability by at least 3 points on NPI-I, compared to 44% of placebo (p=.0016). 2. Significant difference in frequency and severity of irritability on NPI-I between amantadine and placebo groups (p=.0085). 3. No significant differences between amantadine and placebo on NPI-D, BDI-II, GMHS, or BSI-anxiety. 4. Only individuals with moderate to severe aggression at baseline on NPI-A had significant change in aggression after amantadine treatment compared to placebo (p=.046).</td>
</tr>
</tbody>
</table>

Discussion

One placebo-controlled RCT compared the effects of Amantadine on irritability and aggression. The frequency and severity of irritability were reduced when individuals were on Amantadine for 28 days,
compared to placebo. However, Amantadine only significantly reduced aggression in individuals who had moderate-severe aggression at baseline (Hammond et al. 2014). A second RCT furthered Hammond et al. (2014) findings by assessing the effects of Amantadine on irritability and aggression for up to 60 days. Amantadine produced a non-significant reduction in irritability compared to placebo at 28 and 60 days, according to the most problematic and aberrant items on the neuropsychiatric inventory (Hammond et al. 2015).

**Conclusion**

*There is conflicting evidence of the effects of Amantadine on reducing irritability and aggression in individuals with moderate-severe traumatic brain injury.*

**Amantadine requires further research before conclusions can be drawn on its effects on aggression.**

### 8.5.3 Anticonvulsants

Typically following a TBI there is diffuse injury with primary involvement in fronto-subcortical and temporolimbic regions. As a result, seizure disorders following TBI are not uncommon and may result in episodic lack of control. In the use of any medication, a balance must be struck between managing the behaviour and maintaining cognitive functioning. Thus, some anticonvulsants have been found to be a good alternative to antipsychotics and/or benzodiazepines in managing aggression, as they tend to have fewer cognitive side effects (e.g., sedation, confusion, memory impairment).

#### 8.5.3.1 Carbamazepine

Carbamazepine, an antiepileptic has been shown to successfully treat various seizure disorders and OCD. It has been suggested that carbamazepine may be effective in treating aggressive behaviour post TBI, offering an effective alternative to lithium (Azouvi et al. 1999).

**Individual Study**

**Table 8.11 Effects of Carbamazepine on Reducing Aggressive Behaviour Post ABI**

<table>
<thead>
<tr>
<th>Author/Year Country/ Study Design</th>
<th>Methods</th>
<th>Results</th>
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</thead>
<tbody>
<tr>
<td><strong>Azouvi et al.</strong> (1999) France Pre-Post N=10</td>
<td><strong>Population</strong>: TBI; Mean Age=33.7yr; Gender: Male=8, Female=2; Mean GCS Score=5.3; Mean Time Post Injury=58wk. <strong>Intervention</strong>: Carbamazepine (mean dose=9.47±2.9mg/kg/d) for 8wk. <strong>Outcome Measure</strong>: Neurobehavioural Rating Scale (NRS), Agitated Behaviour Scale (ABS), Katz Adjustment Scale (KAS) and Mini-Mental Status Examination (MMSE).</td>
<td>1. Dosage and blood work remained within clinical limits for epilepsy. 2. Total NRS-R and ABS scores showed significant improvement (p=0.02); improvements plateaued after 2wk. 3. At follow-up, significant improvements were shown for only the irritability (p&lt;0.01), and disinhibition (p&lt;0.05) portions of NRS-R. 4. Global NRS-R significantly decreased from baseline (p&lt;0.01). 5. No significant changes on MMSE were observed (p&gt;0.01).</td>
</tr>
</tbody>
</table>
Discussion
Azouvi et al. (1999) in an 8-week open drug trial administered carbamazepine (Tegretol) to 10 individuals with severe brain injury who had significant behavioural challenges that were interfering with care and/or family integration. Results indicated improvement on the behavioural scales at the first assessment (2 weeks), which were maintained only for the scales of irritability and disinhibition by the end of the trial; although, overall neurobehavioural and social functioning had improved. It should be noted that drowsiness was a frequent adverse event which limited the dosage being increased in 40% of the participants.

Conclusion

There is Level 4 evidence that carbamazepine decreases the incidence of aggressive behaviours following a traumatic brain injury.

Carbamazepine may decrease agitated behaviour post-traumatic brain injury.

8.5.3.2 Lamotrigine
The benefits of lamotrigine as an antiepileptic and mood stabilizer have been well established; however, its effectiveness as a mood stabilizer for patients with ABI has yet to be established (Gao & Calabrese 2005; Tidwell & Swims 2003).

Individual Study

Table 8.12 Effects of Lamotrigine on Reducing Aggressive Behaviour Post ABI

<table>
<thead>
<tr>
<th>Author/Year Country/Study Design</th>
<th>Methods</th>
<th>Results</th>
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<tbody>
<tr>
<td>Chahine &amp; Chemali (2006) Lebanon Case Series N=4</td>
<td><strong>Population:</strong> TBI; Mean Age=26yr; Gender: Male=4, Female=0. <strong>Intervention:</strong> Lamotrigine (range 125 to 300mg/d) to reduce inappropriate behaviours (e.g. laughing, impulsivity or verbal aggression). <strong>Outcome Measure:</strong> Frequency of crying, pathological laughing, behaviours of impulsivity, and seizures. Also, notes of depression.</td>
<td>1. All behaviours decreased once the individual was placed on lamotrigine. 2. Crying decreased, and inappropriate laughing ceased. 3. Impulsivity did not cease.</td>
</tr>
</tbody>
</table>

Discussion
Results from a single study, indicate that lamotrigine helps to reduce unwanted behaviours such as pathologic laughter but did not address impulsivity (Chahine & Chemali 2006). All four participants were on other medications to control for additional behaviours, however in each case these medications were eventually eliminated once lamotrigine was introduced. No formal outcome assessments were conducted making it challenging to draw conclusions from this study. Further research is needed.

Conclusion

There is Level 5 evidence to suggest that lamotrigine helps to reduce inappropriate behaviours post-traumatic brain injury.
8.5.3.3 Valproic Acid/Depakene
Valproic acid, an antiepileptic, has been used to successfully treat seizure disorders in both adults and children. Moreover, it has been used to treat bipolar, PTSD and mania (McElroy et al. 1987). It has also been found to reduce episodic explosiveness with an individual with TBI (Geracioti 1994).

Individual Study

Table 8.13 Effects of Valproic Acid on Reducing Aggressive Behaviour Post TBI

<table>
<thead>
<tr>
<th>Author/Year Country/Study Design</th>
<th>Methods</th>
<th>Results</th>
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<tbody>
<tr>
<td>Wroblewski et al. (1997) USA Case Series N=5</td>
<td>Population: TBI; Mean Age=38.2yr; Gender: Male=4, Female=1. Intervention: Valproic acid. Outcome Measure: Aberrant Behaviour Checklist.</td>
<td>1. Each patient was reviewed individually, with no cross-case comparisons. All showed a substantial reduction in target behaviours.</td>
</tr>
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</table>

Discussion

Wroblewski and colleagues (1997) examined the effects of valproic acid (Depakene) on reducing aggressive behaviour in a case series (N=5). Although the study reports that all patients showed a substantial reduction in challenging behaviour (i.e., outbursts, agitation, anger), no statistical analyses were carried out. Researchers relied on visual inspection of data and graphs were only presented for 3 of the 5 patients, which may bias results. Further, patients were also part of a specialized neurobehavioural unit, which may have contributed to the positive results.

Conclusion

There is Level 5 evidence that valproic acid decreases the incidence of aggressive behaviours.

Valproic acid may assist in the reduction of aggressive behaviours; however more research is needed.

8.5.3.4 Divalproex/Epival
Divalproex, another anticonvulsant, is believed to help reduce aggressive behaviours in individuals post TBI.
Individual Study

Table 8.14 Effects of Divalproex on Reducing Agitation Post ABI

<table>
<thead>
<tr>
<th>Author/Year Country/Study Design</th>
<th>Methods</th>
<th>Results</th>
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</table>
| Chatham Showalter & Kimmel (2000) USA Case Series N=29 | Population: TBI; Mean Age=48.2yr; Mean Time Post Injury=28.6d. Intervention: A retrospective chart review of patients receiving divalproex treatment in an attempt to reduce symptoms of agitation following injury. Symptoms of agitation included easily aggravated, escalating temper, biting, punching, restless, etc. Outcome Measure: Agitated Behaviour Scale (ABS). | 1. 8 patients had treatment with Divalproex (mean 714mg) leading to rapid resolution of symptoms to near total recovery. 2. For a second subgroup (n=18), progress notes prior to and during treatment demonstrated decreased and significant improvement in symptoms within 7d of receiving divalproex (mean dose 1,257mg). 3. Most patients were discharged to their homes (n=23) or to other community sites (n=4). |}

Discussion

Divalproex was used to treat symptoms of agitation in 29 patients with brain injuries (Chatham Showalter & Kimmel 2000). Symptoms decreased in the majority of patients, indicating that divalproex may be an effective treatment to reduce agitation following brain injury.

Conclusion

*There is Level 4 evidence that divalproex decreases the incidence of agitation post-traumatic brain injury.*

*Anticonvulsants may be used to decrease the incidence of agitated behaviour; however, more research is needed.*

8.5.4 Anti-Depressants

Two studies examined the effect of antidepressants on reducing agitation and/or aggression in patients with brain injuries (Kant et al. 1998; Mysiw et al. 1988). Kant et al. (1998) examined the effect of sertraline, a serotonin selective re-uptake inhibitor, on reducing aggression and irritability in patients with brain injury, whereas Mysiw et al. (1988) examined the effect of amitriptyline (a tricyclic antidepressant with both serotonergic and noradrenergic reuptake inhibition).

Individual Studies

Table 8.15 Effects of Sertraline and Amitriptyline on Reducing Aggression and Irritability Post ABI

<table>
<thead>
<tr>
<th>Author/Year Country/Study Design/PEDro/N</th>
<th>Methods</th>
<th>Outcome</th>
</tr>
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<tbody>
<tr>
<td>Mysiw et al. (1988) USA Pre-Post</td>
<td>Population: TBI; Mean Age=26.9yr; Gender: Male=43, Female=15. Intervention: Traditional behavioural techniques were used but if agitation interfered</td>
<td>1. 13 of 20 patients treated with amitriptyline experienced significantly reduced levels of agitation after 1wk (p&lt;0.001); decrease in agitation was</td>
</tr>
</tbody>
</table>
Discussion
Both studies showed potential to improve aggressive and agitated behaviour in patients with brain injuries. Kant et al. (1998) examined the effect of sertraline HCl (Zoloft) on reducing aggression and irritability in patients with closed head injuries of varying severities, two years post injury. The patients responded positively at both the four and eight week follow-ups, showing significant reduction in aggressive and irritable behaviour (Kant et al. 1998). The patients treated also had improvements in depression at week four. Mysiw et al. (1988) focused on 20 individuals who displayed agitation during their rehabilitation program and received amitriptyline. 70% of patients displayed significant reductions agitation within the first week (Mysiw et al. 1988). Both studies had similar limitations, those being small sample sizes and no true control groups.

Conclusion

There is Level 4 evidence that sertraline HCl can decrease the incidence of aggression and irritability.

There is Level 4 evidence that amitriptyline may be useful in reducing the incidence of agitated behaviour.

Sertraline HCl may be useful in reducing aggressive and irritable behaviours.

Amitriptyline may be used to decrease agitation.

8.5.5 Beta-Blockers
It has been suggested that Beta-blockers may improve agitation, anxiety and aggressive symptoms following brain injury, and reduce restlessness. Oftentimes, dosage is high, leaving patients vulnerable to adverse effects such as sedation, depression and lethargy, although it does not seem to negatively affect motor recovery post injury (Levy et al. 2005).
8.5.5.1 Pindolol

Pindolol is a beta-blocker unlike many others in that it exerts a partial agonist effect, providing a slight stimulation of the blocked receptor and maintaining a better resting sympathetic tone.

Individual Studies

Table 8.16 Effects of Pindolol on Behaviour Post ABI

<table>
<thead>
<tr>
<th>Author/Year</th>
<th>Country/Study Design/PEDro/N</th>
<th>Methods</th>
<th>Outcome</th>
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<tbody>
<tr>
<td>Greendyke &amp; Kanter (1986) USA RCT PEDro=7 N=9</td>
<td>Population: ABI; Mean Age=52yr; Gender: Male=9, Female=0; Mean Time Post Injury=7.8yr. Intervention: In a crossover design, patients received pindolol or a placebo capsules for the first half of study. The treatment group received 60mg/d of pindolol for 10d, increased up to 100mg. Groups were then crossed-over. Supplemental psychotropic medication was given as needed. Outcome Measure: Frequency of assaultive behaviour.</td>
<td>1. Significant reduction of assaultive episodes, need for supplemental medication and hostility were demonstrated during pindolol treatment (p&lt;0.05). 2. Significant improvements in patients’ willingness to communicate, and cooperation during treatment (p&lt;0.025) and significant reduction of stereotyped behaviours (p&lt;0.01).</td>
<td></td>
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PEDro=Physiotherapy Evidence Database rating scale score (Moseley et al., 2002).

Discussion

Greendyke and Kantor (1986) investigated the effectiveness of the beta-blocker, pindolol, for the improvement of behavioural disturbances. A significant reduction in behaviours that lead to assaults was demonstrated during treatment with pindolol, with the authors stating the optimal dose ranged between 40-60 mg per day. No therapeutic advantage was gained with doses beyond that but rather it lead to adverse events (Greendyke & Kanter 1986). Although the frequency of supplemented psychotropic medications was reduced in the pindolol group, these medications were still given and may have attributed to the reduction in assaultive episodes.

Conclusion

*There is Level 1b evidence that pindolol decreases aggression following brain injury based on one random control trial.*

Pindolol can decrease aggressive behaviour following brain injury.

8.5.5.2 Propranolol

Propranolol is a non-selective beta-blocker that has been used for the reduction of aggressive behaviours associated with compromised brain function. It is not known how this drug works to affect behaviour, however it appears to lack the serious cognitive and affective side effects of other medications or physical restraints used to treat agitation post injury (Levy et al. 2005).
Individual Studies

Table 8.17 Effects of Propranolol on Behaviour Post ABI

<table>
<thead>
<tr>
<th>Author/Year Country/Study Design/PEDro Score</th>
<th>Methods</th>
<th>Outcome</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Greendyke et al.</strong>&lt;br&gt; (1986) USA RCT PEDro=7 N=10</td>
<td><strong>Population:</strong> Mean Age=52yr; Gender: Male=9, Female=0; Mean Time Post Injury=7.8yr.&lt;br&gt; <strong>Intervention:</strong> Patients received long-lasting propranolol (520mg/d) or a placebo. After 11wk, the groups were crossed-over.&lt;br&gt; <strong>Outcome Measure:</strong> Assaultive Behaviour, Supplemental psychotropic medication, Daily Behaviour, Nurses Observation Scale for Inpatient Evaluation.</td>
<td>1. Significantly fewer assaults and attempted assaults occurred during the 11wk propranolol treatment as compared to the 11wk of placebo (p&lt;0.05).&lt;br&gt; 2. No significant changes in social interests, irritability or psychomotor retardation were noted. No abnormalities were noted on laboratory measures.</td>
</tr>
<tr>
<td><strong>Brooke et al.</strong>&lt;br&gt; (1992) USA RCT PEDro=7 N=21</td>
<td><strong>Population:</strong> TBI; Severity of Injury: GCS &lt;8.&lt;br&gt; <strong>Intervention:</strong> Patients randomized to either propanol (n=11; 60mg/d, max 420mg) or placebo (n=10).&lt;br&gt; <strong>Outcome Measure:</strong> Overt Aggression Scale.</td>
<td>1. Control group had more intense episodes of agitation than the treatment group (p&lt;0.05).&lt;br&gt; 2. No significant differences between the two groups in terms of agitation episodes/wk.&lt;br&gt; 3. More participants in the control group were placed in restraints during the study (p&lt;0.05).&lt;br&gt; 4. There were no differences between the two groups in the numbers receiving sedating drugs or drugs for agitation.</td>
</tr>
</tbody>
</table>

PEDro=Physiotherapy Evidence Database rating scale score (Moseley et al., 2002).

Discussion

Greendyke et al. (1986) investigated the effectiveness of the beta-blocker, propranolol, for the improvement of behaviour associated with brain disease in a randomized, crossover trial. Significantly fewer assaults and attempted assaults occurred during the 11-week propranolol treatment as compared to the placebo group. Of the nine patients, five showed marked improvement, two demonstrated moderate improvement, and two showed little or no improvement of assaultive behaviour. It should be noted that the participants also had severe dementia; therefore, this study was not used to draw conclusions for an ABI population as a whole. A later study by Brooke et al. (1992) found that propranolol was effective in reducing the intensity of the agitation but was not significantly more effective in reducing the number of episodes compared to a placebo.

Conclusion

There is Level 1b evidence that propranolol reduces the intensity of agitated symptoms following brain injury.

Propranolol may reduce the intensity of aggressive and agitated symptoms following brain injury.
8.5.6 Antipsychotics

8.5.6.1 Quetiapine (Seroquel)
Quetiapine has been used to reduce aggressive behaviour among those diagnosed with schizophrenia and Alzheimer’s disease (Volavka et al. 2004; Webb & Glueckauf 1994). A closer examination of its impact within a brain injury population is discussed below.

Individual Study

Table 8.18 Effects of Quetiapine on Aggressive Behaviour post ABI

<table>
<thead>
<tr>
<th>Author/Year Country/Study Design</th>
<th>Methods</th>
<th>Outcome</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Kim &amp; Bijlani (2006)</strong> USA Case series N=7</td>
<td><strong>Population:</strong> CHI; Mean Age=48.9yr; Gender: Male=4, Female=3; Mean Time Post Injury=23.1mo. <strong>Intervention:</strong> Patients received Quetiapine (50-100mg/d, max 800mg) Quetiapine daily in bedtime for the first week, then titrated every 3-4d to a maximum of 800mg for 6wk in total (dose ranged from 25 to 300mg). <strong>Outcome Measure:</strong> Overt Aggression Scale-Modified (OAS-M), Clinical Global Impression (CGI), Neurobehavioural Functioning Inventory, Repeatable Battery for the Assessment of Neuropsychological Status (RBANS).</td>
<td>1. Mean dose of Quetiapine was 110.7mg. As a result of the medication, subjects’ OAS scores were significantly reduced (p=0.002). 2. The CGI score significantly improved (p=0.002). 3. Significant improvements were also noted on the aggression subscale (p=0.036). 4. RBANS overall scores indicated a mean improvement of 8.02% (p=0.027).</td>
</tr>
</tbody>
</table>

Discussion
In one case series quetiapine assisted in helping to reduce aggressive behaviour in seven individuals (Kim & Bijlani 2006). They also noted significant improvements in the Overt Aggression Scale-Modified, the Clinical Global Impression scores, and the overall scores of the Repeatable Battery for the Assessment of Neuropsychological Status. Quetiapine may be considered as an alternative to haloperidol or chlorpromazine if additional research finds it is just as effective in treating aggressive behaviours without the side effects (Kim & Bijlani 2006).

Conclusion

_There is Level 4 evidence (from one small study) to suggest that quetiapine helps reduce aggressive behaviour._

_Although there is evidence to suggest that quetiapine does help reduce aggressive behaviour, more research is needed._

8.5.6.2 Ziprasidone
Ziprasidone has been approved for acute agitation in those diagnosed with schizophrenia. It has also been found to work in the treatment of acute mania, often associated with bipolar disorder. For those who sustain a TBI, the period of post traumatic amnesia (PTA), has been defined as a period where the individual is disorientated, and may lack the ability to learn new things and suffer from behaviour
alterations (Brooke et al. 1992). Researchers believe that these behaviour alternations may result from the individual’s lack of self-awareness which may be related to memory alterations that appear after the injury (Noé et al. 2007).

Individual Study

Table 8.19 Effects of Ziprasidone on Agitation Post ABI

<table>
<thead>
<tr>
<th>Author/Year Country/Study Design</th>
<th>Methods</th>
<th>Outcome</th>
</tr>
</thead>
<tbody>
<tr>
<td>Noe et al. (2007) USA Case Series N=5</td>
<td>Population: TBI; Mean Age=26.8yr; Gender: Male=3, Female=2; Mean Time Post Injury=54.6d; Mean GCS Score=6. Intervention: Ziprasidone (30-80mg/d for 35-68d) was given to participants. Outcome Measure: Agitation Behaviour Scale (ABS).</td>
<td>1. Mean dose of the drug was 52.8mg/d. 2. Scores on the ABS decreased within the first 14d (27.3 to 18). 3. Scores on the disinhibition portion of the ABS decreased from 28.6 to 17.1, while scores on the aggressiveness subsection of the scale decreased from 26.1 to 20.4. 4. No side effects were noted.</td>
</tr>
</tbody>
</table>

Discussion

Noe et al. (2007) studied individuals who were still in PTA stage at admission to rehabilitation. Within these participants, a decrease in agitation scores was reported during the first two weeks of ziprasidone administration. It was also noted that all who participated tolerated the medication with no clinical side effects observed. A larger RCT would be beneficial before any firm conclusions are made.

Conclusion

*There is Level 4 evidence from one study to suggest that ziprasidone assists in the controlling of agitation post-traumatic brain injury.*

*Ziprasidone in one small study has been shown to assist in the controlling of agitation; however more research is needed.*

8.5.6.3 Lithium Carbonate

Lithium carbonate has been used for many years in the treatment of mania and bipolar disorder (Kim 2002). It has been suggested that mood disorders, such as mania, occurring after the TBI, may contribute to the development of aggression (Kim 2002; Wroblewski et al. 1997). In the search for a pharmacological agent that reduces aggression following TBI with limited side effects, in comparison to antipsychotics and benzodiazepines, lithium has been tried. Lithium carbonate also functions as a mood stabilizer.
Individual Studies

Table 8.20 Effects of Lithium Carbonate on Aggressive Behaviour Post ABI

<table>
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<tr>
<th>Author/Year Country/Study Design</th>
<th>Methods</th>
<th>Outcome</th>
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<tbody>
<tr>
<td>Glenn et al. (1989) USA. Case Study N=10</td>
<td>Population: TBI=8, CVA=2; Mean Age=31.6yr; Gender: Male=5, Female=5. Intervention: Patients showing mood disorders, aggressive, combative, self-destructive behaviour and/or affective instability were administered lithium. Outcome Measure: Observed improvement.</td>
<td>1. 5 participants showed a significant improvement in rehab programs with no decrease in motor or cognitive performance; 1 showed moderate response, 1 improved dramatically but regressed after 7wk. 2. 4 regressed after medications stopped. 3. 3 participants had neurotoxic side effects.</td>
</tr>
</tbody>
</table>

Discussion

Lithium carbonate was used in a series of case reports with ten individuals with either TBI or stroke (Glenn et al. 1989). Glenn et al. (1989) reported favourable outcomes for the majority of patients (i.e., a decrease in observed aggressive, combative, or self-destructive behaviour or severe affective instability). However, this study highlights that there is a high risk of potential neurotoxicity among individuals with brain injuries, specifically in combination with neuroleptic drugs.

Conclusion

There is Level 5 evidence to suggest that an antimanic agent (lithium carbonate) reduces aggressive/agitated behaviour following a brain injury.

Lithium may reduce behavioural problems but is associated with a high risk of neurotoxicity.

8.5.7 Sexually Disinhibited Behaviour

Sexual dysfunction following TBI has been reported to occur in at least 50% of patients (Emory et al. 1995). Hypersexuality is less common than hyposexuality (decreased libido) but results in a greater negative effect for the individual and results in a great burden of care by limiting independence. Hypersexual behaviour can encompass a range of behaviours, from indiscriminate sexual advances, promiscuity, and exhibitionism, to assault and/or rape (Mania et al. 2006). A recent study revealed inappropriate sexual talk to be the most common inappropriate sexual behaviour in a sample of patients with TBI (Simpson et al. 2013). Treatment for sexual offenders without brain injuries has included pharmacological intervention and or counselling and education. Typically, medication is used to reduce the sexual drive, but it is unclear if it has effect on cognitive processing (i.e., preservative thoughts regarding sex).
Individual Studies

Table 8.21 Effects of Depo-Provera on Sexually Aggressive Behaviour Post ABI

<table>
<thead>
<tr>
<th>Author/Year/ Country/Study design</th>
<th>Methods</th>
<th>Outcome</th>
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<tbody>
<tr>
<td><strong>Population:</strong> TBI; Mean Age=17.5yr; Gender: Male=8, Female=0. <strong>Intervention:</strong> Weekly IM injections of Depo-Provera (400mg) in conjunction with directive, individual-specific counseling for 6mo. <strong>Outcome Measure:</strong> Incidence of hypersexual behaviour, change in testosterone level.</td>
<td>1. Family members report all subjects stopped aberrant behaviour while taking medication. 2. Blood work revealed a drop in testosterone from 834 to 85mg/dL; 3 subjects returned to previous patterns after stopping medication (due to inconsistent family support). 3. 3 subjects dramatically improved and did not stop medication.</td>
<td><strong>Emory et al.</strong> (1995) USA Case Series N=8</td>
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<tr>
<th>Author/Year/ Country/Study design</th>
<th>Methods</th>
<th>Outcome</th>
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<tr>
<td><strong>Population:</strong> TBI; Mean Age at Injury=32.7yr; Gender: Male=250, Female=257; Median Time Post Injury=2.2yr. <strong>Intervention:</strong> Patients receiving community rehabilitation completed the following assessments. <strong>Outcome Measure:</strong> Overt Behaviour Scale, Disability Rating Scale, Sydney Psychosocial Reintegration Scale-2, Health of the Nation Outcome Scale- ABI, Care and Needs Scale.</td>
<td>1. Point prevalence rate of inappropriate sexual behaviour over 3mo was 8.9%. 2. Inappropriate sexual talk comprised 57.9% of all inappropriate sexual behaviours, followed by genital and non-genital touching behaviours (29.8%) and exhibitionism/public masturbation (10.5%). 3. In 43/45 cases, inappropriate sexual behaviours were accompanied by other challenging behaviours (e.g. inappropriate social behaviours, aggression, etc.). 4. Individuals who were younger, and had more severe injuries were more likely to display inappropriate sexual behaviours. 5. Patients displaying inappropriate behaviours had higher levels of challenging behaviours overall, lower levels of social participation and more neuropsychiatric impairments.</td>
<td><strong>Simpson et al.</strong> (2013) Australia Cross-Sectional N=507</td>
</tr>
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</table>

Discussion

As shown by the findings from Simpson et al. (2013), inappropriate sexual behaviour is a concern for individuals post injury; specifically, verbal inappropriateness among younger and more severely injured individuals. In a retrospective study, Depo-Provera, an anti-androgen drug, was evaluated in terms of its efficacy for controlling sexual aggression in eight males with TBI experiencing onset of sexual aggression three years post injury (Emory et al. 1995). Weekly intramuscular injections of Depo-Provera (400 mg) in conjunction with monthly psychoeducational counseling resulted in a cessation of hypersexual behaviour and reduced testosterone levels. Three subjects re-offended when the drug was stopped, three remained on it and two stopped taking the drug and had maintained cessation of hypersexual behaviour.

Conclusion

*There is Level 4 evidence that Depo-Provera and counselling reduces sexually aggressive behaviour.*
**Medroxyprogesterone intramuscularly may reduce sexual aggression.**

8.5.8 Methotrimeprazine
Methotrimeprazine (Nozinan) is a psychotropic medication. It has antipsychotic (mediated by dopamine blocking), tranquilizing, and analgesic properties. It appears to have an effect on opiate (pain) receptors as well (Maryniak et al. 2001).

**Individual Studies**

<table>
<thead>
<tr>
<th>Author/Year/Country/Study Design</th>
<th>Methods</th>
<th>Outcomes</th>
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<tbody>
<tr>
<td>Maryniak et al. (2001) Canada Case Series N=120</td>
<td>Population: TBI=95, ABI=25; Mean Age=37.8y; Gender: Male=89, Female=31. Intervention: Retrospective review of patients attending an inpatient ABI rehabilitation unit. Patients administered methotrimeprazine (MTZ) were analyzed. Outcome Measure: Agitated Behaviour Scale.</td>
<td>1. 58% had agitation but 56 patients were treated with MTZ (10-25mg, 4×/d) with a mean length of treatment of 41.9d. 2. MTZ, for the most part (96% of patients), was both safe and effective for controlling agitation.</td>
</tr>
</tbody>
</table>

**Discussion**
The oral administration of methotrimeprazine (MTZ) for agitation was evaluated in a retrospective chart review of 56 patients during inpatient rehabilitation (Maryniak et al. 2001). This was the first report on MTZ’s use in treating agitation after ABI and the authors found that in most cases MTZ was both safe and effective for controlling agitation. No standardized outcome measures were used within this study, and there was no control group; therefore, a more rigorous study examining the safety and efficacy of MTZ within an ABI population is necessary before a level of evidence statement can be provided.

**Conclusion**

*There is Level 4 evidence that methotrimeprazine is safe and effective for controlling agitation after an acquired brain injury.*

*Methylphenidate may be safe for controlling agitation following an acquired brain injury.*

8.5.9 Methylphenidate
One RCT examined the effect of methylphenidate on the control of anger following a brain injury (Mooney & Haas 1993).
Individual Studies

Table 8.23 Effects of Methylphenidate on Anger and Attention Post ABI

<table>
<thead>
<tr>
<th>Author/Year/Country/Study design/PEDro Score</th>
<th>Methods</th>
<th>Outcome</th>
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<tbody>
<tr>
<td><strong>Mooney &amp; Haas (1993)</strong>&lt;br&gt;USA&lt;br&gt;RCT&lt;br&gt;PEDro=5&lt;br&gt;N=38</td>
<td><strong>Population</strong>: TBI; Mean Age=29.45yr; Gender: Male=38, Female=0; Mean Time Post Injury=27.08mo. <strong>Intervention</strong>: Patients in the treatment group (n=19) received methylphenidate (30mg/d). Those in the control group received a placebo (n=19) for 6wk. <strong>Outcome Measure</strong>: State-Trait Anger Scale, the Belligerence cluster score from the Katz Adjustment Scale and the Anger-Hostility factor score of the Profile of Mood States.</td>
<td>1. Following statistical control over the possible bias (difference in baseline anger scores), there was a significant main effect for the drug treatment (p&lt;0.001).&lt;br&gt;2. Analyzing the anger outcome measures, a significant drug by time interaction effect was noted (p=0.002).</td>
</tr>
</tbody>
</table>

PEDro=Physiotherapy Evidence Database rating scale score (Moseley et al. 2002)

Discussion

In a RCT, Mooney and Haas (1993) demonstrated that methylphenidate helped to significantly reduce anger following brain-injury as demonstrated using several anger outcome measures. Despite the differences between the groups on one anger measure, a significant group main effect of the drug treatment was demonstrated.

Conclusion

*There is Level 2 evidence (from one random control trial) to suggest that treatment with methylphenidate following brain injury can significantly reduce anger.*

**Methylphenidate is effective in reducing anger following a brain injury.**

8.5.10 Neuroleptic Butyrophenones

8.5.10.1 Droperidol (Inapsine)

Droperidol is a butyrophenone antipsychotic agent that closely resembles haloperidol in structure. It has been used for the treatment of psychosis in Europe (Stanislav & Childs 2000).

Individual Studies

Table 8.24 Effects of Droperidol for Improving Behaviour Post ABI

<table>
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<tr>
<th>Author/Year/Country/Study design</th>
<th>Methods</th>
<th>Outcome</th>
</tr>
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<tbody>
<tr>
<td><strong>Stanislav &amp; Childs (2000)</strong>&lt;br&gt;USA&lt;br&gt;Pre-Post&lt;br&gt;N=27</td>
<td><strong>Population</strong>: TBI; Gender: Male=21, Female=6. <strong>Intervention</strong>: Intramuscular injection of droperidol administered as needed to relieve agitation. <strong>Outcome Measure</strong>: Episodes of agitation.</td>
<td>1. Mean dose was 3.25mg; a single dose reduced agitation in 96% of patients.&lt;br&gt;2. The time to achieve calming following episodes of agitation was significantly shortened with droperidol compared to...</td>
</tr>
</tbody>
</table>
Discussion
When an individual is agitated, not only is the effectiveness of the medication administered important but also the time it takes to have a calming effect. One retrospective controlled trial found that a single-dose of droperidol calmed patients displaying agitated behaviour faster than other drugs (haloperidol, lorazepam, and diphenhydramine) (Stanislav & Childs 2000). The study also found that droperidol calmed individuals without heavily sedating the patients like some of the comparative medications did. It is worth noting however that a large proportion of the sample had psychiatric co-morbidities, this should be kept in mind when generalizing the findings.

Conclusion

There is Level 4 evidence that administration of a single-dose of droperidol calms agitated patients with acquired brain injuries more quickly than other agents.

Droperidol may be an effective agent for calming agitated patients.

8.5.10.2 Haloperidol

Haloperidol is a psychotropic drug found to reduce agitation. It also blocks or disrupts dopamine receptors. Thus, while it improves agitation, there is a theoretical concern that it may impede recovery by reducing arousal.

Individual Studies

Table 8.25 Effects of Haloperidol on Agitation Post ABI

<table>
<thead>
<tr>
<th>Author/Year/Country/Study design</th>
<th>Methods</th>
<th>Outcomes</th>
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</thead>
<tbody>
<tr>
<td>Rao et al. (1985) USA Case Series N=26</td>
<td>Population: Severe TBI; Age Range=16-48yr. Intervention: Retrospective review of individuals whose agitation was treated with haloperidol (n=11; 2-15mg/d) and those who were not (n=15). Outcome Measure: Patient Evaluation Conference Systems.</td>
<td>1. Those treated had a longer length of PTA (p&lt;0.03). 2. No statistically significant differences were shown between those who were and were not treated in terms of independent living at discharge (64% versus 60%, respectively) or independence in managing behaviour (40% versus 60%). 3. 3 of those non treated obtained independence in intellectual skills but none of the treated patients did this.</td>
</tr>
</tbody>
</table>

Discussion

In a retrospective chart review, agitation was managed in eleven patients with haloperidol and in fifteen patients without haloperidol (Rao et al. 1985). No significant differences were found between the two groups with regards to success of rehabilitation outcome; however, none of the patients in the treatment group obtained independence in intellectual skills (Rao et al. 1985).
**Conclusion:**

*There is Level 4 evidence that haloperidol does not have a negative effect on the success of rehabilitation.*

*Haloperidol appears to have little negative effect on recovery following traumatic brain injuries.*

**Summary Regarding the Use of Pharmaceuticals to Reduce Aggressive Behaviour**

The use of pharmacological agents can help to prevent injury to the patient and others. An ideal medication should have, “a rapid onset of action, achieve maximal effect with a single dose, cause minimal adverse effects, and allow the patient to resume normal daily activities as quickly as possible without causing protracted sedation or cognitive impairments” (pg 263-4, Stanislav & Childs 2000). A fairly consistent limitation across the studies in this section is the lack of a control group. Ideally, the efficacy of pharmacological interventions for agitation would be studied using a randomized, double-blinded, placebo design; however, few of these trials have been conducted (Levy et al. 2005). When investigating aggressive symptomology following brain injury there is difficulty in comparing across studies, and different treatment types, due to the lack of consistency in how aggression is measured. For example, some studies used standard outcome measures while others relied on reported/observational behaviour ratings.

**Conclusion**

*There is limited evidence that pharmacological interventions can reduce verbal, physical and/or sexual aggressive behaviours. Rigorous, randomized controlled trials are needed.*

**8.5.11 Behavioural Management Post ABI**

Common sequelae to brain injury are behavioural disturbances that impact the patients’ relationships and recovery. In some cases, individuals with brain injury develop behavioural difficulties that impact their compliance with rehabilitation, resulting in early discharge and/or limited participation in rehabilitation activities (Alderman 1991; Alderman et al. 2013). When challenging behaviours take the form of aggressive acts, this may prevent or decrease functional gains in neurorehabilitation (Alderman et al. 1999). In a cross-sectional study of 69 subjects admitted to a brain injury unit, Lequerica et al. (2007) found an inverse relationship between agitation and the individual’s engagement in physical and occupational therapy.

It is difficult to compare across studies when evaluating interventions using behavioural management techniques as they are tailored to the needs/requirements of the person being treated. Studies may examine diverse techniques to manage challenging behaviours following an ABI, including antecedent controls, positive reinforcement, and token economies, whereas other studies have examined the efficacy of specific training programs (i.e., anger management, social skills training, etc.) in reducing agitation/aggression.
8.5.12 Specific Behavioural Techniques

Behavioural techniques have been used for many years with a variety of disorders. Techniques are often used to teach new skills, socially appropriate behaviour and improve independent functioning. Teaching behavioural techniques does not involve coercing people to do anything against their will; instead it encourages positive behaviours through an individualized approach. Behavioural analysis examines the relationship between events and behaviour with the goal of increasing social interactions and independence (Ashley et al. 1995). In the past, the alternative to behavioural strategies has been sedation, physical restraint, and/or institutionalisation. Not only are behavioural techniques applicable in a variety of settings and with a variety of behaviours, but they also address the main goals of rehabilitation – the development of functional life skills (Jacobs 1993).

Individual Studies

Table 8.26 Effects of Antecedent Behavioural Interventions on Reducing Aggressive Behaviour Post ABI

<table>
<thead>
<tr>
<th>Author/Year/Country/Study design</th>
<th>Methods</th>
<th>Outcome</th>
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<tbody>
<tr>
<td><strong>Aboulafia-Brakha et al.</strong> (2013) Switzerland Pre-Post</td>
<td>N\text{\textsubscript{final}}=10, N\text{\textsubscript{final}}=9</td>
<td>1. Significant improvement in feelings of aggression, as measured by AQ-12, was shown from pre-treatment to follow-up (p=0.02). 2. No significant improvement on AQ-12 was found pre to post-treatment (p=0.84), or from post-treatment to follow-up (p=0.57). 3. No significant improvements were shown for UPPS Impulsive Behaviour Scale, FrSBe, EQ, HADS, or SF-36 between pre- and post-treatment (p&gt;0.05).</td>
</tr>
<tr>
<td><strong>Hart et al.</strong> (2012) USA Pre-Post</td>
<td>N=10</td>
<td>1. The group overall were of low to average intelligence and were found to have significant executive dysfunction. 2. Following the intervention, self-reports of anger declined significantly (p&lt;0.03).</td>
</tr>
<tr>
<td><strong>Wesolowski et al.</strong> (1999) USA Pre-Post</td>
<td>N=3</td>
<td>1. Number of unauthorized breaks for each participant decreased with the introduction of “mini” breaks. 2. This decrease in the number of unauthorized breaks persisted to the 6-week follow up period.</td>
</tr>
<tr>
<td><strong>Schlund &amp; Pace</strong> (1999) USA Pre-Post</td>
<td>N=3</td>
<td>1. Variability and frequency of maladaptive behaviour generally decreased from</td>
</tr>
<tr>
<td>Author/Year/ Country/Study design</td>
<td>Methods</td>
<td>Outcome</td>
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</table>
| **Pre-Post**
| N=3 | Injury=5.7yr.  
Intervention: Systematic feedback was provided on patients’ frequency of maladaptive behaviour  
Outcome Measure: Cognistat. | baseline (2 to 5.1/wk) to completion (0.18 to 1.8/wk). |
| **Burke et al.** (1988)
| USA
| Pre-Post
| N=5 | Population: TBI; Mean Age=23.2yr; Gender: Male=5, Female=0.  
Intervention: Behaviour therapy with emphasis on reinforcement and antecedent conditions.  
Outcome Measure: Number of physically aggressive behaviours. | 1. Measurements at 1wk post treatment showed a 97% decrease in aggressive behaviour from baseline levels; 100% by the third week.  
2. There was a significant reduction in behaviour at all time-points compared to baseline (p<0.001).  
3. No incidents of aggression were recorded during a 6mo follow-up. |
| **Eames & Wood** (1985)
| UK
| Pre-Post
| N=24 | Population: ABI=22, Stroke=1, Other=1; Mean Age=26.8yr; Gender: Male=18, Female=6; Mean GCS Score=7.8; Mean Time Post Injury=44.7mo.  
Intervention: Specialized TBI unit that used a wide range of physical, CBT, occupational and social techniques based on positive reinforcement and a token economy.  
Outcome Measure: Patient Placement. | 1. More than 2/3 of patients had improved placements after treatment; only one person had a substantial improvement.  
2. Fewer than 1/3 of patients made no change, and no one was demoted to a worse setting. |
| **Feeney & Ylvisaker** (1995)
| USA
| Case Study
| N=3 | Population: Severe TBI; Mean Age=18.3yr.  
Intervention: A-B-C-A designed antecedent behavioural and cognitive intervention comprised of photographic and written cues.  
Outcome Measure: Aberrant Behaviour Checklist (ABC), and percentage of assigned work completed. | 1. All three cases showed a sharp improvement in completed work while under therapy, a decrease in aggressive behaviours and ABC ratings indicated decreased intensity.  
2. Subjects 1 and 2 showed a mild increase in aggressive behaviours with written cues, which decreased when substituted with photographic cues. |
| **Alderman et al.** (1999)
| UK
| Case Study
| N=3 | Population: TBI; Mean Age=39.7yr; Gender: Male=2, Female=1.  
Intervention: Behaviour modification intervention.  
Outcome Measure: Overt Aggression Scale-Modified for Neurorehabilitation. | 1. There was a downward trend in the mean frequency of shouting per minute over time (p<0.01) following the initial intervention and decreased significantly from baseline following the second intervention (p<0.01). |

**Discussion**

Different behavioural interventions have been trialed in hopes of reducing aggressive behaviour. One such intervention is a psychoeducational treatment program called anger self-management training, for irritability and anger (Hart et al. 2012). The 8 session program was designed to help the individual identify anger signals, and to learn specific problem solving skills. Following treatment, anger declined significantly (p<0.03) (Hart et al. 2012). Another success was an antecedent behavioural intervention, by structuring the environment with high support and then reducing it was able to significantly reduce aggressive behaviour (Feeney & Ylvisaker 1995). Aboulafia et al. (2013) used CBT with emphasis on reduction of aggression and found that self-reported aggressive behaviours were significantly fewer from pre-intervention to 4-5 month follow-up. This finding aligns with a review by Waldron et al (2013) reporting that CBT is efficacious at reducing symptomology when the therapy is targeted for a specific problem (e.g. aggression). In summary, a recent meta-analysis for behavioural interventions discovered an overall substantial reduction in aggressive behaviours for single and group-based therapy (Byrne & Coetzer 2016).
Three studies explored the impact of systematic data based feedback on maladaptive behaviour. Schlund and Pace (1999) demonstrated that by using frequency data as feedback (as opposed to only verbal-based feedback), the occurrence of “maladaptive” behaviour could be reduced. However, their group consisted of three mildly cognitively impaired individuals attending a medical rehab program five days a week. Maladaptive behaviours consisted of pseudoseizures, non-compliance with rules, verbal aggression and sexually inappropriate behaviour. Wesolowski et al. (1999) utilized a “non-contingent escape” paradigm (i.e., planned mini-breaks in work periods) to increase compliance in three TBI clients in order to effect positive change with vocational placement. Burke et al. (1988) used a program that was structured so that positive behaviours, that were incompatible with aggression, would be more likely to occur, thereby decreasing aggressive behaviour. Percentage of change scores from baseline revealed success.

Eames et al. (1985) examined the quality of life of 24 severely injured ABI patients following intensive behavioural treatment utilizing a token economy (admission to a structured unit). Results indicated that generally the quality of life had improved and was maintained, as measured by improved relationships with care-givers and an improvement in living arrangements. However, when examining results reported, actual behaviours of aggression, sexual inappropriate behaviours, drive/motivation and odd behaviours increased in occurrence from discharge to follow-up.

Conclusion

There is Level 4 evidence to suggest that anger self-management training is effective in reducing irritability and anger after a traumatic brain injury.

There is Level 4 evidence that behavioural approach using antecedent management and/or feedback of consequences reduces undesirable behaviour (e.g., aggression/agitation).

Anger self-management training is effective in teaching those with a traumatic brain injury identify anger signals and develop more appropriate ways of dealing with anger and frustration.

Cognitive Behavioural Therapy with focus on anger and aggression management may be effective at reducing aggressive behaviours.

Antecedent management and/or feedback of consequences may reduce undesirable behaviour.

8.5.13 Multi-intervention Training Programs

Training programs that combine a number of behavioural interventions have been utilized with some success. For example, anger management, social skills, and coping skills training programs have been used in the past to alleviate aggressive/agitated behaviour in individuals with an ABI.
Individual Studies

Table 8.27 Effects of Training Programs on Alleviating Aggressive Behaviour Post ABI

<table>
<thead>
<tr>
<th>Author/Year/ Country/Study design /PEDro Score</th>
<th>Methods</th>
<th>Outcome</th>
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<tbody>
<tr>
<td><strong>McDonald et al. (2008)</strong>&lt;br&gt; Australia RCT PEDro=6 N=39</td>
<td><strong>Population:</strong> TBI; Gender: Male=28, Female=11. <strong>Treatment Group (n=13):</strong> Mean Age=35.5yr; Mean Time Post Injury=4.0yr. <strong>Social Group (n=13):</strong> Mean Age=34.3yr; Mean Time Post Injury=4.3yr. <strong>Intervention:</strong> Participants were randomly allocated to control (non-therapeutic social group; n=13), waitlist (deferred treatment group; n=13) or the social skills group (treatment group; n=13). Those in the skills training group attended 12wk program of group and individual sessions totaling 4hr/wk. Control group was subjected to 4hr/wk for 12wk of social activities only.&lt;br&gt;<strong>Outcome Measure:</strong> Partner Directed Behaviour Scale (PDBS), Personal Conversational Style Scale, Depression Anxiety and Stress Scale, The Awareness of Social Inference Test.</td>
<td>1. Results indicate no interaction effects for the social group relative to the waitlist group.&lt;br&gt;2. Those in the social skills training group made significant improvement on the PDBS scale compared to the placebo and waitlist group (p&lt;0.004).&lt;br&gt;3. Changes were not noted for any group when looking at social functioning and social participation post treatment.&lt;br&gt;4. Treatment effects were found to be modest at best and limited to direct measures of social behaviour.</td>
</tr>
<tr>
<td><strong>Carnevale et al. (2006)</strong>&lt;br&gt; USA RCT PEDro=5 N=37</td>
<td><strong>Population:</strong> TBI=24, ABI=13; Mean Age=40.5yr; Gender: Male=28, Female=7; Mean Time Post Injury=7.6yr. <strong>Intervention:</strong> Control group (n=12) received no treatment, Education group (n=13) received education only, and Natural Setting Behaviour Management (NSBM) group (n=12) received both education and an individualized target-behaviour program.&lt;br&gt;<strong>Outcome Measure:</strong> Questionnaires on Resources and Stress (QRS), Maslach Burnout Inventory (MBI), Neurobehavioural Functioning Inventory Revised (NFI-R).</td>
<td>1. The NSBM had more improvement in behaviour than the other two groups at 30wk (p&lt;0.002).&lt;br&gt;2. A significant difference was noted between the education group and the NSBM group (p&lt;0.04).</td>
</tr>
<tr>
<td><strong>Anson &amp; Ponsford (2006a)</strong>&lt;br&gt; Australia RCT PEDro=5 N=31</td>
<td><strong>Population:</strong> TBI; Gender: Male=26, Female=5; Group A (n=15): Mean Age=38.9yr; Mean Time Post Injury=755.8d; Group B (n=16): Mean Age=37.8yr; Mean Time Post Injury=340.8d. <strong>Intervention:</strong> For Group A (n=15), baseline phase was 5wk, followed by 5wk of intervention, and a 5wk follow-up phase. For Group B (n=16), baseline was 10wk, followed by 5wk of intervention and a 10wk follow-up phase. The CSG consisted of 10 group sessions and ran for 90min 2×/wk.</td>
<td>1. No significant changes in anxiety or self-esteem scores were noted following the CSG (p&gt;0.05).&lt;br&gt;2. Although levels of depression and psychosocial dysfunction were significantly different between the two groups (p&lt;0.05) participation in the CSG did not have an effect on their scores.&lt;br&gt;3. Both groups significantly increased their adaptive coping skills following the CSG (p&lt;0.01).</td>
</tr>
<tr>
<td><strong>Medd &amp; Tate (2000)</strong>&lt;br&gt; Australia RCT PEDro=5</td>
<td><strong>Population:</strong> TBI; Gender: Male=14, Female=2. <strong>Treatment Group (n=8):</strong> Mean Age=35.88yr; Mean Time Post Injury=37.25mo; <strong>Waiting List Group (n=8):</strong> Mean Age=34yr; Mean Time Post Injury=37.25mo.</td>
<td>1. TREAT group had significantly higher pre-intervention levels of Anger Expression-Out (AX-O) than the WAIT group (p=0.004).</td>
</tr>
</tbody>
</table>
### Module 8: Mental Health Issues Post ABI

<table>
<thead>
<tr>
<th>Author/Year/ Country/Study design /PEDrO Score</th>
<th>Methods</th>
<th>Outcome</th>
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<tbody>
<tr>
<td>N=16</td>
<td>Injury=74.25mo. &lt;br&gt; <strong>Intervention:</strong> Participants were randomly allocated to either the treatment (TREAT; n=8) or the waitlist group (WAIT; n=8). The TREAT group received 5-8 individualized sessions of components based on the Commonwealth Rehabilitation Service Anger Management Program. &lt;br&gt; <strong>Outcome Measure:</strong> State-Trait Anger Expression Inventory, Hospital Anxiety and Depression Scale (HADS), Self-esteem Inventories (SEI), Patient Competency Rating Scale (PCRS).</td>
<td>2. TREAT group showed a greater improvement in AX-O and trait anger, pre and post treatment, compared to the WAIT group (p=0.006 and p=0.054, respectively).&lt;br&gt;3. No significant differences were found for the HADS, SEI, or the PCRS between groups.</td>
</tr>
<tr>
<td>O'Leary (2000) USA Post-test N=5</td>
<td><strong>Population:</strong> ABI; Age Range=21-42yr; Gender: Male=5, Female=0; Time Post Injury Range=4mo-5yr. &lt;br&gt; <strong>Intervention:</strong> 10wk training program for anger management and coping skills through the use of written materials, role-play, audiotapes, group discussions and lectures. &lt;br&gt; <strong>Outcome Measure:</strong> Frequency of verbal and physical aggression.</td>
<td>1. Training reduced the number of incidents of both verbal and physical aggression for all participants.</td>
</tr>
<tr>
<td>Brotherton et al. (1988) USA Case Series N=4</td>
<td><strong>Population:</strong> CHI; Mean Age=23.5yr; Gender: Male=3, Female=1; Mean Time Post Injury=5.75yr. &lt;br&gt; <strong>Intervention:</strong> Social skills training program comprised of self-manipulation, speech dysfunctions, personal attention, reinforcing feedback, and positive statements. &lt;br&gt; <strong>Outcome Measure:</strong> Self-manipulation, posture, speech dysfluencies, personal attention.</td>
<td>1. The intervention was effective in 3 of 4 patients treated (however, not all behaviours were equally amenable to treatment).&lt;br&gt;2. Behaviours showing clear training effects also showed good maintenance 1yr after training.</td>
</tr>
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</table>

**PEDrO=Physiotherapy Evidence Database rating scale score (Moseley et al., 2002).**

**Discussion**

A RCT conducted by McDonald et al. (2008) compared social skills training, social activity, and a control group. Those in the social skills group showing a positive improvement in behaviour compared to the other interventions but the treatment effect was modest at best. Therefore, improving social behaviour, changing social perceptions, and improving mood and self-esteem seem to be somewhat influential in improving behaviour (McDonald et al. 2008). In reviewing the studies education alone was not effective in improving behaviour; however, education in combination with other interventions resulted in positive behavioural change. Carnevale et al. (2006) found that at 30 weeks following treatment, significant changes in behaviour were apparent for participants receiving an individualized program and education compared to education alone. Other interventions have been shown to benefit individuals post injury with alleviating aggression, such as an anger management therapy program (Medd & Tate 2000), a combination of anger management and coping skills training (O'Leary 2000) and social skills training (Brotherton et al. 1988); although the evidence is weak.
Conclusions

There is Level 1b evidence that social skills training has a limited impact on changing inappropriate behaviours and mood disturbances of those who have sustained a severe traumatic brain injury.

There is Level 2 evidence that community based program combining education and an individualized behaviour plan (e.g., Natural Setting Behaviour Management intervention) helps to change behaviour.

There is Level 2 evidence that participating in a Coping Skills Group assists in improving adaptive coping in the long term.

There is Level 2 evidence that anger management reduces aggressive behaviour.

Anger management and social skills training reduce aggressive behaviour.

8.5.14 Music Therapy
Music therapy is an approach that “consists of using music therapeutically to address physical, psychological, cognitive and/or social functioning for patients of all ages” (American Music Therapy Association 2004). It was first used with World War I veterans in hospital and was formally recognized as a therapeutic tool in 1950. Music therapy has been used with a variety of patients (neurological, psychiatric, medical, pervasive and developmental disorders) and has been found to result in physiological changes (e.g. respiration, blood pressure, heart rate, decrease cortisol levels and increase endorphins) and increased wellbeing. More recently music therapy has been used with patients with TBI to decrease agitation.

Individual Studies

Table 8.28 Effects of Music Therapy on Agitation Post ABI

<table>
<thead>
<tr>
<th>Author/Year/Country/Study Design</th>
<th>Methods</th>
<th>Outcome</th>
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<tbody>
<tr>
<td><strong>Formisano et al. (2001)</strong></td>
<td><strong>Population:</strong> TBI=18, Other=16; Mean Age=35.94yr; Gender: Male=17, Female=17; GCS Score=8. <strong>Intervention:</strong> Music Therapy treatment based on Nordoff and Robbins, performed 3x/wk for 20-40min. Evaluation occurred 6 different times. <strong>Outcome Measure:</strong> Glasgow Outcome Scale, Disability Rating Scale (DRS), Coma Recovery Scale (CRS).</td>
<td>1. During music therapy patients showed a reduction in undesired behaviours. 2. No improved interaction with the environment was recorded (DRS, CRS). 3. Positive effects reported in 27 of 34 patients 1mo after starting treatment and at follow-up.</td>
</tr>
</tbody>
</table>

Discussion

One study, Formisano et al. (2001) reported that music therapy had a beneficial effect in reducing post-coma agitation and inertia in 62% of their subjects in a slow-to-recover group one month after starting music therapy. More research is needed.
Conclusions

There is Level 4 evidence that music therapy reduces psychomotor agitation post coma following a severe traumatic brain injury in a slow-to-recover group.

Music therapy may reduce psychomotor agitation post coma and improve mood following severe traumatic brain injury.

8.6 Addictive Behaviours Post ABI

8.6.1 ABI and Substance Abuse

Several studies have examined the rates of substance abuse in those who have sustained a TBI and found that 44 to 79% of individuals have an alcohol addiction at time of injury, while another 12 to 33% reported having a drug addiction (Kolakowsky-Hayner et al. 2002; Taylor et al. 2003; West et al. 2009). The Diagnostic and Statistical Manual (DSM-IV-TR) outlines criteria that must be satisfied to determine if an individual has an addiction or dependence issue; however, the definitions of ‘abuse’ and ‘addiction’ vary between studies. A study examining the effects of alcohol and other substances on various neuropsychological measures found those who reported using alcohol or other substances prior to their injury, scored significantly lower than those who did not have a history of substance use (Kelly et al. 1997). It has been noted that of those who sustain their injury in a motor vehicle collision (one of the leading causes of TBIs), almost half were found to be intoxicated (DeLambo et al. 2008; Wehman et al. 2000; West et al. 2009). Acute intoxication has been found, in some studies, to impact the duration of coma, length of time in post-traumatic amnesia (PTA), overall length of stay, post recovery cognitive outcomes and self-care abilities (Bombardier & Thurber 1998; Vickery et al. 2008).

Studies have found that substance abuse issues occur more frequently with those who have sustained TBI than members of the general public (Taylor et al. 2003) and many will return to drinking within two years of injury (Bombardier & Thurber 1998). Hibbard et al. (1998) reported that as many as 40% of the TBI population meet the criteria for substance abuse or dependence as defined by the DSM-IV. Post injury, even small amounts of alcohol can result in more significant cognitive impairments as the individual works through the recovery process (Tweedly et al. 2012). The link between depression or other mood disorders and substance abuse has also been shown to be quite strong both pre and post ABI (Jorge & Starkstein 2005).

Individual Studies

Table 8.29 Prevalence of Substance Use and Abuse Pre and Post ABI

<table>
<thead>
<tr>
<th>Author/Year/Country/Study Design</th>
<th>Methods</th>
<th>Results</th>
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<tbody>
<tr>
<td>Kwok et al. (2013) Canada Case Control N=102</td>
<td>Population: TBI=75, nTBI=27; Mean Age=43.3yr; Gender: Male=70, Female=32. Intervention: Regular care reported if education sessions were given to patients during rehabilitation. Outcome Measure: Number of patients: 1) who consumed alcohol or drugs at time of injury, 2)</td>
<td>1. 24.5% of patients had documentation of alcohol and/or drugs in their body at the time of injury; of which 88% had alcohol and 12% drugs. 2. 36.3% of the sample abused alcohol (56.8% of these individuals) or illicit drugs (13.5% of these individuals) and 29.7% abused both.</td>
</tr>
<tr>
<td>Andelic et al.</td>
<td>Mental Health Issues Post ABI</td>
<td>Ponsford et al.</td>
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<tr>
<td>(2010)</td>
<td>Population: TBI; Mean Age=32.2yr; Gender: Male=87, Female=24; Mean GCS Score=7. Intervention: Cut down, Annoyed, Guilty, Eye-opener (CAGE) questionnäre used as a standard patient interview screening substance abuse followed by CT scan. Outcome Measure: Glasgow Outcome Scale, Injury Severity Score.</td>
<td>(2007)</td>
</tr>
<tr>
<td>Norway Observational N=111</td>
<td></td>
<td>Australia Case-Control N=121</td>
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</table>

1. 26% of the subjects reported pre-injury substance abuse.  
2. Patients with less severe TBI more frequently reported substance use at time of injury while patients with more severe TBI more frequently reported pre-injury substance abuse (30% versus 23%; p=0.01).  
3. An adjusted regression analysis revealed that subjects with a CAGE score≥2 (indicative of pre-injury substance abuse) were likely to have a more severe TBI (p=0.04).  
1. At 1yr post injury, significantly more (30%) had abstained from alcohol completely compared to those who abstained before their injury (8.4%; p<0.001).  
2. 17.4% of the participants reported drinking at hazardous levels and another 11.5% indicated they had alcohol dependence.  
3. At the 2yr assessment, more were abstaining (21% versus 30%) but amongst those who were drinking, the frequency of drinking had increased.  
4. The use of other drugs also decreased at 1yr post injury, but increased at the 2yr mark.  
5. Drinking and substance abuse was noted as a problem for approximately 30% of the 76 individuals who completed the surveys.  
1. More males than females were intoxicated or using drugs at injury and had substance abuse (p<0.05).  
4. 18.6% of patients were referred for counselling.  
1. 65 individuals were found to have an alcohol related problem pre injury.  
2. 25 continued to have an alcohol related problem 1yr post injury.  
3. Those identified as having alcohol abuse problems pre injury were 10.9 times more likely to have a problem post injury in comparison to those who did not report alcohol abuse pre-injury.  
1. Differences among alcohol subtypes were found in total drinks per week, SMAST and physical dependency (p<0.001 for all).  
2. Subtypes differed in their drug use (p<0.01) with type IV reporting less drug use than the other groups.  
3. The contribution of alcohol to cause of injury differed among subtypes.
Evidence-Based Review of Moderate to Severe Acquired Brain Injury

Module 8 - Mental Health Issues Post ABI

Outcome Measure: Short Michigan Alcohol Screening Test (SMAST), Physical Dependence Scale, Readiness to Change Questionnaire, Blood Alcohol Level (BAL), number of drinks per week, illicit drug use, preferences for alcohol-related behavioural change strategies and extent to which substance use was cause of injury.

(p<0.001) with type I reporting more alcohol-related injuries.

4. Type IV were least likely to have documentation of alcohol-related problems while type I were most likely to have documentation than type II (p<0.001).

5. At time of admission, type I and type II were more likely to have a positive BAL than type IV (p<0.001).

Discussion

There is currently a large variation in the rates of substance abuse reported in the TBI population; the studies listed above confirm this discrepancy. The prevalence of pre-injury alcohol abuse was reported between 11.5% and 49% (Andelic et al. 2010; Bombardier et al. 2002; Kwok et al. 2013; Ponsford et al. 2007) while illicit drug use was reported to be between 30% and 38% (Bombardier et al. 2002; Kwok et al. 2013). The problem with comparing the reported pre-injury substance abuse rates is that the inclusion criteria for many of the studies differ. Studies which only include subjects with a positive Blood Alcohol Concentration (BAC) at time of admission will report an inflated incidence since non-users are automatically excluded. Bombardier and colleagues (2002) reported that the number of drinks per week pre-injury reported by their sample was in the 84th percentile of average American alcohol consumption. This suggests that substance abuse is a much greater problem in the TBI population than in the general population. Furthermore, a history of substance abuse may be a risk factor for future TBI. Interestingly, substance abuse is more often affiliated with moderate to severe injuries while intoxication at time of injury is more often affiliated with mild injuries (Andelic et al. 2010). Studies suggest that alcohol consumption and substance use decline within the first year of injury (Bombardier et al. 2003; Jorge 2005; Kelly et al. 1997; Ponsford et al. 2007), but those who returned to drinking two years post injury were likely to consume more than before the injury, drink excessively, and be dependent on alcohol (Bombardier et al. 2002; Ponsford et al. 2007). Ponsford and colleagues (2007) reported the same trend for the use of illicit drugs. TBI victims who abused alcohol pre-injury were 10 times more likely to demonstrate problematic alcohol use post injury (Bombardier et al. 2003).

Individual characteristics were also found to determine the likelihood that a patient with a TBI will have difficulties controlling their substance use. High consequences associated with drinking are thought to mediate the frequency of alcohol consumption and alcohol dependence (Turner et al. 2003). Individuals
who drink excessively and have large negative consequences associated with their drinking are more likely to report alcohol as the cause of their TBI and are more likely to report pre-injury substance abuse (Turner et al. 2003).

**Substance abuse and intoxication at time of injury is a frequent phenomenon in the traumatic brain injury population.**

**Substance addiction pre injury is predictive of substance addiction post injury.**

### 8.6.2 Substance Abuse and Assessing the Severity of Injury

When assessing the severity of injury several issues have been raised. The first is the use of the Glasgow Coma Scale (GCS). It has been suggested that the GCS is unreliable when using it to establish functioning level at time of injury for those who have been drinking and/or using other substances (Jagger et al. 1984). However, Stuke et al. (2007) in a recent study found the GCS was not affected by the BAC of individuals admitted to a local trauma centre. This finding has been supported by some (Kelly et al. 1997; Sperry et al. 2006); and rejected by others (O’Phelan et al. 2008). To date there is conflicting evidence when looking at the effects of alcohol on the level of injury, survival rates and GCS.

#### 8.6.2.1 Effect of Substance Use on Initial Assessments and Severity of Injury

Alcohol use has been identified as a contributing factor in the cause of brain injury but more destructively as a factor which promotes poor long-term recovery from injury and makes assessment of injury more difficult (Vickery et al. 2008). Recent evidence implies that alcohol has a neuroprotective effect on neuronal recovery post TBI (Andelic et al. 2010; Berry et al. 2010; Shahin et al. 2010). The evidence surrounding the effects of alcohol at time of injury is conflicting.

**Individual Studies**

**Table 8.30 Effects of Substance Use at Time of Injury on Severity of Injury Post ABI**

<table>
<thead>
<tr>
<th>Author/Year/Country/Study Design/PEDro Score</th>
<th>Methods</th>
<th>Results</th>
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<tbody>
<tr>
<td><strong>Andelic et al. (2010)</strong> Norway Observational N=111</td>
<td><strong>Population:</strong> TBI; Mean Age=32.2yr; Gender: Male=87, Female=24; Mean GCS Score=7. <strong>Intervention:</strong> Cut down, Annoyed, Guilty, Eye-opener (CAGE) questionnaire used as a standard patient interview screening substance abuse followed by CT scan. <strong>Outcome Measure:</strong> Glasgow Outcome Scale, Injury Severity Score.</td>
<td>1. 26% of the subjects reported pre-injury substance abuse. 2. Patients with a less severe TBI more frequently reported substance use at time of injury while patients with a more severe TBI more frequently reported pre-injury substance abuse (30% versus 23%; p=0.01). 3. An adjusted regression analysis revealed that subjects with a CAGE score≥2 (indicative of pre-injury substance abuse) were likely to have a more severe TBI (p=0.04).</td>
</tr>
<tr>
<td><strong>Berry et al. (2010)</strong> USA Case Series</td>
<td><strong>Population:</strong> TBI; Mean Age=42yr; Gender: Male=5901, Female=1403; Mean GCS Score=11.3. <strong>Intervention:</strong> Patients who tested positive for ETOH</td>
<td>1. 44.1% of patients tested positive for ETOH on admission. 2. In comparison to ETOH-negative patients, ETOH-positive patients were...</td>
</tr>
</tbody>
</table>
### Evidence-Based Review of Moderate to Severe Acquired Brain Injury

**Outcome Measure:** Abbreviated Injury Score, Injury Severity Score, GCS.

1. The pre-hospital GCS was significantly lower in the intoxicated group (p<0.001).
2. The median change in GCS from pre-hospital to emergency department worsened in the non-intoxicated group (p=0.039) and remained stable in the intoxicated group (p=0.09).
3. The change in GCS between the emergency department and day 1 in the intoxicated group was significantly greater than the non-intoxicated group (p<0.001).
4. BAC is positively related to changes in GCS; higher BAC is associated with better improvement (p<0.0001).
5. AIS was also significantly associated with GCS changes indicating high AIS is related to greater improvement (p<0.0001).

### Population

**Shahin et al.** (2010) USA Case Control

- **N=188**
- **Population:** TBI; *Intoxicated Group (n=100)*: Mean Age=31yr; Gender: Male=88, Female=12; *Non-intoxicated Group (n=88)*: Mean Age=34yr; Gender: Male=77, Female=23.
- **Intervention:** Patients admitted to the Neurosurgical Intensive Care Unit (ICU) were divided into intoxicated (BAC ≥0.08%) or non-intoxicated (BAC <0.08%) groups.
- **Outcome Measure:** Abbreviated Injury Score (AIS), GCS, Blood Alcohol Content (BAC).

1. Although there were more males in the alcohol group, there were no significant differences on the GCS between the genders.
2. Alcohol-positive patients were more likely to have sustained a head injury (p<0.001).
3. Overall alcohol use did not result in a clinically significant reduction in GCS.

### Population

**Vickery et al.** (2008) USA Cohort

- **N=1,748**
- **Population:** TBI; Mean Age=38yr; Gender: Male=1261, Female=487.
- **Intervention:** Data obtained from the Traumatic Brain Injury Model Systems.
- **Outcome Measure:** GCS, Length of Stay (LOS), Disability Rating Scale (DRS), Functional Independence Measure (FIM).

1. Acute LOS was correlated with GCS (p<0.01).
2. Age, education level, LOS, GCS and BALs were related to DRS (p<0.05).
3. Alcohol related variables were not associated with FIM cognitive scores or FIM motor scores.

### Population

**Stuke et al.** (2007) Cohort

- **N=108,929**
- **Population:** Alcohol-Positive Group (n=55,732): Mean Age=31yr; Gender: Male=44,586, Female=11,146; Alcohol-Negative Group (n=53,197): Mean Age=30yr; Gender: Male=35,642, Female=17,555.
- **Intervention:** Patients positive for alcohol testing were compared to those negative for alcohol testing.
- **Outcome Measure:** GCS, Abbreviated Injury Score.

1. Although there were more males in the alcohol group, there were no significant differences on the GCS between the genders.
2. Alcohol-positive patients were more likely to have sustained a head injury (p<0.001).
3. Overall alcohol use did not result in a clinically significant reduction in GCS.

### Population

**Sperry et al.** (2006) USA Case Control

- **N=1,075**
- **Population:** Intoxicated Group (n=504): Mean Age=34.7yr; Gender: Male=441, Female=63; Mean GCS Score=10.3; Non-intoxicated Group (n=571): Mean Age=36.7yr; Gender: Male=431, Female=140; Mean GCS Score=10.1.
- **Intervention:** Patients with TBI were grouped based on tested positive for intoxication and those that tested negative for intoxication.
- **Outcome Measure:** GCS, Abbreviated Injury Score.

1. Mean BAL for intoxicated patients was 202±77mg/dL.
2. More intoxicated subjects were injured through physical assault (18% versus 11%); whereas non-intoxicated subjects were more likely to be injured in a motor vehicle collision (MVC) (46% versus 40%).
3. No linear relationship was found between BAC and GCS.
Discussion
Several studies have investigated the effects of alcohol and/or other chemical substances on GCS, and length of stay in intensive care (Sperry et al. 2006; Vickery et al. 2008). It has been noted by Andelic and colleagues (2010), that patients diagnosed with a less severe TBI more frequently report substance use at the time of injury while those diagnosed with a more severe injury frequently report pre-injury substance abuse. Sperry et al. (2006) found no relationship between alcohol intoxication and GCS, nor did they find a linear relationship between BAC and GCS. However, a study found a higher BAC was associated with a better improvement in GCS over time (Shahin et al. 2010). Although it has been suggested that the presence of alcohol or other substances leads to a greater risk for poorer outcomes, evidence is still inconclusive.

The impact that blood alcohol levels have on Glasgow Coma Scale, Injury Severity Score, mortality, and long term outcomes has yet to be determined.

8.6.2.2 Effects of Substance Use on Mortality
The protective role of elevated levels of serum ethanol levels and TBI continues to be debated. Recent research suggests that alcohol acts as a neuroprotective agent and plays a role in survival post injury (Berry et al. 2010). Further, earlier studies have found that mortality was not more common in those who had been intoxicated at time of injury (Kelly 1995). Despite the quantity of studies looking at levels of intoxication, length of hospitalization, TBI severity and mortality, a solid link has not yet be made (Berry et al. 2010; Kelly et al. 1997).

Individual Studies

Table 8.31 Effect of Substance Use on Mortality Post ABI

<table>
<thead>
<tr>
<th>Author/Year/Country/Study Design/PEDro Score</th>
<th>Methods</th>
<th>Results</th>
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<tbody>
<tr>
<td><strong>Berry et al., (2010)</strong> USA Case Series N=7,304</td>
<td><strong>Population</strong>: TBI; Mean Age=42yr; Gender: Male=5901, Female=1403; Mean GCS Score=11.3 <strong>Intervention</strong>: Patients who tested positive for acute alcohol (ETOH) intoxication (n=3219) were compared to the patients who tested negative for ETOH (n=4085). <strong>Outcome Measure</strong>: Abbreviated Injury Score, Injury Severity Score, GCS.</td>
<td>1. A lower mortality rate was observed in ETOH-positive patients (p=0.005). 2. A logistic regression revealed the presence of ETOH in patient’s blood was associated with reduced mortality (p=0.035).</td>
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<tr>
<td><strong>Salim et al., (2009)</strong> USA Case Control N=481</td>
<td><strong>Population</strong>: TBI; Mean Age=38.6yr; Gender: Male=400, Female=81. <strong>Intervention</strong>: Comparison of two study groups (ETOH-positive and ETOH-negative). <strong>Outcome Measure</strong>: Injury Severity Score (ISS), survival, ventilator days, ICU and hospital length of stay, and complications.</td>
<td>1. 179 (37%) subjects were ETOH-positive. 2. ETOH-positive group had a higher percentage of males (p=0.001), lower percentage of penetrating injuries (p=0.002) and lower ISS (p=0.05). 3. Hospital mortality was lower in the ETOH-positive group compared to the ETOH-negative group (p=0.004). ETOH-positive subjects had higher rates of sepsis (p=0.01).</td>
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<td>Study</td>
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<td>Salim et al. (2009)</td>
<td>USA</td>
<td>Case Control</td>
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<td>Shandro et al. (2009)</td>
<td>USA</td>
<td>Cohort</td>
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<tr>
<td>O’Phelan et al. (2008)</td>
<td>USA</td>
<td>Case Series</td>
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<tr>
<td>Tien et al. (2006)</td>
<td>Canada</td>
<td>Cohort</td>
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<tr>
<td>Alexander et al. (2004)</td>
<td>USA</td>
<td>Case Control</td>
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4. The mean serum ETOH level was greater for survivors than non-survivors (p=0.001).

1. 14,419 (37.9%) subjects tested positive for ETOH. The ETOH-positive subjects were younger (p<0.001), had lower ISS (p<0.001) and lower GCS (p<0.001).
2. ETOH-positive subjects were more likely to have positive toxicology for illicit drug use (p<0.001).
3. After adjusting for the differences between the tested and non-tested groups, ETOH-positive subjects had reduced mortality (p=0.005) and more complications (p<0.001).

1. Results indicate that alcohol status had no effect on the survival rates of patients either in the acute stage, at 3mo post injury or at 1yr post injury.
2. There was a non-significant trend toward lower mortality in patients with higher BAC levels.

1. Of the 483 charts reviewed, 331 (68.5%) patients were found to be intoxicated at the time of injury.
2. Older age (p=0.033), higher ISS (p=0.117), and lower GCS (p=0.009) were associated with higher mortality. A positive toxicology screen was associated with lower mortality (p<0.001).
3. Those who tested positive for methamphetamine and alcohol were found to have decreased mortality.

1. Males were more likely to have a higher BAC than females.
2. Younger individuals were more likely to have higher BAC than older individuals.
3. In individuals with a severe head injury, the overall risk of dying was significantly lower in patients with a low to moderate BAC compared with no BAC (p=0.0008). Patients with a higher BAC had higher risk of dying compared with those with no BAC, albeit non-significantly (p=0.1).

1. A relationship was found between the serum alcohol levels and GCS.
2. There was also a relationship between CBF and serum alcohol levels (p=0.02) but there was no significant association.
Outcome Measure: Glasgow Outcome Scale (GOS), GCS, cerebral blood flow (CBF), mortality.

<table>
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<th>between serum alcohol levels and GOS at 3, 6 or 12mo.</th>
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<td>3. Serum alcohol levels &gt;100mg/dL were associated with a decrease in global CBF.</td>
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<td>4. The three groups had similar mortality rates.</td>
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### Discussion

Over the past couple of decades several studies have investigated the effect of BAL on mortality post TBI. Tien et al. (2006) found that moderate or low BAC levels lowered the risk of dying in those who had sustained a severe TBI. Overall, study findings suggest elevated BAL is not associated with an increased risk of mortality post injury (Berry et al. 2010; O’Phelan et al. 2008; Salim et al. 2009; Shandro et al. 2009; Tien et al. 2006). Further, O’Phelan et al. (2008) looked at the effect of BAL, cannabis and amphetamines, and found that both alcohol and methamphetamine were associated with a decrease in mortality. Further research needs to be conducted to determine conclusively the effects alcohol and other substances have on severity of TBI.

*Although alcohol and elevated blood alcohol levels have been linked to an increase risk of sustaining a TBI, there is evidence to suggest that elevated blood alcohol levels are not linked to an increase risk of mortality post injury.*

*The possible neuroprotective role acute alcohol intoxication plays in TBI warrants further investigation.*

### 8.6.3 Post-injury Recovery and Substance Addiction

If individuals continue to use or abuse alcohol or drugs post injury, their recovery is negatively impacted. Continued use of alcohol or other substances may increase levels of aggressiveness, risk of seizures, decrease their satisfaction with life and increase family stress. Substance abuse often impacts the neurotransmitter process making it difficult to assess the impact that the brain injury has on the individual. Many individuals have been found to spend more time in rehabilitation programs, as alcohol addiction has been found to accentuate sensory motor, cognitive and communication problems post injury (Wehman et al. 2000). Continued involvement with alcohol and other substances increases the risk of developing medical complications.

Involvement in rehabilitation deters or prevents individuals from using various substances as patients are monitored rather closely (Bjork & Grant 2009). However, once patients are discharged from inpatient rehabilitation, no monitoring exists and patients may return to their previous behaviours or begin using drugs and alcohol as a coping strategy. Alcohol and other substance addictions may lead to a failure to survive independently in the community (Burke et al. 1988).
### Table 8.32 Influence of Substance Use or Abuse on Neuropsychological Outcomes Post ABI

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<thead>
<tr>
<th>Author/Year/ Country/Study Design/PEDro/N</th>
<th>Methods</th>
<th>Results</th>
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<td><strong>Ponsford et al.</strong>&lt;br&gt;(2013)&lt;br&gt;Australia&lt;br&gt;Pre-Post&lt;br&gt;N=50</td>
<td><strong>Population</strong>: TBI; Mean Age=35.2yr; Gender: Male=45, Female=5; Mean Time Post Injury=7.2mo.&lt;br&gt;<strong>Intervention</strong>: Brief Alcohol Intervention.&lt;br&gt;<strong>Outcome Measure</strong>: Alcohol Use Disorders Identification Test, Time Line Follow-Back (TLFB), California Verbal Learning Test (CVLT-II), Modified Six Elements Test (MSET), The National Adult Reading Test (NART), The Symbol Digit Modalities Test (SDMT).</td>
<td>1. 27.8% of participants drank at harmful or hazardous levels prior to their injury.&lt;br&gt;2. At 6-9mo follow-up, 52% of patients reported alcohol abstinence but 16.7% of patients were still drinking at harmful or hazardous levels.&lt;br&gt;3. At 12-15mo follow-up, 28% of patients were not drinking but 20% of participants reported harmful or hazardous consumption rates.&lt;br&gt;4. Based on TLFB, frequency and quantity of alcohol consumption did not significantly change (p&gt;0.05).&lt;br&gt;5. SDMT was negatively associated with older age (p&lt;0.001) and hazardous pre injury drinking (p&lt;0.05).&lt;br&gt;6. CVLT-II was similarly associated with age (p&lt;0.001) and hazardous pre injury drinking (p&lt;0.05) and was positively associated with NART-IQ (p=0.01).&lt;br&gt;7. MSET was at 6-9mo follow-up was negatively associated with age (p&lt;0.05) and drinking in the prior month (p&lt;0.05) but not hazardous pre injury drinking (p&gt;0.05).</td>
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<td><strong>Schutte &amp; Hanks</strong>&lt;br&gt;(2010)&lt;br&gt;USA&lt;br&gt;Case Series&lt;br&gt;N=482</td>
<td><strong>Population</strong>: TBI; Mean Age=39.5yr; Gender: Male=395; Female=87; Mean GCS Score=9.9.&lt;br&gt;<strong>Intervention</strong>: Examine the impact of BAL on injury severity and outcomes.&lt;br&gt;<strong>Outcome Measure</strong>: GCS, Trail Making Test (TMT), Rey Auditory Verbal Learning Test (RAVLT), Wisconsin Care Sort Test-64 (WCST), Total Functional Independence Measure (FIM).</td>
<td>1. A significant negative correlation between GCS and BAL was found (r=-0.23; p&lt;0.01).&lt;br&gt;2. BAL only predicted functional outcome at admission to rehabilitation (p&lt;0.05) while age at time of injury predicted functional outcomes at admission, discharge and 1yr.&lt;br&gt;3. TMT-A, TMT-B and WCST (p&lt;0.01 for all) were predictors of cognitive outcomes at 1yr.&lt;br&gt;4. BAL did not predict cognitive outcomes, while age at time of injury predicted TMT-A, TMT-B and WCST performance at 1yr (p&lt;0.05 for all).</td>
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<td><strong>Wilde et al.</strong>&lt;br&gt;(2004)&lt;br&gt;USA&lt;br&gt;Case Control&lt;br&gt;N=77</td>
<td><strong>Population</strong>: TBI; TBI+BAL Group (n=25): Mean Age=29.12yr; Mean GCS Score=8.48; Mean Time Post Injury=1180.64d. TBI-only Group (n=52): Mean Age=26.9yr; Mean GCS Score=8.3; Mean Time Post Injury=900.69d.&lt;br&gt;<strong>Intervention</strong>: Subjects were divided into two groups and compared: TBI+BAL (&gt;10 mg/dL of serum alcohol at admission) and TBI-only (no evidence of positive BAL at admission).&lt;br&gt;<strong>Outcome Measure</strong>: Wechsler Adult Intelligence</td>
<td>1. There was a significant difference between the ventricle-to-brain ratio in the two groups (p=0.009).&lt;br&gt;2. TBI+BAL subjects had more atrophy when examining brain volume (p=0.002), cerebrospinal fluid (CSF) (p=0.002) and total ventricular volume (p=0.045).&lt;br&gt;3. Differences between subjects with no alcohol serum, mild, moderate and heavy alcohol serum were significant for</td>
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**Tate et al.** (1999)  
USA  
Case Series  
N=67

| Scale-Revised (WAIS-R), Wechsler Memory Scale-Revised (WMS-R), outpatient MRIs. | 1. Subjects with high admission BAL were more likely to have a history of alcohol abuse (p<0.0001).  
2. Subjects with mild TBI had lower average BAL than those with moderate-severe TBI (p<0.05).  
3. Poorer performance on WMS-R was negatively correlated with higher admission BAL (p<0.05).  
4. Fewer failures on WCST was correlated with increased BAL (p=0.022).  
5. For partial correlations, BAL was negatively correlated with two aspects of the WMS-R (p<0.05) and the block design section of the WAIS-R (p=0.023).  
6. Admission BAL predicted poor verbal recall (p=0.0156), poor information retention (p=0.0359) and poor visuospatial constructional ability (p=0.0196).  
7. Education and TBI severity also predicted visuospatial constructional ability (WAIS-R, p<0.05).  
8. TBI severity predicted number of conceptual categories and set failures on the WCST (p<0.05).  
9. Age and education predicted time to complete TMT-A and TMT-B (p<0.05), while TBI severity predicted TMT-A performance only (p<0.05). |

**Bombardier & Thurber** (1998)  
USA  
Case Series  
N=58

| Scale-Revised (WAIS-R), Wechsler Memory Scale-Revised (WMS-R), outpatient MRIs. | 1. NCSE orientation (p<0.01), NCSE naming (p<0.005), NCSE verbal memory (p<0.005) and NCSE similarity (p<0.05) scores were inversely correlated with BAL at time of injury.  
2. Time to complete TMT-A was positively correlated with BAL (p<0.05).  
3. Patients with positive BAL at time of injury, NCSE attention span (p<0.05) and NCSE judgment (p<0.05) were inversely correlated with BAL.  
4. After 30d BAL, NCSE memory (p<0.05) and NCSE similarities (p<0.05) were negatively correlated.  
5. Before 30d, NCSE orientation, NCSE naming, RAVLT total and TMT-A time were correlated with BAL. |

**Kelly et al.** (1997)  
USA/Israel

| Scale-Revised (WAIS-R), Wechsler Memory Scale-Revised (WMS-R), outpatient MRIs. | 1. Results indicated that those in the NS group showed better neuropsychological performance on the WAIS-R and the |
### Discussion

Studies investigating the impact of substance use or abuse on neuropsychological outcomes post ABI have resulted in conflicting results. Schutte and Hanks (2010) found that BAL only predicted functional outcome at rehabilitation admission. BAL did not predict cognitive outcomes while age at time of injury was significantly associated with cognitive measures at one year follow-up. Comparatively, recent research with a smaller sample size found many cognitive measures to be associated with hazardous pre-injury drinking and age (Ponsford et al. 2013).

In an earlier study by Tate et al. (1999), BALs were predictive of poorer performance on a variety of neuropsychological measures during post-acute recovery. Overall study authors suggest an increase in BALs predicts greater cognitive impairment. Similar results were noted in a study conducted by Bombardier and Thurber (1998). Here BALs predicted poor performance on orientation tasks, concentration and mental speed, naming abilities, verbal memory and abstract reasoning. Wilde and colleagues (2004) also noted that an increase in alcohol abuse was associated with increased brain atrophy post injury.

> **Earlier studies indicated that elevated blood alcohol levels are associated with poorer performance on a variety of cognitive communication tasks; however, these finding have generally not been supported in most recent studies.**

> **Recent research has found age at injury to be negatively associated with cognitive outcome. More research needs to be conducted investigating the impact of alcohol on cognitive outcomes post injury.**

### 8.6.4 Substance Abuse Treatment Post ABI

Several theories have been put forth regarding the types of programs that might reduce substance abuse in the TBI population, but little research was found supporting these theories. A study was conducted by Corrigan and Bogner (2007) looking at using financial incentives to encourage those with a TBI and substance abuse problem to remain in treatment. In a systematic review, Corrigan and colleagues (2010) concluded that research focused on interventions for substance abuse specifically excluded participants with severe TBI.
## Table 8.33 Compliance with Substance Addiction Treatment Programs Post ABI

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<tr>
<th>Author/Year/ Country/Study Design/ PEDro/N</th>
<th>Methods</th>
<th>Results</th>
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<tr>
<td><strong>Ponsford et al., (2012)</strong>&lt;br&gt; Australia&lt;br&gt; RCT&lt;br&gt; PEDro=7&lt;br&gt; N=50</td>
<td>Population: TBI; Mean Age=35yr. Intervention: Patients randomly received MI along with information (MI+INFO; n=18), just information (INFO; n=15), or informal discussion (ID control; n=17). Outcome Measure: Alcohol Use Disorders Identification Test, California Verbal Learning Test-II, Readiness to Change Questionnaire, Hospital and Anxiety and Depression Scale (HADS) and Westmead PTA scale.</td>
<td>1. No significant difference in overall frequency and quantity of alcohol consumption 6mo following the intervention (p&gt;0.05).&lt;br&gt; 2. 29.5% participants in the ID control group, 6.7% of the INFO group and 16.7% of the MI+INFO group were drinking &gt;1x/wk.&lt;br&gt; 3. There was an increase over time in the probabilities of higher levels of drinking frequency and quantity drinking behaviours with no statistical significant differences between groups (p&gt;0.05).&lt;br&gt; 4. Having reached the action stage of readiness to change was associated with low estimated risk of frequent drinking (p&lt;0.001) and of heavier drinking (p&lt;0.001).&lt;br&gt; 5. An extra year of education was associated with a higher risk of more frequent drinking and a higher risk of heavier drinking (p&lt;0.05).&lt;br&gt; 6. HADS-Depression score indicated greater depression was associated with a risk of heavier drinking (p&lt;0.05).</td>
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<td><strong>Sander et al., (2012)</strong>&lt;br&gt; USA&lt;br&gt; RCT&lt;br&gt; PEDro=5&lt;br&gt; N=104</td>
<td>Population: TBI; Intervention Group (n=54): Mean Age=36.1yr; Gender: Male=44, Female=10; Median GCS Score=14. Control Group (n=50): Mean Age=35.4yr; Gender: Male=41, Female=9; Median GCS Score=12. Intervention: Patients were randomly allocated to receive intervention or standard care. Those in the treatment group participated in a 10min educational DVD describing potential negative effects of alcohol abuse after TBI, then asked to consider pros/cons of substance abuse. Control group received information and referral services typically given to those with a substance issues. Outcome Measure: CAGE Alcohol Questionnaire, Alcohol Expectancy Questionnaire-III, Readiness to Change Question (RTC).</td>
<td>1. Individuals who attributed their TBI to alcohol use, indicated alcohol use could result in physical and cognitive impairment.&lt;br&gt; 2. Overall the treatment did not have any effect on RTC or problem alcohol use.</td>
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<td><strong>Tweeddy et al., (2012)</strong>&lt;br&gt; USA&lt;br&gt; RCT&lt;br&gt; PEDro=5&lt;br&gt; N=60</td>
<td>Population: TBI; ID Group (n=20): Mean Age=36.5yr; Gender: Male=15, Female=5; Mean Time Post Injury=8mo. INFO Group (n=20): Mean Age=35.1yr; Gender: Male=14, Female=6; Mean Time Post Injury=7.95mo. MI+INFO Group (n=20): Mean Age=33.9yr; Gender:</td>
<td>1. At the 6-9mo follow-up all reported a large consumption rate for alcohol.&lt;br&gt; 2. Those in the ID group reported consuming more alcohol over time; however the total TLFB scores were not</td>
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<td>Author/Year/ Country/Study Design</td>
<td>Population: TBI; Mean Age=42.5yr; Gender: Male=46, Female=28.</td>
<td>Intervention: One of 3 groups: provision of financial incentives to not miss appointments (n=24); reduction of logistical barriers to attending appointments (n=26); and attention control (n=24). Intervention was delivered via a phone call.</td>
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<td>Corrigan &amp; Bogner (2007) USA RCT PEDro=5 N=74</td>
<td>Male=16, Female=4; Mean Time Post Injury=7.79mo.</td>
<td><strong>Intervention:</strong> Randomly assigned to (1) Informal Discussion (ID) group: general 30min discussion about changes that had occurred since their injury, (2) the Brief Information group: informal discussion and packages with a short DVD a booklet outlining cognitive, physiological and behavioural changes that can occur following a TBI, or (3) the Information Plus Brief MI group received both interventions. Assessment took approximately 2hr to complete.</td>
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| Corrigan et al. (2005) USA RCT PEDro=5 N=195 | Male=138, Female=57. | **Intervention:** Participants were placed in one of four groups: barrier reduction, MI, financial incentive or attention control. | **Outcomes**

1. Receiving financial incentives resulted in fewer missed appointments, compared to those who had specific barriers (poor memory, transportation issues) removed (p<0.001), but did not have any significant effect on premature termination from treatment.
2. There was no statistically significant differences in the number of missed appointments between the barrier group and the control group (p<0.318).
3. Interventions offered did not affect the client or counsellor therapeutic relationship.

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**Treatment Outcome**

| Author/Year/ Country/Study Design | Population: TBI; Mean Age=26yr; Gender: Male=56, Female=16; Mean Time Post Injury=44.3mo. | **Intervention:** Substance abuse treatment | **Outcomes**
1. 75% of participants had a positive substance abuse outcome; 18% maintained abstinence, 32% attained

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**Module 8-Mental Health Issues Post ABI-V11**

Evidence-Based Review of Moderate to Severe Acquired Brain Injury

plan. Some of the subjects had a community team while others did not. **Outcome Measure:** Quantity-Frequency-Variability Index, General Health and History Questionnaire, Addiction Severity Index.

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<td>1.</td>
<td>abstinence and 25% reduced alcohol use.</td>
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<td>2.</td>
<td>A significant difference in abstinence was found between initial assessment and 1yr follow-up (p&lt;0.05).</td>
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<td>3.</td>
<td>Participants with a community team had more positive substance abuse outcomes (p&lt;0.05).</td>
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<tr>
<td>4.</td>
<td>A greater proportion of patients with community teams attained abstinence, but a greater proportion without teams maintained it.</td>
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Discussion

Recently several studies have been conducted looking at the effect of motivational interviewing coupled with information sessions and the impact it had on patients returning to drinking post TBI (Ponsford et al. 2012; Sander et al. 2012; Tweedly et al. 2012). In two of these studies individuals were randomly assigned to an informal discussion group, an information group or an information plus brief motivational group. Participants were also stratified by gender (Ponsford et al. 2012; Tweedly et al. 2012). Results from each of these studies revealed that the intervention provided did not have a significant effect on drinking post injury or on willingness to change drinking habits. Sander et al. (2012) found those with a more severe injury expected alcohol use would negatively impact cognitive and physical impairments. Ponsford et al. (2012) noted that higher education and higher levels of depression were also associated with greater alcohol consumption.

In a study conducted in Corrigan and Bogner (2007), subjects with a diagnosed substance abuse problem were randomly assigned to one of three groups. All interventions were administered during a telephone interview. The three intervention groups were 1) provision of financial incentives to not miss appointments 2) reduction of logistical barriers to attending appointments and 3) attention control. Results demonstrated that offering a financial incentive (group 1) was more effective in promoting compliance in attending treatment sessions than either other intervention which aimed to reduce barriers.

Conclusion

*There is Level 2 evidence suggesting that neither education nor motivational interviewing has a significant impact on excessive alcohol consumption post-traumatic brain injury.*

*There is Level 2 evidence supporting the use of financial incentives to encourage participants to continue with their substance addiction therapy following an acquired brain injury; however addressing the barriers preventing individuals from attending was not found to be successful.*

*Education and motivational interviewing do not appear to have a strong impact on excessive alcohol consumption post-traumatic brain injury.*

*Providing financial incentives does encourage those with a traumatic brain injury and a substance addiction to attend treatment more so than offering solutions to other barriers.*
8.7 Restraints

8.7.1 Restraint Use

Due to the continued concern regarding the safety of both patients and staff in hospitals and long term facilities, the use of restraints continues to be part of common clinical practice; however their use remains controversial. Following an ABI the incidence and prevalence of agitation or aggressive behaviours ranges from 10% to 96%. Studies have found as many as 13%-32% of survivors may be restrained while undergoing care in either an acute or rehabilitative hospital (Gregory & Bonfiglio 1995; McNett et al. 2012; Morrison et al. 1987; Stubbs & Alderman 2008). Due to the broad definition of agitation, the reported numbers of agitated patients may be misleading; consequently, questions are being raised about how many individuals actually need to be restrained (Eisenberg et al. 2009).

The term “restraint” includes the use of either chemical (medications) or physical (mechanical) restraints or a combination of both (Marks 1992). Chemical restraints used to assist in controlling behaviours that occur during agitated states include many pharmaceutical agents with primary or secondary psychotropic effects, including: beta blockers, anti-depressants, psychostimulants, anti-Parkinson’s agents and anticonvulsants (Gregory & Bonfiglio 1995; McNett et al. 2012). Medication treatments used in non-emergent situations to reduce the need for physical restraints include propranolol, atypical neuroleptics and valproic acid (Busch & Shore 2000). Physical restraints have been defined as any manual method that immobilizes or reduces the ability of individuals to move their arms, legs, body, or head freely (Busch & Shore 2000; Stevens 2012). Typically they are not meant to be a part of the standard practice of care (Amato et al. 2006). Physical restraints include the use of bed rails, feeding trays, mittens (tying of hands), chest straps (seat belts), ankle and wrist restraints, and jacket restraints (Busch & Shore 2000; Gregory & Bonfiglio 1995; Marks 1992; Morrison et al. 1987).

Policies related to the application of restraints often state that the use of restraints should meet the following criteria: (1) be individualized and offer as much dignity to the individual as the situation allows; (2) be humanely and professionally administered; (3) have safety protocols in place; (4) patient must be monitored; (5) careful documentation of the type of restraint, the reason for it, and the means for observation while in the restraint; (6) the method or choice of restraint must be the least restrictive option available (American Nurses Association 2012; College of Nurses of Ontario 2009; Ministry of Health and Long Term Care for the Province of Ontario 2001; St. Joseph’s Health Care 2012). In accordance with the province’s legislation, the College of Nurses for Ontario suggests that the following information is to be recorded when using restraints: significant patient behaviours, alternative considered and used, date and time of application, reason given to patient, type used, reason for choice and patient’s response.

The decision to use restraints whether mechanical or chemical is generally made by physicians or nurses on the unit. In a recent survey, hospital physicians were asked to review a series of vignettes and to comment on the likelihood of ordering restraints (Sandhu et al. 2010). Those most likely to order restraints were family physicians and surgeons while geriatricians were least likely. Further, male doctors were more likely to order restraints than female doctors and they were more likely to order them for male patients. The use of restraints must be accompanied by a consent form signed by the family or caregiver indicating they are in agreement; in emergency situations, this form may not be required. Whether or not individuals were restrained, a study by Schleenbaker and colleagues (1994) found restraint orders, written as “restrain as needed”, were in that charts of more than 75% of individuals admitted for rehabilitation. Of those who were admitted for a TBI approximately 90% had restraint
orders appearing on their chart.

Despite guidelines and policies around restraint use, the literature suggests that there is need for improvement around the documentation and use of restraints in clinical practice. In a retrospective audit conducted in a Canadian hospital, Kow and Hogan (2000) found either chemical or physical restraints were used in 11.5% of patients. Of concern, despite hospital policy, “orders” approving the use of restraints were missing from some charts and the nursing documentation pertaining to the use of restraints was often vague and questionable. The lack of documentation or an “order” to use a restraint has been echoed by others in the literature (Mackpherson et al. 1990; McNett et al. 2012; Minnick et al. 2007; Mion et al. 1996; Morrison et al. 1987; Schleenbaker et al. 1994), with one study noting nursing and physicians found getting a physician’s order and properly documenting were not always necessary (Mion et al. 1996).

8.7.2 Reasons Cited for their Use

A great deal of research has been conducted looking at the use of physical restraints in nursing homes or in acute care hospitals (Evans & FitzGerald 2002; Ludwick et al. 2008). Nursing literature indicates that the use of restraints is influenced by the values, education, and beliefs of the nurses themselves, as well as the behaviours and demographic characteristics of the patients (Ludwick et al. 2008). Results from a recent study indicate impulsiveness, pulling at devices or removing endotracheal tubes, central venous lines and other life support measures, and decreased attention span are often cited as reasons for the use of a restraint (McNett et al. 2012). Additional reasons were controlling agitation or acts of aggression, behaviour control related to altered mental status and confusion, prevention of wandering, patient safety related to impaired mobility, supporting patient’s posture or sitting balance and preventing disruption of therapy (Evans & FitzGerald 2002; Minnick et al. 2007; Sandhu et al. 2010). Finally, many care professionals indicate the use of restraints prevents the individual from falling and further injuring themselves (Kow & Hogan 2000; Minnick et al. 2007; Mion et al. 1996; Sandhu et al. 2010; Schleenbaker et al. 1994; Suen et al. 2006) and protects the safety of family and staff (Kow & Hogan 2000; Mion et al. 1996). Despite the use of restraints to prevent falls there is no evidence to suggest this procedure is effective; on the contrary there is some evidence to suggest it puts patients at a greater risk of injury (Busch & Shore 2000; Evans & FitzGerald 2002; Mion et al. 1996; Sandhu et al. 2010). Unfortunately, despite there being many legitimate reasons for using restraints, some reasons are not justified; one study found over 70% of nurses felt restraints enabled them to spend less time on nursing care (Suen et al. 2006). Alternative strategies to restraint use (e.g. manipulating the environment, supervision, companionship, reviewing prescribed medications) were not known to many nurses (Suen et al. 2006).

Patients in physical restraints have been found to have higher rates of clinical agitation, as did patients who require constant supervision (McNett et al. 2012; Minnick et al. 2007; Morrison et al. 1987; Visscher et al. 2011). A higher level of aggression was also related to an increase length of stay, lower Functional Independence Measure scores and Mini Mental State Examination (Visscher et al. 2011). Recently McNett et al. (2012) noted reorientation, redirection, constant supervision, the administration of benzodiazepines, restraints, and/or modifying the patient’s environment as common ways to manage agitation post TBI. Visscher and colleagues (2011) found 42% of the study population, which included patients with ABI, had engaged in one or more aggressive acts prior to the patients being restrained; three or more aggressive acts were dealt with daily. Using the Staff Observation Aggression Scale-Revised, 67% of the aggressive incidents were judged to be mild in severity and 33% were severe. Often these incidences were triggered by asking the individual to engage in an activity or take medications, or the individual required help with his or her activities of daily living (Visscher et al. 2011).
8.7.3 Effectiveness of Physical Restraints

Many hospitals use physical restraints to ensure the safety of patients, staff and family members. No clinical evidence supports their use with individuals who have sustained an ABI (Marks 1992). The use of restraints is considered acceptable if the restraint is used to ensure the patient’s safety, if less restrictive interventions have been ineffective in preventing harm to the patient or others, the restraint is implemented safely, and appropriate techniques are used as determined by hospital or organizational policy (Recupero et al. 2011). The risk of harm to the patient must be taken into consideration when using physical restraints, thus all restraints must be discontinued at the earliest possible time, and patients must be monitored to ensure their safety (Busch & Shore 2000). Currently there is not enough data available to determine the efficacy of using physical restraints to reduce agitated or aggressive behaviour post ABI (Duxbury & Wright 2011).

Despite their use, there is no evidence to support the use of restraints in those who have sustained an acquired brain injury/traumatic brain injury.

8.7.4 Reducing the Use of Physical Restraints (PR)

In many facilities the number one reason cited for the use of physical restraints is the prevention of falls. Several studies have looked at a variety of education programs aimed at staff to reduce the use of physical restraints; however no studies were found that investigated the effectiveness of these programs on an ABI/TBI unit.

Individual Studies

Table 8.34 Education Programs to Reduce the Use of Physical Restraints

<table>
<thead>
<tr>
<th>Author, Year, Country</th>
<th>Objective, Intervention Implemented and Facility</th>
<th>Results</th>
</tr>
</thead>
<tbody>
<tr>
<td>Gulpers et al. (2011) Netherlands Prospective Controlled Trial N=26</td>
<td>Sample: 405 nursing home residents. Setting: 13 nursing homes. Intervention: Each ward (n=26) received either EXBELT training (e.g. policy change, education, consultations, and awareness of alternatives) or nothing (control group). Outcome: Use of belt restraints.</td>
<td>1. The EXBELT group had a significantly greater reduction in the use of belts restraints than the control group (p=0.005); the reduction was specifically in belts used while in wheelchairs.</td>
</tr>
<tr>
<td>Huizing et al. (2009) Netherlands RCT N=15</td>
<td>Sample: 241 psychogeriatric nursing home residents. Setting: 7 nursing homes. Intervention: Each ward (n=15) was given either an educational intervention (education program and consultations with a nurse specialist; 5, 2hr sessions) or to the control group. Outcome: Use of physical restraints.</td>
<td>1. 51.5% of residents had physical restraints used at baseline. 2. The intervention did not reduce the use of restraints; the use of restraints increased in both groups (treatment group: 49% to 60%; control group: 54% to 64%).</td>
</tr>
<tr>
<td>Rask et al. (2007) Prospective Controlled Trial USA N=19</td>
<td>Setting: 19 nursing homes. Intervention: Homes with the Falls Management Program (FMP) consisting of organizational leadership buy-in, education, a designated falls co-ordinator, training, and ongoing consultations were compared to control nursing homes (no FMP). Outcome: Rates of falls and restraint use.</td>
<td>1. A significant reduction in restraint use was found in the FMP group (p&lt;0.001) and the control group (p=0.002). 2. A reduction in falls was only seen in the FMP group but the reduction was not significant (p=0.59).</td>
</tr>
</tbody>
</table>
Discussion
Three studies investigating the effectiveness of education programs designed to reduce the use of physical restraints on individuals in nursing homes were examined. All three programs were education based (Gulpers et al. 2011; Huizing et al. 2009; Rask et al. 2007); however, two studies included more substantial changes. Rask et al. (2007) included staff buy-in and the creation of a falls coordinator to increase accountability. Gulpers et al. (2011) make reference to policy changes. Although Huizing et al. (2009) did not find education alone was effective in reducing the use of restraints, the two studies that included multiple components in their interventions and took a more active approach had more favourable outcomes (Gulpers et al. 2011; Rask et al. 2007).

Staff education programs to reduce the use of physical restraints, without increasing the risk of falls, have been shown to be somewhat successful with staff in nursing homes. Further research needs to be completed looking at the impact these education programs would have on those staff working in rehabilitation hospitals.

Conclusion
Restraint policies are often prefaced with the hospital’s philosophy regarding the use of restraints. They have been defined as an unusual and temporary measure, either physical or pharmacological, to limit the activity or control the behaviour of an individual. In the earlier study conducted by Mion and colleagues (1996) they state “reducing the use of physical restraints is a challenge” and almost twenty years later despite current hospital policies and the risk of patient injury it continues to be a challenge. It appears as though the clinicians’ perceptions of the benefits of physical restraints is without any empirical data to support the purported benefit (Mion et al. 1996). The use of restraints to meet the needs of staff striving to maintain order, routines and rules is no longer considered acceptable.
8.8 Summary

1. There is conflicting evidence that sertraline is effective in the treatment of major depression post-traumatic brain injury.

2. There is Level 2 evidence that citalopram aids in the reduction of depression post-acquired brain injury.

3. There is Level 4 evidence that citalopram and carbamazepine may be efficacious in the treatment of depression, anxiety and mood disorders.

4. There is Level 2 evidence to suggest that the administration of desipramine assists in improving mood and reducing depression.

5. There is Level 1a evidence that individuals with a traumatic brain injury who participate in exercise programs report feeling less depressed and report experiencing greater quality of life post injury.

6. There is Level 1b evidence that mindfulness-based stress reduction programmes may be efficacious in reducing depressed mood.

7. There is Level 3 evidence that music therapy does improve depression and anxiety post-acquired brain injury.

8. There is Level 4 evidence that Systematic Motivational Counselling may reduce negative affect.

9. There is Level 1b evidence that both Cognitive Behavioural Therapy and supportive psychotherapy may decrease symptoms associated with depression.

10. There is Level 2 evidence that positive psychology, involving patients writing down things they enjoy, is beneficial in improving happiness scores.

11. There is Level 4 evidence that rehabilitation decreases self-reported depression scores.

12. There is Level 1b evidence that Cognitive Behavioural Therapy does reduce anxiety post-acquired brain injury.

13. There is Level 1b evidence that reducing hopelessness post-traumatic brain injury may be effective at decreasing suicidal ideation.

14. There is conflicting evidence of the effects of Amantadine on reducing irritability and aggression in individuals with moderate-severe traumatic brain injury.

15. There is Level 4 evidence that carbamazepine decreases the incidence of aggressive behaviours following a traumatic brain injury.
16. There is Level 5 evidence to suggest that lamotrigine helps to reduce inappropriate behaviours following post-traumatic brain injury.

17. There is Level 5 evidence that valproic acid decreases the incidence of aggressive behaviours.

18. There is Level 4 evidence that divalproex decreases the incidence of agitation following post-traumatic brain injury.

19. There is Level 4 evidence that sertraline HCL can decrease the incidence of aggression and irritability.

20. There is Level 4 evidence that amitriptyline may be useful in reducing the incidence of agitated behaviour.

21. There is Level 1b evidence that pindolol decreases aggression following brain injury based on one random control trial.

22. There is Level 1b evidence that propranolol reduces the intensity of agitated symptoms following brain injury.

23. There is Level 4 evidence (from one small study) to suggest that quetiapine helps reduce aggressive behaviour.

24. There is Level 4 evidence from one study to suggest that ziprasidone assists in the controlling of agitation following post-traumatic brain injury.

25. There is Level 5 evidence to suggest that an antimanic agent (lithium carbonate) reduces aggressive/agitated behaviour following a brain injury.

26. There is Level 4 evidence that Depo-Provera and counselling reduces sexually aggressive behaviour.

27. There is Level 4 evidence that methotrimeprazine is safe and effective for controlling agitation after an acquired brain injury.

28. There is Level 2 evidence (from one random control trial) to suggest that treatment with methylphenidate following brain injury can significantly reduce anger.

29. There is Level 4 evidence that administration of a single-dose of droperidol calms agitated patients with acquired brain injuries more quickly than other agents.

30. There is Level 4 evidence that haloperidol does not have a negative effect on the success of rehabilitation.

31. There is Level 4 evidence to suggest that anger self-management training is effective in reducing irritability and anger after a traumatic brain injury.
32. There is Level 4 evidence that behavioural approach using antecedent management and/or feedback of consequences reduces undesirable behaviour (e.g., aggression/agitation).

33. There is Level 1b evidence that social skills training has a limited impact on changing inappropriate behaviours and mood disturbances of those who have sustained a severe traumatic brain injury.

34. There is Level 2 evidence that community based program combining education and an individualized behaviour plan (e.g., Natural Setting Behaviour Management intervention) helps to change behaviour.

35. There is Level 2 evidence that participating in a Coping Skills Group assists in improving adaptive coping in the long term.

36. There is Level 2 evidence that anger management reduces aggressive behaviour.

37. There is Level 4 evidence that music therapy reduces psychomotor agitation post coma following a severe traumatic brain injury in a slow-to-recover group.

38. There is Level 2 evidence suggesting that neither education nor motivational interviewing has a significant impact on excessive alcohol consumption post-traumatic brain injury.

39. There is Level 2 evidence supporting the use of financial incentives to encourage participants to continue with their substance addiction therapy following an acquired brain injury; however addressing the barriers preventing individuals from attending was not found to be successful.
8.9 Reference List


