

# ERABI

EVIDENCE-BASED REVIEW  
of moderate to severe  
ACQUIRED BRAIN INJURY

## 4. Motor and Sensory Impairment Rehabilitation Post Acquired Brain Injury

Shawn Marshall MD FRCPC, Shannon Janzen MSc, Tristan Duffy MD(c), Pavlina Faltynek MSc, Magdalena Mirkowski MSc MScOT OT Reg. (Ont.), Scott Janssen MSc, Robert Teasell MD FRCPC

ERABI  
Parkwood Institute  
550 Wellington Road, London ON

## Table of Contents

<b>4.1 Introduction .....</b>	<b>10</b>
<b>4.2 Motor Impairment .....</b>	<b>10</b>
<b>4.2.1 Upper Extremity Interventions .....</b>	<b>10</b>
4.2.1.1 <i>Constraint Induced Movement Therapy .....</i>	11
4.2.1.2 <i>Hand Splinting and Stretching .....</i>	12
4.2.1.3 <i>Interventions for Fine Motor Coordination .....</i>	14
4.2.1.4 <i>Virtual Reality for Upper Extremity Rehabilitation .....</i>	16
<b>4.2.2 Lower Extremity Interventions .....</b>	<b>17</b>
4.2.2.1 <i>Partial Body Weight Supported Gait Training .....</i>	17
4.2.2.2 <i>Multimodal Interventions .....</i>	19
4.2.2.3 <i>Virtual Reality for Lower Extremity Rehabilitation .....</i>	22
<b>4.2.3 Combined Upper and Lower Extremity Interventions .....</b>	<b>23</b>
4.2.3.1 <i>Virtual Reality .....</i>	23
<b>4.2.4 Exercise Programs .....</b>	<b>24</b>
4.2.4.1 <i>Aerobic Training .....</i>	24
<b>4.2.5 Spasticity Interventions .....</b>	<b>29</b>
4.2.5.1 <i>Botulinum Toxin Injections .....</i>	29
4.2.5.2 <i>Nerve Blocking Agents .....</i>	32
4.2.5.3 <i>Electrical Stimulation .....</i>	33
4.2.5.4 <i>Oral Antispasticity Drugs .....</i>	34
4.2.5.5 <i>Intrathecal Baclofen .....</i>	36
4.2.5.6 <i>Casting .....</i>	41
4.2.5.7 <i>Adjustable Orthosis .....</i>	44
4.2.5.8 <i>Hand Splinting and Stretching .....</i>	44
4.2.5.9 <i>Multimodal Interventions .....</i>	46
<b>4.3 Visual Dysfunction .....</b>	<b>48</b>
<b>4.4 Vestibular Dysfunction .....</b>	<b>51</b>
<b>4.5 Pain Post TBI .....</b>	<b>54</b>
<b>4.5.1 Assessing Pain Post TBI .....</b>	<b>57</b>
4.5.1.1 <i>Visual Analog Scale .....</i>	57
4.5.1.2 <i>Numeric Rating Scale .....</i>	57
4.5.1.3 <i>McGill Pain Questionnaire .....</i>	57
4.5.1.4 <i>Headache Disability Inventory .....</i>	57

4.5.1.5 Headache Diary .....	57
<b>4.5.2 Pain Syndromes Post ABI .....</b>	<b>58</b>
4.5.2.1 Neuropathic Pain.....	58
4.5.2.2 Central Pain Syndromes.....	58
4.5.2.3 Post Traumatic Headaches.....	58
<b>4.5.3 Non-Pharmacological Interventions for Pain and Post Traumatic Headache.....</b>	<b>59</b>
4.5.3.1 Biofeedback to Manage Post Traumatic Headache .....	59
4.5.3.2 Cognitive Behavioural Therapy.....	60
4.5.3.3 Relaxation Training .....	61
4.5.3.4 Manual Therapy .....	61
4.5.3.5 Acupuncture .....	61
4.5.3.6 Cryotherapy and Thermotherapy .....	62
<b>4.5.4 Pharmacological Management of Pain and Post Traumatic Headache .....</b>	<b>62</b>
4.5.4.1 Anticonvulsants .....	63
4.5.4.2 Antidepressants.....	64
4.5.4.3 Topical Analgesics .....	65
4.5.4.4 Opioids .....	66
4.5.4.5 Cannabinoids.....	66
<b>4.6 Conclusions .....</b>	<b>67</b>
<b>4.7 Summary.....</b>	<b>68</b>
<b>4.8 References .....</b>	<b>72</b>

## Table Directory

Table 4.1	Constraint Induced Movement Therapy for Upper Extremity Rehabilitation Post ABI
Table 4.2	Hand Splinting and Stretching for Upper Extremity Rehabilitation Post ABI
Table 4.3	Interventions for Fine Motor Rehabilitation Post ABI
Table 4.4	Virtual Reality Interventions for the Rehabilitation of Upper Extremities
Table 4.5	Partial Body Weight Supported Gait Training for Lower Extremity Rehabilitation Post ABI
Table 4.6	Multimodal Interventions for Lower Extremity Rehabilitation Post ABI
Table 4.7	Virtual Reality Interventions for Lower Extremity Post-ABI
Table 4.8	Virtual Reality Interventions for Upper and Lower Motor Rehabilitation Post ABI
Table 4.9	Aerobic Training for Lower Extremity Rehabilitation Post ABI
Table 4.10	Botulinum Toxin for the Treatment of Spasticity Post ABI
Table 4.11	Percutaneous Phenol Block for the Treatment of Spasticity Post ABI
Table 4.12	Electrical Stimulation for the Treatment of Spasticity Post ABI
Table 4.13	Oral Antispasticity Agents for the Treatment of Spasticity Post ABI
Table 4.14	Intrathecal Baclofen for the Treatment of Spasticity Post ABI
Table 4.15	Casting Techniques for the Treatment of Spasticity Post ABI
Table 4.16	Hand Splinting and Stretching for the Treatment of Spasticity Post ABI
Table 4.17	Multimodal Interventions for the Treatment of Spasticity Post ABI.
Table 4.18	Interventions for the Treatment of Visual Dysfunction Post ABI
Table 4.19	Interventions for the Treatment of Vestibular Dysfunction Post ABI
Table 4.20	Cognitive Behavioural Therapy for Pain Management Post ABI
Table 4.21	Cold Therapy for Pain Management Post ABI
Table 4.22	Antiepileptic Medications to Treat Pain Post TBI (Gould, 2007; Guay, 2003; Zasler et al., 2011)
Table 4.23	Antidepressants to Treat Pain Post ABI (Gould, 2007; Zasler et al., 2011)
Table 4.24	Topical Anesthetics to Treat Pain Post TBI (Gould, 2007)

## Figure Directory

Figure 1: Treatment for Sub-acute/Chronic Pain Post Traumatic Brain Injury

### Abbreviations

ABI	Acquired Brain Injury
BPPV	Benign Paroxysmal Positional Vertigo
BTX-A	Botulinum Toxin Type A
CBT	Cognitive Behavioural Therapy
CIMT	Constraint Induced Movement Therapy
NRS	Numeric Rating Scale
PTH	Post Traumatic Headaches
RCT	Randomized Controlled Trial
TBI	Traumatic Brain Injury
UMNS	Upper Motor Neuron Syndrome
VAS	Visual Analog Scale

## Key Points

Constraint induced movement therapy may improve function and use of the affected upper limb post ABI.

Overnight hand splinting may not improve upper limb function post ABI.

Soft hand splinting, but not manual therapy, may be beneficial for improving hand opening post ABI.

Functional dexterity tasks may be superior to tabletop fine motor control activities for improving fine motor coordination post ABI.

Gesture recognition biofeedback and visual feedback-based training may improve fine motor function post ABI.

Virtual reality interventions may be an effective intervention for the recovery of upper extremity function post ABI.

Partial body weight supported gait training likely does not improve ambulation, mobility, or balance when compared to conventional gait training post ABI.

Robotic assisted treadmill training may be similar to manually assisted treadmill training at improving gait speed and mobility post ABI.

Electrical muscle stimulation with passive exercise may improve lower extremity muscle atrophy post ABI.

Sit-to-stand training and Intensive Mobility Training may improve lower extremity motor function post ABI.

Virtual reality can be used for the remediation of motor function in the lower extremities post-ABI.

Virtual reality training likely improves balance in individuals post ABI, however it may not be more effective than conventional physiotherapy programs.

Aerobic exercise programs, whether home-based or in the community, appear to improve motor function and balance post-ABI.

Further research is needed in order to determine which components of exercise are the most effective for motor rehabilitation post-ABI.

Botulinum toxin type A injections, whether through a single point or multisite, likely reduce localized spasticity following ABI.

Phenol blocks of the musculocutaneous nerve may help decrease spasticity and improve range of motion temporarily up to five months post injection in individuals with ABI.

Electrical stimulation may acutely (24 hours) decrease spasticity in patients post ABI.

Oral baclofen appears to reduce lower extremity, but not upper extremity, spasticity in individuals with an ABI.

Bolus injections of intrathecal baclofen likely produce short-term reductions in upper and lower extremity spasticity and improvements in walking performance post ABI.

Prolonged intrathecal baclofen may reduce upper and lower extremity spasticity long-term post ABI.

Serial casting likely improves contractures and spasticity in individuals with an ABI compared to stretching; however, contracture improvement may not be maintained long-term.

Below-knee casting and stretching might increase passive ankle dorsiflexion in patients post ABI.

Serial below-knee casting may improve ankle range of motion and muscle extensibility in patients post TBI; however, this intervention may be associated with tissue breakdown.

Serial casting, whether for a short or long duration, might improve range of motion in individuals with an ABI. However, short duration casting may have a lower complication rate than long duration.

Hand splinting combined with stretching may be an effective treatment for spasticity and range of motion.

Botulinum toxin injections in combination with casting may be as effective as casting alone at reducing leg spasticity in patients post ABI.

Electrical stimulation in combination with tilt table standing and splinting may acutely improve spasticity (6 weeks) in patients post ABI.

Neural tension technique may be just as effective as random passive movement for improving lower extremity spasticity post ABI.

Computer based restitution training and rehabilitation programs directed at improving visual function likely improve the vision of those who sustain a TBI.

Base-in prisms and bi-nasal occluders may be effective in treating ambient vision disturbances.

Saccadic oculomotor rehabilitation may improve eye movements and reading in patients post ABI.

Combined aerobic dance and slide and step programs may improve balance and coordination post TBI.

A vestibular rehabilitation program may improve symptoms of vertigo in patients following TBI.



Cognitive behavioural therapy may be useful in managing post-traumatic headaches; however, may not be useful for headache-associated pain.

Cold therapy is likely not as effective as manual therapy at reducing post traumatic headache pain in patients post TBI.

## 4. Motor and Sensory Impairment Rehabilitation Post Acquired Brain Injury

### 4.1 Introduction

The primary cause of motor impairment and movement dysfunction post acquired brain injury (ABI) is upper motor neuron syndrome (UMNS), which can result in positive symptoms of enhanced stretch reflexes (spasticity) and released flexor reflexes in the lower limbs, such as the Babinski sign and mass synergy patterns, as well as negative symptoms including loss of dexterity and weakness (Mayer, 1997). These symptoms of UMNS have physiological implications for muscles that may subsequently develop stiffness and contractures, thereby further negatively affecting effective movement (Mayer, 1997).

For UMNS following brain injury, both the extent and timing of the individual's symptoms should be considered when deciding on a course of action. Focal or diffuse spasticity may appear following an ABI and frequently follow common patterns in the upper and lower limbs (Mayer, 1997). Time post injury is another important consideration as spontaneous neurological recovery may continue for 9 to 15 months post injury. However, the potential for functional motor recovery beyond that point is possible through medical interventions, such as the correction of a deformity or the use of pharmacological agents that allow for improved motor control (Mayer et al., 1996). Motor impairment can also result from the independent effects of prolonged immobilization and bed rest during the acute period. Prolonged immobility affects multiple body systems, although it is the direct effect on the musculoskeletal and cardiovascular systems that impact motor function the most (Bushbacher & Porter, 2000).

Following diffuse central nervous system injury there are potential impairments involving the cognitive, behavioural, and physical domains. It is the physical domain that is emphasized early on within the rehabilitation process, as most acute in-patient rehabilitation programs focus on the improvement of activities of daily living (ADLs) a patient can perform— as assessed by outcome measures such as the Functional Independence Measure or the Barthel Index (Linacre et al., 1994; McDowell, 2006). The emphasis on physical impairments during rehabilitation is common because both the patient and family members are more likely to recognize and acknowledge physical impairments, in contrast to cognitive and behavioural impairments.

This module reviews the available evidence pertaining to interventions for motor and sensory rehabilitation following ABI.

### 4.2 Motor Impairment

Motor rehabilitation is a common focus of interventions provided to an individual post ABI. Motor rehabilitation is essential in helping the patient return to performing their ADLs, thus reestablishing independence post ABI. The following sections evaluate the interventions currently available for upper and lower extremity motor impairment, including spasticity.

#### 4.2.1 Upper Extremity Interventions

Upper limb motor impairments are common in individuals with an ABI (Lannin et al. 2003). Interventions for the upper limb can focus broadly on arm mobility or on more specific outcomes such as finger

dexterity. Despite the importance of upper extremity rehabilitation post ABI, there are limited studies evaluating available interventions.

#### 4.2.1.1 Constraint Induced Movement Therapy

Constraint induced movement therapy (CIMT) is an intervention directed at improving the function of the more affected upper extremity following brain injury. The two primary components involve: 1) intensive motor training of the more affected upper extremity and 2) motor restriction of the less affected upper extremity (Dettmers et al., 2005). CIMT originated from research suggesting that the affected limb post brain injury is negatively impacted by “learned non-use” due to increased dependence on the intact limb (Grotta et al., 2004).

Although there is evidence in the stroke population to suggest that CIMT is clinically effective, many patients do not qualify for this type of therapy, which requires voluntary extension of the wrist and fingers, due to limited movement in the affected upper extremity. A further significant limitation of CIMT is the amount of resources required for its implementation (Grotta et al., 2004). Two studies evaluating the effect of CIMT post traumatic brain injury (TBI) were identified (Table 4.1).

**Table 4.1 Constraint Induced Movement Therapy for Upper Extremity Rehabilitation Post ABI**

Author/Year/ Country/ Study Design/N	Methods	Outcomes
<a href="#">Shaw et al.</a> (2005) USA Pre-Post N=22	<p><b>Population:</b> TBI; Mean Age=39.3 yr; Gender: Male=14, Female=8; Mean Time Post Injury=8.9 yr.</p> <p><b>Intervention:</b> Participants received constraint induced movement therapy (CIMT; 6 hr, 5 days/wk for 2 wk) in the laboratory engaging in massed practice shaping or task specific procedures while wearing a protective safety mitt on their less-affected upper limb (UL) for ≥90% of the time. Participants were encouraged to use the mitt outside the lab as well.</p> <p><b>Outcome Measure:</b> Fugl Meyer (FM) Motor Performance Assessment, Wolf Motor Function Test (WMFT), and Motor Activity Log (MAL).</p>	<ol style="list-style-type: none"> <li>1. Significant improvements in real-world use across all post-intervention testing occasions as measured by the MAL (mean change=1.6, <math>p&lt;0.001</math>).</li> <li>2. Significant post-treatment improvements in more affected UL FM scores (mean change=4.2, <math>p&lt;0.001</math>), and WMFT scores (mean change=0.4, <math>p&lt;0.01</math>).</li> <li>3. Based on the FM scores, the largest gains were in the upper arm, compared to the hand or wrist.</li> <li>4. Based on a median split (57%) of adherence to mitt wearing outside the lab, less-adherent participants had smaller treatment gains than those who were more-adherent.</li> <li>5. On the MAL, less adherent participants showed a trend towards smaller gains than more adherent subjects (<math>p=0.065</math>).</li> </ol>
<a href="#">Page &amp; Levine</a> (2003) USA Pre-Post N=3	<p><b>Population:</b> TBI; Mean Age=21 yr; Gender: Male=2, Female=1.</p> <p><b>Intervention:</b> Physical and occupational therapy sessions (30 min each, 3x/wk for 10 wk) were provided. The less affected upper limb was also restrained (5 hr/day for 5 days/wk) using modified constraint induced therapy (mCIT).</p> <p><b>Outcome Measure:</b> Action Research Arm Test (ARAT), Motor Activity Log (MAL), and Wolf Motor Function Test (WMFT).</p>	<ol style="list-style-type: none"> <li>1. Pre-intervention subjects exhibited learned non-use (MAL, Amount of Use scores &lt;1.0).</li> <li>2. After the intervention, MAL scores improved: Amount of use=2.0 and quality of use=2.2.</li> <li>3. Subjects 1, 2 and 3 had functional improvements on the ARAT (14.0, 5.5, and 6.0 respectively) and the WMFT (1.15, 1.7 and 1.35 respectively).</li> </ol>

## Discussion

The effectiveness of modified CIMT was studied by Page and Levine (2003), with participants showing improvements in both the amount and quality of use of the more affected limb. CIMT was also studied by Shaw et al. (2005) and showed similar results. Significant improvements were seen in both laboratory and real world spontaneous use of the more affected upper limb following two weeks of CIMT (Shaw et al. 2015). Although all participants benefited from the intervention, the gains made by those placed in the “less adherent” group were strongly correlated with the participant’s degree of adherence (Shaw et al., 2005). This correlation was not evident in the “more adherent” group; with the authors suggesting that adherence beyond a certain level does not contribute to additional benefits (Shaw et al., 2005). The gains were maintained at one month, however use of the affected limb decreased by 21% at two years post treatment. Given these two studies, CIMT for the upper extremity appears to have a positive impact on upper limb motor recovery post ABI.

### Conclusions

***There is level 4 evidence that constraint induced movement therapy (CIMT) or modified CIMT may improve upper extremity function in individuals post ABI.***

**Constraint induced movement therapy may improve function and use of the affected upper limb post ABI.**

#### 4.2.1.2 Hand Splinting and Stretching

The purpose of hand splinting following an ABI is to prevent contractures and deformities, and to reduce spasticity. There are biomechanical and neurophysiologic rationales for splinting the spastic hand (Lannin et al., 2003); the biomechanical approach attempts to prevent contractures by physically preventing shortening of muscle and connective tissues. Conversely, the neurophysiologic approach is based on the concept that the splint can inhibit reflexive contraction of the muscle. Ultimately, the aim is to reduce deformity and contractures in the hand (Table 4.2).

**Table 4.2 Hand Splinting and Stretching for Upper Extremity Rehabilitation Post ABI**

Author/Year/ Country/Study Design/ PEDro Score/ N	Methods	Outcomes
<a href="#">Thibaut et al.</a> (2015) Belgium RCT PEDro=4 N=17	<b>Population:</b> TBI=7, Anoxia=5, Aneurysm=5; Mean Age=41 yr; Gender: Male=9, Female=8; Mean Time Post Injury=35 mo; Severity: Severe. <b>Intervention:</b> Participants were randomized to receive one of the following exercise protocols on each of their upper limbs: manual stretching and control (no Intervention) (G1, n=8), soft splinting and control (G2, n=12), or soft splinting and manual stretching (G3, n=14). Each exercise was done for 30 min followed by a 60 min break. Outcomes were assessed before (T1) and after (T2)	<ol style="list-style-type: none"> <li>1. In G1, there were no significant changes in MAS, MTS, ROM, or HO after stretching or after the control protocol.</li> <li>2. In G2, the mean MAS score of flinger flexor muscles improved significantly after splinting from T1 to T2 (p=0.014) and the improvement was maintained at T3 (p=0.022). There was no significant change for the control.</li> <li>3. In G3, the mean MAS score of finger flexor muscles improved significantly after both splinting (p=0.014) and stretching (p=0.022) from T1 to T2, but neither improvement was maintained at T3.</li> <li>4. In G2, the mean HO score improved significantly after splinting from T1 to T2 (p=0.009), but the</li> </ol>

Author/Year/ Country/Study Design/ PEDro Score/ N	Methods	Outcomes
	each protocol, and after each break (T3). <b>Outcome Measures:</b> Modified Ashworth Scale (MAS), Modified Tardieu Scale (MTS), Range of Motion (ROM), and Hand Opening (HO).	improvement was not maintained at T3. There was no significant change for the control. 5. In G3, the mean HO score improved significantly after splinting ( $p=0.005$ ) from T1 to T2, but the improvement was not maintained at T3. There was no significant change in mean HO score after stretching ( $p=0.249$ ). 6. In G3 and G2, there were no significant changes in MTS or ROM after the interventions.
<a href="#">Lannin et al. (2003)</a> Australia RCT PEDro=8 N=28	<b>Population:</b> ABI; Gender: Male=13, Female=15. <i>Experimental Group (n=17):</i> Mean Age=65 yr; Mean Time Post Injury=47 days. <i>Control Group (n=11):</i> Mean Age=68 yr; Mean Time Post Injury=57 days. <b>Intervention:</b> The experimental group wore an immobilizing hand splint in a functional position ( $10^{\circ}$ - $30^{\circ}$ wrist extension) for 4 wk, for no longer than 12 hr each night. The control group received standard care (motor training and stretching). <b>Outcome Measure:</b> Length of wrist and finger flexor muscles, Hand and arm function, and Visual Analog Scale for Pain (VAS), and Motor Assessment Scale (MAS).	1. Effects of splinting were not statistically significant. 2. Splinting increased wrist extension by a mean of $1^{\circ}$ post intervention and reduced wrist extension by a mean of $2^{\circ}$ at follow-up. 3. Splinting decreased upper-limb function after intervention (MAS; mean 0.3 points) and at follow-up (mean 0.8 points). 4. Splinting decreased performance of hand movements by a mean of 0.4 points (MAS) post intervention and 0.5 points at follow-up. 5. Splinting decreased overall upper-limb function by 0.1 points after intervention and by 0.2 points at follow-up (MAS). 6. Increased reported intensity of upper-limb pain (mean: 0.2cm) on VAS after intervention and by 1cm at follow-up.

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

## Discussion

One study evaluated the effect of night time hand splinting in conjunction with conventional therapy compared to therapy alone (Lannin et al., 2003). Overall, the results did not demonstrate significant benefits of nocturnal hand splinting. A second randomized controlled trial (RCT) compared manual stretching, soft hand splinting, and manual stretching plus soft hand splinting to determine the optimal intervention (Thibaut et al., 2015). Results suggested that soft hand splinting for 30 minutes resulted in improved hand opening and reduced spasticity of the flexor finger muscles, however improvements in hand opening were not maintained after the break period. The hand splint was said to be feasible to use in daily care, as the splint was comfortable and easy to apply. There is a need to further research the effect of splinting in individuals with ABI as this practice is used in both acute and rehabilitation settings.

## Conclusions

***There is level 1b evidence that nocturnal hand splinting may not improve upper extremity range of motion or function compared to standard care in individuals post ABI.***

***There is level 4 evidence that soft hand splinting, but not manual therapy, may improve hand opening in individuals post ABI.***

**Overnight hand splinting may not improve upper limb function post ABI.**

**Soft hand splinting, but not manual therapy, may be beneficial for improving hand opening post ABI.**

#### 4.2.1.3 Interventions for Fine Motor Coordination

As discussed previously, the negative symptoms of UMNS, independent of spasticity, include: weakness, slowness of movement, and loss of finger dexterity (Mayer, 1997). Although gross motor function may return early in the recovery period following an ABI, fine motor deficits may persist and present a considerable challenge for both the individual and the clinicians treating them. The following studies highlight some of the treatment modalities that are being utilized to improve fine motor ability post ABI (Table 4.3).

**Table 4.3 Interventions for Fine Motor Rehabilitation Post ABI**

Author/Year/ Country/Study Design/PEDro Score/N	Methods	Outcomes
<a href="#">Neistadt</a> (1994) USA RCT PEDro=5 N=45	<b>Population:</b> TBI=42, Anoxia=3; Mean Age=33.2 yr; Gender: Male=45, Female=0, Mean Time Post Injury=7.9 yr. <b>Intervention:</b> Participants were assigned to either the parquetry block assembly (n=22) or the meal preparation group (n=23). Subjects received individual training sessions (3, 30 min sessions for 6 wk) in addition to their regular program. <b>Outcome Measure:</b> WAIS-R (Block Design Test), Parquetry Block Test, RKE-R, and Jebsen-Taylor Test of Hand Function.	<ol style="list-style-type: none"> <li>For picking up small objects with the dominant hand, the meal preparation group showed a significantly greater improvement over the puzzle group (p=0.027).</li> <li>There was no significant difference in improvement between the two groups for simulated page turning with dominant hand (p=0.655), simulated page turning with the non-dominant hand (p=0.182), and picking up small objects with the non-dominant hand (p=0.265).</li> </ol>
<a href="#">Yungher &amp; Craelius</a> (2012) USA PCT N=19	<b>Population:</b> TBI=8, Stroke=4, Healthy Subjects=7; <i>Experimental Group</i> (n=12): Mean Age=39.8 yr; Gender: male=8, female=4. <i>Healthy Control Group</i> (n=7): Mean Age=46.4 yr. <b>Intervention:</b> The use of gesture recognition biofeedback (GRB), which uses surface muscle pressures of the forearm to provide real-time visual biofeedback, was compared to standard repetitive training without feedback. Measures were completed before and after each condition. <b>Outcome Measure:</b> Nine-hole peg test (HPT).	<ol style="list-style-type: none"> <li>HPT scores for the experimental group ranged from 28.6 to 263 sec, and 15.78 to 25.56 sec for the control group.</li> <li>For those with impairments (n=12), in training with feedback there was an average decrease in HPT time to completion of 15.5%, and with no feedback there was an increase in time by 2.07%.</li> <li>For those with impairments, GRB training resulted in an improvement of 27.3% (p&lt;0.05), without the GRB training there was a 2.07% decline in performance.</li> <li>In the controls, GRB training has minimal effect. The time to completion was faster in this group, compared to the impaired group, at baseline, and with and without feedback (p&lt;0.05).</li> </ol>
<a href="#">Kriz et al.</a> (1995) Germany	<b>Population:</b> TBI=3, Stroke=2, Intracerebral bleeding=3, Viral	<ol style="list-style-type: none"> <li>No significant change in control subject's performance (p&gt;0.10). 9 of 10 participants with</li> </ol>

Pre-Post N=27	<p>Encephalitis=1, Cerebral Abscess=1, Healthy Controls=17. Gender: Male=17, Female=10; <i>Impaired Group (n=10)</i>: Mean Age=33.8 yr; Mean Time Post Injury=14.7 mo. <i>Healthy Control group (n=17)</i>: Age Range=22-42 yr.</p> <p><b>Intervention:</b> Patients completed a feedback-based training intervention that involved tracking moving targets with grip force, using a precision grip. Patients trained over 10 weekly 30 min sessions. Training terminated when normal performance was achieved.</p> <p><b>Outcome Measure:</b> Force control using grip strength, Tracking errors, and Transfers.</p>	<p>impairments reduced tracking errors significantly (<math>p&lt;0.05</math>) and improved in transfer tasks (<math>p&lt;0.05</math>).</p> <p>2. Impaired initial performance and improvement was not uniform and could be attributed to individual aspects of force control.</p>
------------------	---	---

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

### Discussion

Neistadt (1994) examined fine motor coordination in a group of adult men with TBIs after two types of coordination retraining activities: tabletop activities (i.e., peg board activities, puzzles etc.) and functional activities (i.e., meal preparation). The study results suggested that functional activities may be more effective than table top activities in promoting fine motor coordination in persons with brain injury, as indicated by the improvement in “picking up small objects with the dominant hand” that the meal preparation group experienced (Neistadt 1994). Another study found that visual feedback-based training of grip force is beneficial for individuals post brain injury (Kriz et al., 1995). More specifically, a light weight force transducer was held between the pulp of index finger and thumb of the impaired hand. In response to visual cues delivered via computer monitor, all tasks involved the gradual increase and decrease of grip force in training and transfer protocols. Regardless of the individual pattern of impairments, all but one patient succeeded in improving their tracking performance and transferring regained capabilities to other tasks (Kriz et al. 1995).

The most recent fine motor coordination study compared the use of gesture recognition biofeedback to standard repetitive training without feedback (Yungher & Craelius, 2012). The results from the study showed a significant decrease in task completion time for those who received feedback, in comparison to those who did not. This intervention is both simple to execute (e.g., no precise placement of sensors, etc.) and the assessment is straightforward. The authors suggest that this intervention leads to improvements in fine motor function of the hand with minimal supervision (Yungher & Craelius, 2012). Despite these studies, there is limited evidence to guide clinical practice in this area.

### Conclusions

***There is level 2 evidence that functional retraining activities may be more effective than tabletop fine motor control retraining activities for improving fine motor function in the dominant hand in individuals post ABI.***

***There is level 4 evidence that visual feedback-based grip force training may improve tracking accuracy and transfer tasks in individuals post ABI.***



*There is level 2 evidence that gesture recognition biofeedback may improve fine motor function compared to standard repetitive training without feedback in individuals post ABI.*

**Functional dexterity tasks may be superior to tabletop fine motor control activities for improving fine motor coordination post ABI.**

**Gesture recognition biofeedback and visual feedback-based training may improve fine motor function post ABI.**

#### 4.2.1.4 Virtual Reality for Upper Extremity Rehabilitation

With the advancement of technology, virtual reality is now a viable motor rehabilitation intervention for individuals following an ABI.

**Table 4.4 Virtual Reality Interventions for the Rehabilitation of Upper Extremities**

Author/Year/ Country/Study Design/PEDro Score/N	Methods	Outcomes
<a href="#">Mumford et al.</a> (2012) Australia Pre-Post N=9	<p><b>Population:</b> Severe TBI; Mean Age=30.9 yr; Gender: Male=5, Female=4; Mean Time Post Injury=33.8 mo.</p> <p><b>Intervention:</b> Participants had two pre-intervention assessments (4 wk apart), then received the Virtual Reality (VR) intervention, followed by a follow-up assessment. The intervention consisted of 12, 1-hr training sessions with the Elements virtual reality system (VR therapy, tracking camera, and tangible working LCD surface), over 4 wk in addition to their usual care.</p> <p><b>Outcome measure:</b> System-measured variables, Box and Block Test (BBT), McCarron Assessment of Neuromuscular Dysfunction (MAND), Neurobehavioural Functioning Inventory (NFI).</p>	<ol style="list-style-type: none"> <li>1. The intervention provided significant improvements on accuracy percentage for both left (46.26 to 64.25; <math>p=0.01</math>) and right hand (56.86 to 73.62; <math>p=0.02</math>).</li> <li>2. No significant changes were seen from pre to post treatment on left hand speed, but there was for right hands (0.23 m/s to 0.31 m/s; <math>p=0.01</math>).</li> <li>3. Efficiency scores improved significantly only for the right hands (92.61 to 97.68; <math>p=0.002</math>).</li> <li>4. BBT showed significant improvements from pre to post test for both the left (30.44 to 35.98; <math>p=0.04</math>) and right (46.66 to 53.33; <math>p=0.007</math>) hands.</li> <li>5. No significant improvements were noted on the MAND.</li> <li>6. From pre to post treatment, significant improvements in total NFI scores were demonstrated with a reduction from 128.67 to 112.89 (<math>p=0.005</math>), however in each subscale, only the memory/attention subscale improved significantly (<math>p=0.049</math>).</li> </ol>
<a href="#">Sietsema et al.</a> (1993) USA Prospective Controlled Trial N=20	<p><b>Population:</b> TBI; Mean Age=31.6 yr; Gender: Male=17, Female=3; Mean Time Post Injury=6 yr.</p> <p><b>Intervention:</b> Two interventions were compared: an Occupational Embedded Intervention and rote exercise. The occupational embedded intervention involved</p>	<ol style="list-style-type: none"> <li>1. There were no significant order effects.</li> <li>2. There was a significant increase in range of motion concerning hip to wrist movement in the occupational embedded condition compared with the rote exercise group (mean reach length 71.60 cm versus 59.38 cm, <math>p&lt;0.001</math>).</li> </ol>



	<p>leaning forward and reaching out the affected arm to play a computer-controlled game. The rote exercise involved leaning forward and reaching out the affected arm on command. Each participant had two 20 min sessions, separated by 1 wk.</p> <p><b>Outcome Measure:</b> Range of motion (trunk inclination, shoulder flexion, elbow extension), Total Movement (leaning forward and reaching)</p>	<p>3. The occupational embedded group had a range of motion for scapula-to-wrist that was a mean of 3.52cm greater than the rote exercise group; however, this was not statistically significant.</p>
--	---	---

### Discussion

Mumford et al. (2012) used virtual reality therapy with an interactive LCD surface and tracking cameras over 12 1-hour sessions. The authors found that accuracy and dexterity improved significantly in both upper extremities, but speed and efficiency only improved significantly for the right arms of patients. Sietsema et al. (1993) reported that individuals who used a computer-controlled game aimed at improving reaching had better range of motion in the hip and wrist than individuals who completed rote exercise. Despite the study being performed in 1993, the game used by Sietsema et al. (1993) is still available for use.

### Conclusions

***There is level 2 evidence that virtual reality training may improve neurobehavioral functioning as well as reaching accuracy and movements post-ABI.***

**Virtual reality interventions may be an effective intervention for the recovery of upper extremity function post ABI.**

#### 4.2.2 Lower Extremity Interventions

Outcomes targeted by lower extremity interventions following ABI tend to be gait and balance related. Gait improvement can be beneficial for re-establishing independence post ABI. Current methods being used for lower extremity rehabilitation include — but are not limited to — casting, orthosis, and partial body weight supported gait training.

##### 4.2.2.1 Partial Body Weight Supported Gait Training

Movement disorders post ABI decrease the independence of a person due to loss of ambulation. The inability to maintain an erect posture, due to a lack of sufficient strength and balance, may prevent the training necessary for the restoration of self-ambulation following brain injury. Partial body weight supported gait training is postulated to result in earlier gait rehabilitation and earlier weight-bearing to increase strength and reduce spasticity. Additionally, this gait intervention allows for the simulation of task-specific walking movements and enables rehabilitation therapists to assist patients in the components of gait, rather than focusing on bearing the patient's body weight. This type of gait training physically supports patients in a way that does not generate compensatory ambulation strategies that may develop while using a cane or a walker (Seif-Naraghi & Herman, 1999). In addition, partial body

weight support reduces the demands on muscles while the patient works on improving the coordination of the movement. Body weight support can be gradually adjusted, as the patient improves, to encourage postural control and balance (Table 4.4).

**Table 4.5 Partial Body Weight Supported Gait Training for Lower Extremity Rehabilitation Post ABI**

Author/Year/ Country/Study design/PEDro Score/N	Methods	Outcome
<a href="#">Esquenazi et al.</a> (2013) USA RCT PEDro=4 N=16	<p><b>Population:</b> TBI; Gender: Male=7, Female=9. <i>Robotic-Assisted Group (n=8):</i> Mean Age=37.1yr; Mean Time Post Injury=140.3mo. <i>Manually Assisted Group (n=8):</i> Mean Age=41.9yr; Mean Time Post Injury=150.4mo.</p> <p><b>Intervention:</b> All participants received gait training for 45 min, 3 x/wk for a total of 18 sessions. The training was either Robotic-Assisted Treadmill Training (RATT) or Manually Assisted Treadmill Training (MATT).</p> <p><b>Outcome Measure:</b> Over ground walking self-selected velocity (SSV), Maximal Velocity, Spatiotemporal Symmetry, 6-minute Walk Test (6MWT), and Stroke Impact Scale.</p>	<ol style="list-style-type: none"> <li>1. For the RATT group, SSV increased by 49.8% (p=0.01), maximal velocity by 14.9% (p=0.01), step length asymmetry ratio improved during SSV by 33.1% (p=0.01), and the 6MWT improved by 11.7% (p=0.21).</li> <li>2. For the MATT group, SSV increased by 31% (p=0.06), maximal velocity increased 30.8% (p=0.01), step-length asymmetry ratio improved during SSV by 9.1% (p=0.73), and the 6MWT improved by 19.3% (p=0.03).</li> <li>3. No significant between group differences were found for any of the outcome measures.</li> </ol>
<a href="#">Wilson et al.</a> (2006) USA RCT PEDro=7 N=38	<p><b>Population:</b> TBI; Mean Age=29.6 yr; Gender: Male=35, Female=3; Mean Time Post Injury: Experimental Group (n=19)=4 mo, Control Group (n=19)=2.8 mo.</p> <p><b>Intervention:</b> Patients in the control group received standard physical therapy for 8wk. The experimental group had physical therapy supplemented with partial weight-bearing gait training twice weekly.</p> <p><b>Outcome Measure:</b> Functional Independence Measure and Functional Assessment Measure (FIM+FAM), Rivermead Mobility Index (RMI), Gross Motor Subscale (GMS), Standing Balance Scale (SBC), Functional Ambulation Category (FAC).</p>	<ol style="list-style-type: none"> <li>1. The control group had significant improvements on the SBC (p&lt;0.0039), FAC (p&lt;0.0002), RMI (p&lt;0.0001), GMS (p&lt;0.0005), and FIM+FAM (p&lt;0.0002).</li> <li>2. The experimental group had significant improvements on the SBC (p&lt;0.002), FAC (p&lt;0.0002), RMI (p&lt;0.0009), GMS (p&lt;0.0015), and FIM+FAM (p&lt;0.0039).</li> <li>1. No between group differences were found for the SBC, FAC, RMI, GMS, or the FIM+FAM.</li> </ol>
<a href="#">Brown et al.</a> (2005) USA RCT PEDro=5 N=20	<p><b>Population:</b> TBI; Mean Age=40.2 yr; Gender: Male=14, Female=6; Mean Time Post Injury=15.8 yr.</p> <p><b>Intervention:</b> Patients received either body weight support treadmill training (BWSTT; n=10) or conventional over-ground gait training (COGT; n=9) for 15 min plus 30 min of exercise 2 days/wk for 3 mo.</p> <p><b>Outcome Measure:</b> Functional Ambulation Category, Functional Reach, Timed Up and Go, Gait velocity, Stride Width, Left-Right Step Length differential.</p>	<ol style="list-style-type: none"> <li>1. Step Length Differential improved significantly more for the COGT group than for the BWSTT group after 3mo of intervention (p=0.011).</li> <li>3. There were no other significant differences between groups at baseline or after 3mo of intervention for any of the outcome measures.</li> </ol>

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

## Discussion

Brown et al. (2005) conducted an RCT in which 20 ABI patients were randomized to either body weight supported treadmill training or conventional over-ground gait training. The authors reported that body weight supported treadmill training provided no additional benefit over conventional gait training in measures of ambulation following three months of training. However, it was noted that conventional gait training improved Step Length Differential significantly more than the body weight supported treadmill training. Similarly, in another RCT, Wilson et al. (2006) randomized 40 ABI patients to either standard physical therapy or physical therapy supplemented with partial body weight-bearing gait training. The authors also reported that although each group made functional improvements, there were no significant between-group differences on measures of balance, ambulation, and mobility at the end of the eight-week training period (Wilson et al., 2006). Once again, Esquenazi et al. (2013) compared robotic assisted treadmill training to manually assisted treadmill training for individuals with TBI. The researchers noted that while both interventions resulted in significant improvement in gait parameters, there were no differences between the two interventions for gait velocity, endurance, or mobility. From these studies, it appears that body weight supported gait training is not superior to more conventional methods.

### **Conclusions**

***There is level 2 evidence that body weight supported treadmill training may not improve ambulation or mobility compared to conventional gait training in individuals post ABI.***

***There is level 1b evidence that physical therapy with partial weight-bearing gait training may not improve ambulation, mobility, or balance compared to standard physical therapy in individuals post ABI.***

***There is level 2 evidence that robotic assisted body weight supported treadmill training may not improve ambulation or gait velocity compared to manually assisted treadmill training in individuals post ABI.***

**Partial body weight supported gait training likely does not improve ambulation, mobility, or balance when compared to conventional gait training post ABI.**

**Robotic assisted treadmill training may be similar to manually assisted treadmill training at improving gait speed and mobility post ABI.**

#### **4.2.2.2 Multimodal Interventions**

Multimodal interventions provide an opportunity to compare or combine interventions to better evaluate rehabilitation options. Combining interventions allows multiple physical impairments to be targeted in a single program, while comparing them assists in determining the relative effect of each therapy for motor rehabilitation (Table 4.5).

**Table 4.6 Multimodal Interventions for Lower Extremity Rehabilitation Post ABI**

Author/Year/ Country/Study design/PEDro/N	Methods	Outcome
---	---------	---------

<p><a href="#">Clark et al.</a> (2012) Australia RCT PEDro=3 N=42</p>	<p><b>Population:</b> <i>Experimental Group (n=17):</i> TBI=11, Stroke=5, Multiple Sclerosis=1; Mean Age=38.7 yr; Gender: Male=10, Female=7; Median Time Post Injury=9 mo. <i>Control Group (n=25):</i> Healthy controls; Mean Age=27.8 yr; Gender: Male=16, Female=9.</p> <p><b>Intervention:</b> All participants performed 7 alternative gait training methods in a randomized order. Methods included: therapist manual facilitation, use of gait assistive device, treadmill walking with handrail support, and 4 variations of body weight-support treadmill training with combinations of handrail and/or therapist support.</p> <p><b>Outcome Measure:</b> Mediolateral Center of Mass Movement, Stride Time, Stability of Movement.</p>	<ol style="list-style-type: none"> <li>2. Body weight-support treadmill training without any additional support resulted in greater amplitude, altered timing, and reduced movement stability compared with nonpathologic gait.</li> <li>3. Manual facilitation by the therapist most closely matched nonpathologic gait for timing and stability.</li> <li>1. The use of therapist facilitation or handrail support reduced the effect and resulted in treadmill training having lower movement amplitudes when compared to other methods of training.</li> </ol>
<p><a href="#">Canning et al.</a> (2003) Australia RCT PEDro=7 N=22</p>	<p><b>Population:</b> Severe TBI; Gender: Male=16, Female=6. <i>Experimental Group (n=12):</i> Mean Age=24.75 yr; Mean Time Post Injury=75.25 d. <i>Control Group (n=10):</i> Mean Age=25.6 yr; Mean Time Post Injury=84.6 d.</p> <p><b>Intervention:</b> Patients were divided randomly into either a regular rehabilitation program group (control) group or the intervention group which received the rehabilitation program as well as 4wk of intensive training of sit-to-stand and step-up exercises.</p> <p><b>Outcome Measure:</b> Sit-to-stand repetitions, Peak Oxygen Consumption (exercise capacity), Oxygen Consumption Workload Test (exercise efficiency).</p>	<ol style="list-style-type: none"> <li>1. The experimental group performed a mean of 87 repetitions of sit-to-stand and 42 repetitions of step-ups per working day.</li> <li>2. The intervention group had a 62% improvement in the number of sit-to-stands performed in 3min (motor performance) compared to 18% increase in the control group (p=0.03).</li> <li>3. There were no significant between-group differences in the improvements made in exercise capacity (p=0.36) or efficiency (p=0.38).</li> <li>4. The increase in exercise capacity for the intervention group was significant with an increase in VO<sub>2</sub>peak from 0.75 L/min to 1.14 L/min (p&lt;0.01).</li> </ol>
<p><a href="#">Peters et al.</a> (2014) USA Pre-Post N=10</p>	<p><b>Population:</b> TBI; Median Age=35.4 yr; Gender: Male=6, Female=2; Median Time Post Injury=9.9 yr.</p> <p><b>Intervention:</b> Participants went through 20 days of intensive mobility training (5 days/wk for 4wk). Sessions included gait training with body-weight-supported treadmill, balance activities, strength coordination, and range of motion training.</p> <p><b>Outcome Measure:</b> Berg Balance Scale, Dynamic Gait Index (DGI), 10 Metre Walk Test (10MWT), 6 Minute Walk Test (6MWT), 30 sec Sit-to-Stand test, Timed Up and Go (TUG) test, Walking While Talking Test average errors/alternating letters, Falls Efficacy Scale (FES), Quality of Life after Brain Injury, Global Rate of Change Scale, Fatigue.</p>	<ol style="list-style-type: none"> <li>1. The average session was 150.1±2.7 min.</li> <li>2. Fatigue scores ranged from 0 to 2.5 (out of 10) before sessions and 3 to 5.5 after.</li> <li>3. From pre-test to post-test, significant improvements were seen for the FES (p=0.01), DGI (p=0.049), 10MWT (p=0.03), TUG (p=0.01), and 6MWT (p=0.03).</li> <li>1. From pre-test to follow-up (3 mo), significant improvements were sustained for the 10MWT (p=0.02) and the TUG (p=0.03).</li> </ol>
<p><a href="#">Hirose et al.</a> (2013) Japan</p>	<p><b>Population:</b> TBI=8, Stroke=7; Gender: Male=11, Female=4. <i>Control Group (n=6):</i> Mean</p>	<ol style="list-style-type: none"> <li>2. There was a significant difference in the rate of atrophy between the EMS and control group, the</li> </ol>

PCT N=15	Age=59.8 yr. <i>Intervention Group (n=9)</i> : Mean Age=49.9 yr. <b>Intervention:</b> The control group received passive exercise and the intervention group received electrical muscle stimulation (EMS) weekly (30 min with stimulation cycles of 10 sec) in addition to passive exercise for 6 wk. <b>Outcome measure:</b> Rate of Atrophy.	EMS group showing less, in all 4 compartments (anterior and posterior thigh and leg) at day 14 ( $p<0.001$ ). 5. At 6wk the cross-sectional area was examined again, showing a significant difference between groups, with the EMS group showing less atrophy. ( $p<0.001$ ).
-------------	--	--

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

## Discussion

Canning et al. (2003) in a single blinded RCT compared the addition of an intensive sit-to-stand training program to a traditional rehabilitation program. The experimental group demonstrated an increased ability to repeat sit-to-stand within a defined time frame in comparison to the traditional rehabilitation group, however there were no differences between groups in the increase of exercise capacity or efficiency. Hirose et al. (2013) used passive exercise as a control, and compared it with electrical muscle stimulation (EMS) to determine the effects of EMS on muscle atrophy in the lower limbs. The use of EMS resulted in significantly reduced amounts of atrophy when compared to passive exercise.

Clark et al. (2012) demonstrated that using body-weight-support treadmill training with handrail support reduces the amount of center of mass displacement and movement instability. However, they also noted that support alters timing and variability components of gait patterns. Although the study explored seven gait training methods, Clark et al. (2012) concluded that no one method provides the optimal stimulus and that combining various methods may be the most beneficial. Peters et al. (2014) identified that with intensive therapy using body-weight-support treadmill training, balance activities, strength coordination, and range of motion activities, individuals can significantly improve their walking speed and Timed Up and Go test scores. The benefits lasted up to three months post intervention.

## Conclusions

***There is level 1b evidence that sit-to-stand training combined with usual rehabilitation may improve motor performance in sit-to-stand tasks compared to usual rehabilitation in individuals post ABI.***

***There is level 2 evidence that electrical muscle stimulation with passive exercise may reduce lower extremity muscle atrophy compared to passive exercise in individuals post ABI.***

***There is level 4 evidence that Intensive Mobility Training may improve ambulation and mobility in individuals post ABI.***

**Electrical muscle stimulation with passive exercise may improve lower extremity muscle atrophy post ABI.**

**Sit-to-stand training and Intensive Mobility Training may improve lower extremity motor function post ABI.**

#### 4.2.2.3 Virtual Reality for Lower Extremity Rehabilitation

In addition to providing support for the rehabilitation of upper extremity function, virtual reality interventions have also been used to examine their efficacy on lower extremity remediation.

**Table 4.7 Virtual Reality Interventions for Lower Extremity Post-ABI**

Author/Year/ Country/Study Design/N	Methods	Outcome
<a href="#">Cuthbert et al.</a> (2014) USA RCT PEDro=6 N=20	<p><b>Population:</b> TBI; Gender: Male=13, Female=7; Range of Time Post Injury=24-122 days.</p> <p><b>Intervention:</b> Participants were randomly assigned to either Extra Standard Balance Care (ESC; n=10) (standard physical therapy) or Virtual Reality (VR) balance therapy (n=10) using the Nintendo Wii. Both groups received standard physical therapy 4 x/wk. The ESC group had an additional 15 min of balance-specific therapy and the VR therapy group had 15 min of balance training using the Wii Fit.</p> <p><b>Outcome Measure:</b> Physical Activity Enjoyment Scale (PACES), Berg Balance Scale (BBS), Functional Gait Assessment (FGA).</p>	<ol style="list-style-type: none"> <li>1. There was no statistically significant difference between therapy groups on PACES scores at mid-treatment (<math>p=0.59</math>) or at treatment completion (<math>p=0.34</math>).</li> <li>2. The VR therapy group had a significant improvement on the BBS over time (0.19 points per day, <math>p=0.03</math>); however, there were no significant between group differences (VR therapy had a 1.13 point higher improvement than the ESC group, <math>p=0.70</math>).</li> <li>3. Within group improvements were found on the FGA (ESC=0.20, <math>p=0.01</math> and VR therapy=0.23, <math>p&lt;0.01</math>); however, there was no statistically significant between group difference found (<math>p=0.73</math>).</li> </ol>
<a href="#">Foo et al.</a> (2013) Australia Post-Test N=20	<p><b>Population:</b> TBI=11, Tumour=3, Stroke=2, Cerebral Palsy=2, SCI=1, Anoxic Brain Injury=1; Mean Age=43.3 yr; Mean Time Post Injury=23.3 mo.</p> <p><b>Intervention:</b> Participants completed two tasks (static standing and sit-to-stand) three times each, with and without visual feedback. Feedback was provided using the Wii Balance Board.</p> <p><b>Outcome measure:</b> Weight-bearing Asymmetry.</p>	<ol style="list-style-type: none"> <li>1. During the static balance task, weight-bearing asymmetry was significantly reduced with visual feedback (<math>p=0.005</math>).</li> <li>2. There was no significant difference with visual feedback for the dynamic test (<math>p=0.737</math>); however, those with higher weight-bearing asymmetry were the most responsive to feedback.</li> </ol>

#### Discussion

Cuthbert et al. (2014) also demonstrated a significant within-group improvement on balance using virtual reality-based therapy; however, the gains made using this intervention were not significantly different compared to participants receiving standard physical therapy. Finally, during static balance tasks, visual feedback provided using a Wii Balance board helped reduce weight-bearing asymmetry (Foo et al., 2013).

#### Conclusions

*There is level 1b evidence that virtual reality training compared to balance training may not be more effective for improving lower extremity function post-ABI. However, virtual reality training was shown to improve function independently.*

*There is level 4 evidence that visual feedback may reduce weight-bearing asymmetry in the lower extremities post-ABI.*

Virtual reality can be used for the remediation of motor function in the lower extremities post-ABI.

#### 4.2.3 Combined Upper and Lower Extremity Interventions

Unlike the studies referenced above, some programs combine interventions that treat both the upper and lower extremities. One of the challenges with combined rehabilitation is the choice of testing method. With a larger range of potential outcomes, it can be difficult to choose the proper test to evaluate the effect of the intervention.

##### 4.2.3.1 Virtual Reality

Virtual reality training has been gaining popularity in recent years. The advancement of virtual reality treatments has partly been influenced by commercial availability of programs, such as the Wii Fit Balance Board, that provide reliable testing and virtual reality games (Foo et al. 2013). Despite the increasing availability of virtual reality programs, there are a limited number of studies evaluating their efficacy (Table 4.8).

**Table 4.8 Virtual Reality Interventions for Upper and Lower Motor Rehabilitation Post ABI**

Author/Year/ Country/Study Design/N	Methods	Outcome
<a href="#">Ustinova et al.</a> (2014) USA Pre-Post N=30	<b>Population:</b> TBI; Mean Age=30.6 yr; Gender: Male=10, Female=5; Mean Time Post Injury=6.1 yr. <b>Intervention:</b> Participants had completed physical therapy previously and had reached a plateau. All participants received virtual reality (VR) therapy which was a series of VR games that re-trained whole-body coordination, posture, and gait. All games allowed for advancement into more difficult levels. Therapy was a total of 15 sessions, each 50-55 min (typically 2-3 sessions/wk, over 5-6 wk). <b>Outcome Measure:</b> Berg Balance Scale (BBS), Functional Gait Assessment (FGA), Functional Reaching Test (FRT).	<ol style="list-style-type: none"> <li>1. BBS scores increased by a mean of 4.5 points (<math>45.6 \pm 5.15</math> to <math>50.2 \pm 4.4</math>, <math>p &lt; 0.01</math>).</li> <li>2. FGA scores improved by a mean of 4.6 points (<math>20.3 \pm 5.6</math> to <math>24.9 \pm 4.6</math>, <math>p &lt; 0.05</math>).</li> <li>3. FRT scores increased by a mean reaching distance of 2.3 inches (<math>12.5 \pm 2.3</math> to <math>14.8 \pm 2.3</math>, <math>p &lt; 0.01</math>).</li> </ol>
<a href="#">Schafer &amp; Ustinova</a> (2013) USA Prospective Controlled Trial N=30	<b>Population:</b> TBI=15, Healthy Controls=15. <i>TBI Group (n=15):</i> Mean Age=35.3 yr; Gender: Male=6, Female=9; Mean Time Post Injury=6.6 yr; <i>Control Group (n=15):</i> Mean Age=33.4 yr; Gender: Male=7, Female=8. <b>Intervention:</b> Participants completed reach activities in a physical environment (PE; reach to the farthest point possible) and a virtual environment (VE; touching furthest flower seen on the screen with hand avatar). VE touches were done from 50° and 10° angles. In	<ol style="list-style-type: none"> <li>1. The control group showed greater endpoint displacement amplitude (<math>p &lt; 0.01</math>) and COM displacement (<math>p &lt; 0.01</math>) than the TBI group.</li> <li>2. Reaches were performed more slowly among participants with TBI, but the difference between groups was not significant (<math>p &gt; 0.05</math>).</li> <li>3. Reaching amplitude was ~9% further for both groups in the VE than the PE (<math>p &lt; 0.05</math>).</li> <li>4. For both groups, reaches were farther in the PE after performing in the VE. The TBI group increased their reach by ~5% (<math>p &lt; 0.05</math>).</li> </ol>



Author/Year/ Country/Study Design/N	Methods	Outcome
	each setting, three reaches were completed with the dominant hand. <b>Outcome measure:</b> Centre of Mass (COM) Displacement, Endpoint Displacement Amplitude, Movement Time, Peak Velocity.	

### Discussion

Virtual reality interventions have been shown to be beneficial for improving balance post ABI. Ustinova et al. (2014) had participants complete 15 sessions of virtual reality therapy targeting the recovery of postural and coordination abnormalities, and demonstrated significant improvements for balance and dynamic stability following treatment. Schafer and Ustinova (2013) compared reaches in the physical environment after having participants with TBI and controls practice reaches in a virtual environment. Reaching distances in the physical environment increased for both groups, but a greater effect was noted among those with TBI.

### Conclusions

*There is level 1b evidence that virtual reality-based training may not improve balance and gait compared to standard physical therapy in individuals post ABI.*

*There is level 4 evidence that virtual reality therapy may improve balance, gait, and functional reaching in individuals post ABI.*

**Virtual reality training likely improves balance in individuals post ABI, however it may not be more effective than conventional physiotherapy programs.**

#### 4.2.4 Exercise Programs

Following an ABI, motor impairments in combination with cognitive impairment can have a significant impact on functional abilities (Boake et al., 2000). Unlike the more uniform focal deficits seen following stroke, the motor deficits following ABI tend to be diverse. These deficits include: impairment of force, endurance, coordination, and balance (Boake et al., 2000). Frequently, rehabilitation efforts are directed at specific motor impairments with the aim of improving overall functional ability.

##### 4.2.4.1 Aerobic Training

Many ABI patients have gone through a period of prolonged bed rest as a result of comorbid injuries or a prolonged loss of consciousness; consequently, cardiovascular changes, muscular atrophy, and loss of lean body mass commonly occur (Boake et al., 2000). General fitness training following ABI has the potential to influence multiple outcomes beyond the mere direct physical benefits such as improved aerobic capacity (Bushbacher & Porter, 2000). When comparing individuals with TBI that exercise to those that do not, the exercisers were less depressed, had less symptoms and better self-reported health status than non-exercising brain injury survivors (Gordon et al., 1998). The following studies examined the effectiveness of aerobic training on motor outcomes in individuals with an ABI (Table 4.9).



Table 4.9 Aerobic Training for Lower Extremity Rehabilitation Post ABI

Author/Year/ Country/Study design/PEDro/N	Methods	Outcome
<a href="#">Hassett et al. (2009)</a> Australia RCT PEDro=7 N=62	<p><b>Population:</b> Severe TBI; <i>Fitness Center Group</i> (n=32): Mean Age=35.4 yr; Gender: Male=27, Female=5; Median Time Post Injury=2.6 mo. <i>Home-Based Group</i> (n=30): Mean Age=33 yr; Gender: Male=26, Female=4; Median Time Post Injury=2.3 mo.</p> <p><b>Intervention:</b> Participants were randomly assigned to either an exercise intervention group at a fitness-center or to a home-based exercise group. Fitness center participants were supervised by a personal trainer (1 hr, 3 x/wk, 12 wk), whereas the home-based exercise group followed an exercise plan prescribed before discharge and were monitored by a physiotherapist. Assessment at baseline, end of intervention and 3mo follow-up.</p> <p><b>Outcome Measure:</b> Modified 20-metre Shuttle Test (MST), Depression Anxiety Stress Scale, Profile of Mood States (POMS), Sydney Psychosocial Reintegration Scale (SPRS), Brain Injury Community Rehabilitation Outcome.</p>	<ol style="list-style-type: none"> <li>On average the fitness center group had better adherence than the home-based group (77% versus 44%, <math>p \leq 0.001</math>). The fitness center group completed a mean of 2.4 sessions/wk compared to the home group who completed 0.5 sessions/wk.</li> <li>At the end of the program, both groups improved their fitness levels on the MST; however, there were no significant differences between groups (<math>p &gt; 0.05</math>).</li> <li>Those in the fitness centre group achieved a significantly greater percentage of goals at the end of the intervention (76% versus 52%, <math>p = 0.005</math>), but this difference diminished at follow-up (<math>p = 0.650</math>).</li> <li>No significant differences were noted when comparing psychosocial functioning or community integration measures between groups except for the POMS Confusion-Bewilderment (<math>p = 0.007</math>) and the SPRS Living Skills (<math>p = 0.009</math>) subscales at the end of intervention only, with greater improvements in the fitness center group.</li> </ol>
<a href="#">Hassett et al. (2011)</a> Australia RCT follow-up N=30	<p><b>Population:</b> Severe TBI=30; Mean Age=33 yr; Gender: Male=26, Female=4; Mean Time Post Injury=2.3 mo.</p> <p><b>Intervention:</b> An in-home exercise program (36 sessions over 12 wk) was completed in a previous study. Participants were then retrospectively divided into adherers (n=10) and non-adherers (n=20) and compared.</p> <p><b>Outcome Measure:</b> Modified 20-metre Shuttle Test, Wechsler Memory Scale III, Wechsler Adult Intelligence Scale III, Controlled Oral Word Association Test, Depression Anxiety and Stress Scale.</p>	<ol style="list-style-type: none"> <li>Non-adherers were significantly younger than adherers (30 versus 39 yr, <math>p = 0.04</math>).</li> <li>Results indicate that a greater number of participants in the adherence group reported walking or jogging pre-injury compared to non-adherers (7 versus 5, <math>p \leq 0.05</math>).</li> <li>A greater portion of adherers had extremely severe injuries compared to non-adherers (90% versus 50%, <math>p \leq 0.05</math>).</li> <li>There were no significant differences between groups on any of the cognitive functioning or psychological health measures.</li> </ol>
<a href="#">Driver et al. (2006)</a> USA RCT PEDro=4 N=18	<p><b>Population:</b> TBI; <i>Exercise Group</i> (n=9): Mean Age=37.8yr; Gender: Male=5, Female=4; Mean Time Post Injury=40.3 mo. <i>Control Group</i> (n=9): Mean Age=35.5 yr; Gender: Male=5, Female=4; Mean Time Post Injury=41.2 mo.</p> <p><b>Intervention:</b> Participants were randomly assigned to either an 8 wk aquatic exercise program involving 1 hr sessions 3 x/wk consisting of aerobic and resistance training or to a control group that received 8wk of vocational rehabilitation class to improve reading and writing skills.</p>	<ol style="list-style-type: none"> <li>The exercise group experienced significant improvements on the health responsibility, physical activity (both <math>p &lt; 0.05</math>), nutrition, spiritual growth (both <math>p &lt; 0.01</math>), and interpersonal relationships (<math>p &lt; 0.001</math>) subscales of the HPLP-II after the intervention, but not the stress management subscale. The control group showed no significant improvements on any the subscales (<math>p &gt; 0.05</math>).</li> <li>At the end of the program, the aquatic exercise group showed significant improvements on the self-esteem, co-ordination, body fat, strength, flexibility and endurance sub-scales of the PSDQ.</li> </ol>

Author/Year/ Country/Study design/PEDro/N	Methods	Outcome
	<b>Outcome Measure:</b> Health Promoting Lifestyle Profile II (HPLP-II), Physical Self-Description Questionnaire (PSDQ).	(all $p < 0.001$ ). The control group showed no significant improvements. 3. No between-group calculations were completed.
<a href="#">Bateman et al.</a> (2001) UK RCT PEDro=7 N=157	<b>Population:</b> TBI=44, Stroke=70, Subarachnoid Hemorrhage=15, Other=28; Gender: Male=97, Female=60. <i>Training Group (n=79):</i> Mean Age=41.7 yr; Mean Time Post Injury=22.2 wk. <i>Control Group (n=79):</i> Mean Age=44.7 yr; Mean Time Post Injury=25.5 wk. <b>Intervention:</b> Participants were divided into either an exercise intervention (intervention group, cycle training) or relaxation training (control group). The interventions were 30 min sessions, 3 x/wk for 12 wk. <b>Outcome Measure:</b> Peak Work Rate, Berg Balance Scale, Rivermead Mobility Index (RMI), Barthel Index, Functional Independence Measure (FIM), Nottingham Extended Activities of Daily Living (NEDLI).	1. The mean increase in peak work rate from baseline to 12 wk was 25.8W and 11.7W, for the training and control group, respectively ( $p=0.02$ ). 2. No significant differences were found between groups on the Berg Balance Scale, RMI, or the Barthel Index. 3. There was a trend towards significance, with the control group making greater improvements on the Berg Balance scale ( $p=0.06$ ) and RMI ( $p=0.07$ ) than the training group. 4. Greater FIM gains and improvements on the NEADLI were found for the control group between 12 and 24 wk ( $p < 0.05$ ) compared to the training group.
<a href="#">Charrette et al.</a> (2016) USA Pre-Post N <sub>Initial</sub> =16, N <sub>Final</sub> =14	<b>Population:</b> TBI=9, Stroke=3, Tumor=1, Encephalopathy=1; Mean Age=44.8yr; Gender: Male=12, Female=2; Mean Time Post Injury=20.5yr; Injury Severity: Moderate-Severe. <b>Intervention:</b> Participants took part in an intensive exercise program consisting of endurance, full-body strength, stretching, and balance exercises (3d/wk for 6wk). Assessments took place at baseline, 6wk (exercise completion) and 12wk (follow-up). <b>Outcome Measure:</b> 6-Minute Walk Test (6MWT), High Level Mobility Assessment Tool (HiMAT), 10-Metre Walk Test (10MWT).	1. There was a significant increase in distance walked from baseline (431ft) to 6wk (1016ft) and 12wk (712ft) during the 6MWT ( $p < 0.05$ ). 2. There was a significant increase in mobility from baseline (3.5) to 6wk (9) and 12wk (8) on the HiMAT ( $p < 0.05$ ). 3. There was a significant increase in gait velocity from baseline (0.59m/s) to 6wk (1.11m/s) and 12wk (1.10m/s) measured by the 10MWT ( $p < 0.05$ ).
<a href="#">Damiano et al.</a> (2016) USA Case Control N <sub>Initial</sub> =24, N <sub>Final</sub> =15	<b>Population:</b> TBI=12, Healthy Subject=12; <i>TBI group (n=12):</i> Mean Age=31.3 yr; Gender: Male=7, Female=5; Time Post Injury>6 mo. <i>Healthy Volunteers (controls; n=12):</i> Mean Age=32.5 yr; Gender: Male=7, Female=5. <b>Intervention:</b> Participants with TBI followed a home-based exercise program with an elliptical (30 min 5 days/wk for 8 wk). Resistance was added progressively each week. Controls did not complete the exercise intervention. Assessments were completed at baseline, 8 wk and follow-up. <b>Outcome Measure:</b> Limits of Stability Test (LOS), Motor Control Test (MCT), High-Level Mobility Assessment Tool (HiMAT), Hamilton Depression Inventory (HAM-D), Sensory	1. There was a significant difference in LOS between the TBI group and controls in 2 directions; backwards (TBI=71.6%, HV=89.3%, $p=0.042$ ) and left (TBI=37%, HV=49.6%, $p=0.037$ ). 2. The TBI group had a significantly poorer DT performance on both motor ( $p=0.047$ ) and cognitive ( $p=0.045$ ) tasks when compared to controls. 3. The TBI group performed significantly worse than controls on HVLT-R ( $p=0.004$ ), PCL-C ( $p=0.02$ ) and HAM-D ( $p=0.04$ ). 4. Within the TBI group, maximal movement during the LOS test had a strong relationship with HVLT-R total recall ( $r=0.74$ , $p=0.008$ ) and delayed recall ( $r=0.81$ , $p=0.003$ ) and was related

Author/Year/ Country/Study design/PEDro/N	Methods	Outcome
	Organization Test, Gait, Cadence, Dual-Task performance (DT), Hopkins Verbal Learning Test-Revised (HVLt-R), Finger Tapping Test (FTT), Pittsburgh Sleep Quality Index (PSQI), Beck Anxiety Inventory, PTSD Checklist-Civilian Version (PCL-C).	to fewer depressive symptoms ( $r=-0.63$ , $p=0.04$ ). 5. Within the TBI group, slower walking velocity and slower FTT was related to higher depression scores ( $r=-0.65$ , $p=0.03$ & $r=-0.72$ , $p=0.04$ , respectively). FTT was also related to poorer sleep quality ( $r=-0.75$ , $p=0.048$ ). 6. Within the TBI group, poorer DT performance was related to higher anxiety ( $r=0.71$ , $p=0.02$ ). 7. MCT and LOS improved following 8 wk of exercise and did not change at follow-up aside from increased LOS forward endpoint excursion ( $p=0.001$ ).
<a href="#">Ustinova et al. (2015)</a> USA Pre-Post N=22	<b>Population:</b> TBI; Mean Age=29.2 yr; Gender: Male=13, Female=9; Mean Time Post Injury=23.6 mo; Mean GCS=11.2. <b>Intervention:</b> Participants completed a therapeutic exercise program supervised by a physical therapist designed for retaining whole-body coordination, posture and gait. The program included twenty 30-40 min sessions, increasing to 55-60 min as the patient became more comfortable (4-5 d/wk for 4-5 wk). <b>Outcome Measure:</b> Berg Balance Scale (BBS), Functional Independence Measure (FIM), Functional Gait Assessment (FGA), Ataxia Scale.	1. There was a significant improvement in static and dynamic balance from the pre-test to post-test on the BBS (45.2 versus 49.2, $p=0.011$ ). 2. There was a significant improvement in gait, as measured by the FGA, from pre to post intervention (22.8 versus 26.9, $p=0.009$ ). 3. Ataxia symptoms significantly decreased from pre-test to post-test (7.3 versus 5.9, $p=0.012$ ) 4. There was no significant difference between pre and post-test on FIM.
<a href="#">Dault and Dugas (2002)</a> Canada PCT N=8	<b>Population:</b> TBI; Mean Age=29.6 yr; Gender: Male=6, Female=2; Mean Time Post Injury=44.4 mo. <b>Intervention:</b> An individualized 12 wk training program (TP; $n=5$ ) combining aerobic dance, and slide and step training for 30 min, 2 x/wk was compared to traditional muscular training (TMT; $n=3$ ) for 60 min, 2 x/wk for 12 wk. <b>Outcome Measure:</b> Test for Sensory Interaction in Balance (CTSIB).	1. Significant pre- and post-training differences were found in the temporal delay for the wrist ( $p<0.01$ ), knee improvement ( $p<0.001$ ), and sway area ( $p<0.05$ ) for the TP group; no significant changes were noted for the TMT group. 2. The temporal delay in the wrist was 83 ms in the TP group and 13 ms in the TMT group.

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

## Discussion

It appears that the introduction of an aerobic training program can have a positive influence on individuals post ABI. Furthermore, general aerobic programs have been found to improve balance (Dault and Dugas, 2002; Ustinova et al., 2015). It is important to note that despite improvements in balance, Ustinova and colleagues (2015) did not find improvements in functional independence after the intervention. This suggests that although exercise programs may improve physical fitness, gains in functional status often occur independently of aerobic exercise training. Charrette et al. (2016) conducted a study of intensive exercise programs, consisting of endurance and full body strength

training, for adults with chronic severe ABI. Results suggest that intensive combination of interventions improves gait distance and velocity, as well as mobility (Charrette et al., 2016).

Aquatic exercise was found to improve almost all subscales on the Health Promoting Lifestyle Profile, including interpersonal relationships, and also self-esteem— as measured by the Physical Self-Description Questionnaire (Driver et al., 2006). This study encourages participation in group exercise post ABI as it can foster feelings of well-being and self-esteem which could have a positive impact upon other rehabilitation strategies (Driver et al., 2006).

Bateman et al. (2001) compared cycling training (experimental group) to relaxation training (control group) and found that cycling training was associated with a significant improvement in exercise capacity; however, there was no significant difference between the groups in regards to balance, mobility, and functional independence (Bateman et al., 2001). This suggests that although exercise programs may improve physical fitness, gains in functional status often occur independently of aerobic exercise training (Bateman et al., 2001).

Hassett et al. (2012) examined the benefits of circuit training with encouragement from a physiotherapist and heart rate monitor feedback in individuals with severe TBI. More specifically, the intervention group had their heart rate monitor uncovered and it beeped when they did not reach their target heart rate, whereas the control group had their monitors covered and muted. Results indicate there was no significant difference between the two groups in terms of the amount of time spent in the heart rate target zone. Earlier Hassett et al. (2009) found individuals assigned to exercise programs showed significant improvement in their cardiorespiratory levels regardless of where they worked out (in a gym or at home) or how often (2.4 sessions per week versus 0.5 sessions per week). However, adherence to the program was higher among those attending a fitness center. When compliance was explored further, those with greater adherence were found to be older, more severely injured and had exercised before the injury (Hassett et al., 2011).

Hoffman et al. (2010) compared individuals who exercised in a community-based program to individuals who did not participate in this program; however, the controls were able to exercise on their own. Although the intervention group was working out more days per week than controls, the total amount of time spent exercising per week was similar between groups, making comparisons challenging. When those who were active (more than 90 minutes of activity per week) were compared to those who were not as active, the authors found that mood was significantly higher in the participants who were exercising for more than 90 minutes each week, regardless of what treatment group they were originally placed in. Thus, any physical exercise is beneficial to patients post ABI. Furthermore, home-based exercise programs have shown to improve depressive symptoms, stability, and gait following intervention (Bellon et al., 2015). It is important to note that lower stability and dual-tasking scores were associated with poorer mental health outcomes (Damiano et al. 2016).

## Conclusions

***There is level 1b evidence that participating in an exercise program at a fitness-center compared to home-based exercise program may lead to greater program adherence but not significantly different motor results in individuals post-ABI.***

*There is level 2 evidence that aerobic training compared to vocational rehabilitation may be more effective at improving co-ordination, strength, flexibility, and endurance in individuals post-ABI.*

*There is level 1b evidence that exercise programs may improve FIM scores, but not balance or mobility compared to relaxation training in individuals post-ABI.*

*There is level 4 evidence that multimodal exercise programs may improve gait and mobility in individuals post-ABI.*

*There is level 3 evidence that a home-based exercise program may improve stability to the level of healthy controls, but may not improve motor control, mobility, or dual-task performance in individuals post-ABI.*

*There is level 2 evidence that aerobic dance training compared to musculature training may improve sensory interaction and balance post-ABI.*

**Aerobic exercise programs, whether home-based or in the community, appear to improve motor function and balance post-ABI**

**Further research is needed in order to determine which components of exercise are the most effective for motor rehabilitation post-ABI.**

#### **4.2.5 Spasticity Interventions**

Spasticity is a common symptom encountered post ABI and is an element of UMNS. Spasticity has been formally defined as “a motor disorder characterized by a velocity-dependent increase in tonic stretch reflexes with exaggerated tendon reflexes, resulting from excitability of the stretch reflex” (Lance, 1980). Common features of spasticity include increased muscle tone, exaggerated tendon jerks, and clonus.

Management of spasticity is not unique to brain injury survivors, since it is often associated with other conditions affecting the central nervous system such as spinal cord injury and multiple sclerosis. Spasticity may require intervention when it interferes with functional abilities such as mobility, positioning, hygiene, or when it is the cause of deformity or pain. Factors that must be taken into consideration when proposing treatment of spasticity include chronicity of the problem, the severity, the pattern of distribution (focal versus diffuse), the locus of injury, as well as comorbidities (Gormley et al., 1997). Some studies have found that spasticity of cerebral origin versus spinal cord injury respond differently to the same medications (Katz & Campagnolo, 1993). Typically, the clinical approach to spasticity is to first employ treatments that tend to be less interventional and costly; however, multiple strategies may need to be administered concurrently.

##### **4.2.5.1 Botulinum Toxin Injections**

Botulinum toxin type A (BTX-A) acts at the pre-synaptic terminal to block acetylcholine release into the neuromuscular junction. When selectively injected into a specific muscle BTX-A is thought to cause local muscle paralysis, thereby alleviating hypertonia caused by excessive neural activity (Jankovic & Brin, 1991). It has been suggested that BTX-A may be useful in the treatment of localized spasticity if oral

interventions such as benzodiazepines, baclofen, dantrolene sodium, or tizanidine cause significant adverse effects (Gracies et al., 1997b). Currently, limited numbers of RCTs exist evaluating botulinum toxin for spasticity in individuals with an ABI (Table 4.10).

**Table 4.10 Botulinum Toxin for the Treatment of Spasticity Post ABI**

Author/Year/ Country/Study Design/PEDro Score/N	Methods	Outcome
<a href="#">Mayer et al. (2008)</a> USA RCT PEDro=6 N=31	<b>Population:</b> TBI=21, Stroke=8, Hypoxic encephalopathy=2; <i>Motor Point Group:</i> Mean Age=37.9 yr; Mean Time Post Injury=256.7 d. <i>Distributed Group:</i> Mean Age=34.7 yr; Mean Time Post Injury=481.9 yr. <b>Intervention:</b> Patients with severe elbow flexor hypertonia received one of two interventions: the motor point injection technique (1 site biceps and 1 site brachioradialis), or the distributed quadrants technique (4 sites rectangularly configured – 2 biceps and 2 brachioradialis). Following two baseline measures, each elbow was randomized to receive injections of Botox. In total 90 units were given to patients in each group; however the sites and injection techniques varied between the groups. Mean follow up was 23.5±4.4 days. <b>Outcome Measure:</b> The Ashworth Scale, Modified Tardieu Scale.	<ol style="list-style-type: none"> <li>1. The median decrease in Ashworth Scores after intervention was 1 point in both groups (<math>p=0.53</math>) and the Tardieu catch angle post intervention did not differ significantly between groups (<math>p=0.31</math>).</li> <li>2. Both groups showed significant improvement from baseline on all outcomes measured (all <math>p&lt;0.01</math>); however, there were no between-group differences at 3 wk.</li> <li>3. For both groups, a clinicophysiology effect was observed at 3 wk post-intervention.</li> </ol>
<a href="#">Intiso et al. (2014)</a> Italy Pre-Post N=22	<b>Population:</b> ABI=16, Cerebral Palsy=6; Mean Age=38.1 yr; Gender: Male=12, Female=10; <i>Brain Injury:</i> Mean Time Post Injury=3.8 yr. <b>Intervention:</b> Patients with severe spasticity of the upper and lower limbs received injections of incobotulinum toxin A (BoNT-A; up to 840 IU). <b>Outcome Measure:</b> Modified Ashworth Scale (MAS), Glasgow Outcome Scale (GOS), Frenchay Arm Test (FAT), Barthel Index (BI), Visual Analog Scale, Visual Analogue Scale–Pain (VAS).	<ol style="list-style-type: none"> <li>1. Seventeen patients had spastic hemiparesis and 5 had paraparesis.</li> <li>2. A significant reduction in spasticity was seen at 4 and 16wk post intervention, shown by a decrease in mean MAS scores in the elbow, wrist, finger and hand (all <math>p&lt;0.05</math>) and ankle (<math>p&lt;0.03</math>).</li> <li>3. No significant improvements were seen on the GOS, BI, or FAT at 4 or 16 wk.</li> <li>1. A significant reduction in pain was seen from baseline (<math>7.6\pm1.1</math>) to 4 (<math>3.5\pm0.7</math>) and 16 wk (<math>3.6\pm0.5</math>) post intervention (<math>p&lt;0.001</math>).</li> </ol>
<a href="#">Clemenzi et al. (2012)</a> Italy Pre-Post N=21	<b>Population:</b> TBI=11, ABI=10; Mean Age=42.2 yr; Gender: Male=16, Female=5; Median Time Post Injury=5 yr; Severity: Severe. <b>Intervention:</b> Patients received repeated injections of Botulinum Toxin Type A (maximum dose 600 U diluted in 50 ml <sup>-1</sup> ) followed by rehabilitation program that consisted of hand and/or foot adhesive taping maintained for 7 days and checked daily. <b>Outcome Measure:</b> Barthel Index (BI),	<ol style="list-style-type: none"> <li>1. Spasticity was in the lower limb in 33.3% of patients, upper limb in 9.5%, and both in 57.1%.</li> <li>2. MAS lowered at the follow up, and improvement in spasticity was seen at the second and last injection (T3) time points compared to baseline (<math>p&lt;0.0001</math>).</li> <li>3. BI significantly improved at follow up (T3) in relation to initial scores (<math>p=0.0001</math>).</li> <li>4. VAS score improved at the end of the second injection, a reduction in score was noted after each injection.</li> </ol>



Author/Year/ Country/Study Design/PEDro Score/N	Methods	Outcome
	Modified Ashworth Score (MAS), Visual Analogue Scale- pain (VAS).	5. Greater improvement on BI was correlated to a shorter period between ABI onset and first injection ( $p<0.0001$ ), the same effect was not discovered for MAS or VAS.
<a href="#">Yablon et al. (1996)</a> USA Case Series N=21	<p><b>Population:</b> TBI; Mean Age=28.2 yr; Gender: Male=12, Female=9; Mean Time Post Injury: Acute Group=142.7 days, Chronic Group=89.5 mo.</p> <p><b>Intervention:</b> Subjects received Botulinum Toxin A injections (20-40 units per muscle) into the upper extremity. Targeted muscles included: the flexor carpi radialis, flexor carpi ulnaris, flexor digitorum profundus, and flexor digitorum superficialis. Some patients also received injections into the biceps and brachialis due to coexisting spasticity in the elbow flexors. After injection, patients received therapeutic modalities as needed. Patients were grouped based on time between injury and injection: acute (&lt;12 mo; n=9) or chronic (<math>\geq 12</math> mo; n=12).</p> <p><b>Outcome Measure:</b> Modified Ashworth Scale (MAS), passive ROM at the wrist.</p>	<ol style="list-style-type: none"> <li>1. The acute group showed significant improvements in ROM (wrist extension improved by a mean of <math>42.9\pm 24.7^\circ</math>, <math>p=0.001</math>) and spasticity severity (mean MAS improvement <math>1.5\pm 0.5</math> points, <math>p=0.01</math>).</li> <li>2. All patients in the acute group showed an improvement in spasticity and no patient worsened or remained unchanged.</li> <li>3. The chronic group showed significant improvements in ROM (wrist extension improved by a mean of <math>36.2\pm 21.7^\circ</math>, <math>p&lt;0.001</math>) and spasticity severity (mean MAS improvement <math>1.47\pm 0.9</math> points, <math>p=0.002</math>).</li> </ol>

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

## Discussion

Five studies examining the effects of BTX-A on spasticity following ABI were identified. Intiso et al. (2014) showed a reduction in spasticity for the upper extremity (elbow, wrist, and hand), as well as ankle joints at one and four months post intervention. Although pain was also significantly reduced, no significant improvements in function were shown— as measured by the Glasgow Outcome Scale and the Frenchay Arm Test (Intiso et al., 2014). These findings were similar to those found by Yablon et al. (1996) who reported that BTX-A injections into the upper extremities improved range of motion and spasticity in 21 patients with ABI. These improvements were shown for patients who received the injections within one year of injury and also for those who received the injection more than one year post injury (Yablon et al., 1996). The time between injury and injection was also studied by Clemenzi et al. (2012). The results were similar to the previous study for pain and spasticity; however, the time between onset and injection did have an effect on functional outcomes. Patients with a shorter period of time between their injury and first injection had greater improvements on the Barthel Index (Clemenzi et al., 2012).

In terms of the administration of BTX-A, Mayer et al. (2008) found that a single motor point injection and multisite distributed injection resulted in similar outcomes, with both groups showing a clinical effect at three weeks post intervention.

## Conclusions

*There is level 4 evidence that botulinum toxin type A injections may be effective in the management of localized spasticity following ABI.*

*There is level 1b evidence that receiving botulinum toxin type A through a single motor point or multisite distributed injections are similar at reducing spasticity in individuals with an ABI.*

**Botulinum toxin type A injections, whether through a single point or multisite, likely reduce localized spasticity following ABI.**

#### 4.2.5.2 Nerve Blocking Agents

Local nerve blocks are a potential management solution in circumstances where there is muscle spasticity affecting only a few muscle groups in a focal pattern. Essentially, a nerve block involves the application of a chemical agent to impair nerve functioning. The effect of the chemical agent may be temporary or permanent (Katz et al., 2000). Temporary acting compounds include local anesthetic agents that block sodium ion channels, typically lasting only a few hours. Local anesthetic agents are used for diagnostic procedures or for assistance with activities such as casting (Gracies et al., 1997a). Agents used for permanent nerve blocks to treat spasticity last between 2 and 36 months, and include ethyl alcohol (>10%) and phenol (>3%). Complications of this type of block have included chronic dysesthesia, pain and permanent peripheral nerve palsies (Gracies et al., 1997a). Studies of nerve blocking agents to improve spasticity in individuals with an ABI are limited (Table 4.11).

**Table 4.11 Percutaneous Phenol Block for the Treatment of Spasticity Post ABI**

Author/Year/ Country/Study Design/N	Methods	Outcome
<a href="#">Keenan et al.</a> (1990) USA Case Series N=17	<b>Population:</b> TBI; Mean Age=25 yr; Gender: Male=12, Female=5; Mean Time Post Injury=6 mo. <b>Intervention:</b> Subjects received a phenol block (3ml of 5% phenol solution in sterile saline) followed by a daily program of active/passive range of motion therapy. Assessments conducted pre-post block, 24 hr after, then at weekly intervals while patients were hospitalized for rehabilitation. Post discharge follow-up occurred for a minimum of 2 yr. <b>Outcome Measure:</b> Muscle tone/ control, Range of Motion.	<ol style="list-style-type: none"> <li>1. Ninety-three percent of extremities showed a short term decrease in motor tone and improved resting position of the elbow.</li> <li>2. Maximum improvements occurred 4 wk post block.</li> <li>3. Resting position improved from 120° to 69°, active arc increased from 46° to 60°, and passive arc from 65° to 118°.</li> <li>4. At follow-up (mean 27 mo post injection), 9 extremities that had relief of spasticity, had recurrence of flexor tone and loss of motion in the elbow.</li> </ol>
<a href="#">Garland et al.</a> (1984) USA Case Series N=11	<b>Population:</b> TBI=11; Mean Age=24 yr; Gender: Male=8, Female=3; Mean Time Post Injury=5.8 mo. <b>Intervention:</b> Subjects received percutaneous phenol injections (1-2 ml of 3 or 5% phenol solution) at motor points of spastic wrist and finger flexors identified using a nerve stimulator. Injected muscles included: the flexor carpi radialis, flexor carpi ulnaris, flexor digitorum	<ol style="list-style-type: none"> <li>1. Mean resting position of the wrist prior to injection was 53°. Nine patients increased resting extension by a mean of 34° and 2 patients lost a mean of 15° of extension.</li> <li>2. Overall, there was a mean increase in resting wrist angle following motor point injections of 25°.</li> <li>3. Active wrist extension improved an average of 30°. Mean increase in passive wrist extension with finger flexed of 5°.</li> </ol>



Author/Year/ Country/Study Design/N	Methods	Outcome
	sublimus, flexor digitorum profundus, and flexor pollicis longus. <b>Outcome Measure:</b> Resting Angle of Wrist, Passive/active Extension of Wrist.	

### Discussion

We identified two studies which evaluated the efficacy of nerve blocks as a treatment for spasticity. Keenan et al. (1990) evaluated the effect of percutaneous phenol block of the musculocutaneous nerve to decrease elbow flexor spasticity. The results indicated that there was improved range of motion of the elbow lasting a mean of five months. In the second study, 11 closed head injury patients with spastic paralysis of the upper extremity were treated with percutaneous phenol injections into the spastic wrist and finger flexors (Garland et al., 1984). The authors reported that relaxation of muscle tone persisted for up to two months following the injections. Furthermore, there was a mean increase in resting wrist angle, active wrist extension, and passive wrist extension with fingers flexed of 25, 30, and 5°, respectively (Garland et al., 1984). Evidently, these studies found that percutaneous phenol blocks are effective in temporarily controlling spasticity in patients post TBI, however due to the retrospective nature of the studies and lack of controls there is insufficient evidence to make definitive conclusions on the efficacy of phenol injections.

### Conclusions

*There is level 4 evidence that phenol nerve blocks may reduce contractures and spasticity at the elbow, wrist, and finger flexors for up to five months post injection in individuals post ABI.*

**Phenol blocks of the musculocutaneous nerve may help decrease spasticity and improve range of motion temporarily up to five months post injection in individuals with ABI.**

#### 4.2.5.3 Electrical Stimulation

Electrical stimulation uses an electrical current to elicit a muscle contraction either directly by stimulating the skeletal muscle (Gregory & Bickel, 2005), or indirectly by stimulating the nerve supplying that muscle. Electrical stimulation has seen some applications with regards to assisting paraplegic patients with standing and walking (Katz et al., 2000). Reports from spinal cord injury populations suggest that electrical stimulation is associated with significant reductions in spasticity for up to 24 hours post stimulation (Halstead et al., 1993) (Table 4.10).

**Table 4.12 Electrical Stimulation for the Treatment of Spasticity Post ABI**

Author/Year/ Country/Study Design/N	Methods	Outcome
<a href="#">Seib et al.</a> (1994) USA Pre-Post	<b>Population:</b> TBI=5, Spinal Cord Injury=5; Mean Age=38 yr; Gender: Male=6, Female=4; Mean Time Post Injury=6.3 yr.	Ipsilateral Effect: 1. There was a significant reduction in spasticity immediately following simulation for all

Author/Year/ Country/Study Design/N	Methods	Outcome
N=10	<p><b>Intervention:</b> After baseline assessments, 20 min of Surface Electrical Stimulation to the ipsilateral (the more spastic side) tibialis anterior. Parameters: 2 sec rise time, 15 sec on, instant fall, 20 sec off. Rate of stimulation was 30 pulses/ sec. Intensity varied on subject tolerance. Assessments occurred at baseline, immediately post intervention, and 24hr after the intervention.</p> <p><b>Outcome Measure:</b> Path length, Spasticity Measurement System.</p>	<p>participants (<math>p&lt;0.05</math>); however, the change in path lengths pre to post stimulation was not significantly different in the TBI group alone (median length 82nm/rad before versus 73nm/rad after).</p> <p>2. Twenty-four hours after stimulation, ipsilateral path length (spasticity) reduced significantly in 8 of 9 subjects (<math>p&lt;0.01</math>).</p> <p>Contralateral Effect:</p> <p>3. Six of 9 participants showed increased contralateral path lengths immediately post intervention.</p> <p>4. TBI median path length increase was from 14nm/rad to 34nm/rad.</p> <p>5. Twenty-four hours post stimulation, 4 patients had decreased spasticity, 3 had an increase and 1 patient had no change.</p>

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

## Discussion

One study by Seib et al. (1994) was identified which examined the effects of electrical stimulation applied to the lower extremity in participants with either a TBI or spinal cord injury. Electrical stimulation significantly decreased spasticity in the stimulated extremity, whereas the tone in the non-stimulated extremity did not change. Furthermore, the effect of one stimulation session was noted to last up to 24 hours post intervention.

## Conclusions

***There is level 4 evidence that electrical stimulation may be effective for decreasing lower extremity spasticity for six or more hours, lasting up to 24 hours, in individuals post ABI.***

**Electrical stimulation may acutely (24 hours) decrease spasticity in patients post ABI.**

### 4.2.5.4 Oral Antispasticity Drugs

Oral agents are often used to manage spasticity particularly when a systemic agent to treat upper and lower extremity spasticity is required (Gracies et al., 1997b). Although antispasticity agents may be used for other medical conditions such as spinal cord injury or multiple sclerosis (Gracies et al., 1997b), the effectiveness of these agents should not be presumed to be similar for brain injury survivors. Multiple medications have been evaluated to treat spasticity of both cerebral and spinal cord origin. The more common medications include GABA agonists, that affect ion flux such as baclofen, benzodiazepines, dantrolene sodium, as well as agents that affect alpha-2 adrenergic receptors such as tizanidine and clonidine. The use of any of these drugs must be weighed against potential side effects, such as sedation, which are complicated by the cognitive and behavioural changes associated with brain injury (Table 4.11).

Table 4.13 Oral Antispasticity Agents for the Treatment of Spasticity Post ABI

Author/Year/ Country/Study Design/N	Methods	Outcome
<a href="#">Meythaler et al.</a> (2004) USA Case Series N=35	<p><b>Population:</b> TBI=22, ABI=6, Stroke=7; Mean Age=31 yr; Gender: Male=22, Female=13.</p> <p><b>Intervention:</b> Oral baclofen regimen beginning at 5 mg 3 x/day increased per protocol to 80 mg/day. Follow-up occurred between 1 and 3 mo after initiation of oral baclofen.</p> <p><b>Outcome Measure:</b> Ashworth Rigidity Scale (ARS), Spasm Frequency Scale (SFS), Deep Tendon Reflexes (DTR).</p>	<ol style="list-style-type: none"> <li>1. Mean dose was 57±26 mg/day for all patients and 55 ± 28 mg/day for patients with TBI.</li> <li>2. After treatment, extremity ARS (3.5±1.1 to 3.2±1.2, p=0.0003) and DTR scores (2.5±0.9 to 2.2±1.2, p=0.0274) decreased significantly.</li> <li>3. No significant changes in lower extremity spasm scores were observed.</li> <li>4. Patients with TBI saw a significant decrease in scores on the ARS (p=0.0044) and DTR (p=0.0003) but not on the SFS (p&gt;0.05).</li> <li>5. Upper extremities showed no significant changes for tone, spasm frequency, or reflexes (p&gt;0.05).</li> </ol>

### Discussion

Meythaler et al. (2004) completed a retrospective study evaluating the use of oral baclofen to manage spasticity in a mixed brain injury and stroke population. Pre and post testing revealed that oral baclofen improved spasticity in the lower extremity assessed using the Ashworth Rigidity Scale and Spasm Frequency Scale; however, no changes for tone, spasm frequency, or reflexes were found for the upper extremity (Meythaler et al., 2004). The authors suggest that the lack of effect may be due in part to receptor specificity issues. Of note, a common adverse effect of the oral baclofen was the onset of considerable sleepiness in 17% of patients (Meythaler et al., 2004).

Of note, Meythaler et al. (2001) completed a randomized, double blinded placebo controlled cross over trial examining tizanidine for the management of spasticity. This study evaluated both stroke (53%) and TBI (47%) survivors. For both lower and upper extremity, there was a significant decrease in the Ashworth scores on the affected side with the active drug compared to placebo. However, significant differences between interventions were not found for upper and lower extremity spasm and reflex scores. Overall the authors felt that tizanidine was effective in decreasing the spastic hypertonia associated with ABI; however, a common side effect was increased somnolence (41%) (Meythaler et al., 2001). Despite the study showing effectiveness, no level of evidence will be assigned for this drug due to more than 50% of the population being stroke.

### Conclusions

*There is level 4 evidence that oral baclofen may improve lower extremity spasticity, but not upper extremity spasticity, in individuals post ABI.*

**Oral baclofen appears to reduce lower extremity, but not upper extremity, spasticity in individuals with an ABI.**

#### 4.2.5.5 Intrathecal Baclofen

A limitation of oral baclofen is the inability to achieve sufficiently high concentrations in the cerebrospinal fluid (CSF) in order to modify spasticity without first causing significant sedation (Gracies et al., 1997b). Intrathecal baclofen refers to direct administration of baclofen into the intrathecal space and CSF at the lumbar level. For therapeutic treatment, a subcutaneous pump is required to provide continuous administration of the medication into the intrathecal space. This treatment procedure, however, is invasive and associated with complications including infection, pump failure, and tube complications such as kinking or disconnection (Gracies et al., 1997b) (Table 4.12).

**Table 4.14 Intrathecal Baclofen for the Treatment of Spasticity Post ABI**

Author/Year/ Country/Study Design/PEDro/N	Methods	Outcome
<a href="#">Meythaler et al. (1996)</a> USA RCT PEDro=7 N=11	<b>Population:</b> TBI=10, Anoxia=1; Mean Age=25yr; Gender: Male=9, Female=2. <b>Intervention:</b> Patients with chronic spastic hypertonia received either a bolus injection of intrathecal baclofen (50 µg) or placebo (normal saline). Crossover occurred a minimum of 48 hr later. Assessment at 1, 2, 4, and 6 hr post injection. <b>Outcome Measure:</b> Ashworth Scale (AS), Spasm Score, Deep Tendon Reflexes.	<ol style="list-style-type: none"> <li>For the lower extremity, after baclofen injection, AS scores decreased by a mean of 2 points (<math>p=0.0033</math>), spasm scores decreased by a mean of 2.1 points (<math>p=0.0032</math>), and reflex scores by 2.3 points (<math>p=0.0032</math>) at 4 h.</li> <li>For the upper extremity, after baclofen injection, AS scores decreased by a mean of 1.4points (<math>p=0.0033</math>), spasm scores by a mean of 1.2points (<math>p=0.0070</math>), and reflex scores by 1.0points (<math>p=0.0111</math>) at 4 h.</li> <li>No significant within-group differences were shown for placebo. Between group differences were significant for all measures for both lower and upper extremity (<math>p\leq 0.0272</math>).</li> </ol>
<a href="#">Wang et al. (2016)</a> Singapore Case Series $N_{Initial}=6$ , $N_{Final}=5$	<b>Population:</b> TBI=5, Encephalopathy=1; Mean Age=31.6 yr; Gender: Male=3, Female=2; Mean Time Post Injury=39.4 mo. <b>Intervention:</b> A retrospective review of patients that were recruited to undergo surgical implantation of an intrathecal baclofen (ITB) pump. After implantation patients received daily physical therapy. Upon discharge patients continued to receive regular outpatient rehabilitation therapies for 3 mo, and ITB pump refills and monitoring by the neurosurgical team for 3-4 mo. Outpatient follow-up was 3-6 mo. <b>Outcome Measure:</b> Modified Ashworth Scale (MAS).	<ol style="list-style-type: none"> <li>The mean reduction in MAS was 1.2 (SD 1.1; <math>p&lt;0.05</math>) at 3 mo and 1.0 (SD 1.2; <math>p=0.06</math>) at the last follow-up. All patients but 1 (no change) had significant reductions in spasticity.</li> </ol>
<a href="#">Chow et al. (2015)</a> Canada Pre-Post N=19	<b>Population:</b> TBI=11, Stroke=8; Mean Age=34.2 yr; Gender: Male=9, Female=10; Mean Time Post Injury=48.7 mo. <b>Intervention:</b> All patients underwent a 50-µg intrathecal baclofen (ITB) bolus injection via lumbar puncture. Patients were evaluated at baseline, 2 hr, 4 hr, and 6 hr post injection. <b>Outcome Measure:</b> Gait speed, stride length, cadence, stance duration, ankle range of motion (ROM)-stance & swing,	<ol style="list-style-type: none"> <li>There was no significant difference in gait speed, stride length, cadence, or stance duration across evaluation points.</li> <li>Ankle ROM in the more-affected leg during stance phase was significantly increased from baseline to 6 hr (<math>p=0.009</math>); however, was not significantly different during swing phase.</li> <li>Peak MG lengthening velocity significantly increased from baseline to 4 hr in the less-affected leg (<math>p=0.005</math>) and to 6 hr in both legs (<math>p\leq 0.01</math>).</li> </ol>

Author/Year/ Country/Study Design/PEDro/N	Methods	Outcome
	peak medial gastrocnemius (MG) lengthening velocity, average Ashworth Score, Plantar Flexors Ashworth Score, Electromyography-lengthening Velocity (EMG-LV), Coactivation Duration (CoD), Coactivation Index (CI).	<ol style="list-style-type: none"> <li>4. Average Ashworth Score and plantar flexors Ashworth scores were significantly different across all time posts in the more-affected leg only (<math>p&lt;0.001</math>).</li> <li>5. Compared with baseline, both frequency (<math>p=0.02</math>) and average gain (<math>p=0.007</math>) of EMG-LV were significantly lower at 2 hr post but did not reach the significance at 4 hr and 6 hr post (<math>p\leq0.040</math>).</li> <li>6. Slope parameters of EMG-LV in the less-affected leg did not change over time (<math>p\geq0.129</math>).</li> <li>7. CoD significantly decreased over time in the more affected leg during all phases of gait (<math>p\leq0.013</math>); and CoI did not significantly change over time in either leg (<math>p&gt;0.107</math>).</li> </ol>
<a href="#">Margetis et al. (2014)</a> Greece Pre-Post N=8	<p><b>Population:</b> TBI=6, Hydrocephalus=1, Cardiac Arrest=1; Mean Age=31.5 yr; Gender: Male=8, Female=0; Mean Time Post Injury=37.25 mo.</p> <p><b>Intervention:</b> Patients who were resistant to oral spasticity treatments received an implanted intrathecal baclofen pump. Mean follow-up period was 38.4 mo.</p> <p><b>Outcome Measure:</b> Modified Ashworth Scale.</p>	<ol style="list-style-type: none"> <li>1. All patients showed improvement in their spasticity scores; mean Modified Ashworth Scale scores were 3.375 pre- and 1.125 post-intervention.</li> </ol>
<a href="#">Posteraro et al. (2013)</a> Italy Pre-Post N=12	<p><b>Population:</b> TBI=8, Hemorrhage=2, Anoxia=2; Mean Age=36 yr; Gender: Male=9, Female=3; Time Post Injury Range=31-150 days.</p> <p><b>Intervention:</b> Patients not experiencing reductions in spasticity following initial interventions with oral baclofen received intrathecal baclofen (ITB). The initial dosage was 50 or 100 mcg depending on the severity of the impairment and was increased by 10% every 3 days until the maximum dosage of 800 mcg was achieved. Assessments occurred before the implant, and at 3 mo and 12 mo follow-ups.</p> <p><b>Outcome Measure:</b> Modified Ashworth Scale (MAS), Spasm Frequency Scale (SFS), Disability Rating Scale (DRS), Level of Cognitive Functioning (LCF).</p>	<ol style="list-style-type: none"> <li>1. Mean ITB dose for participants was 380mcg.</li> <li>2. Six patients received ITB within 3 mo of injury (early); 6 patients received ITB between 3 and 6mo post injury (late).</li> <li>3. At 3 mo, both spasticity and spasms significantly decreased compared to the baseline, based on MAS and SFS scores (<math>p&lt;0.001</math> and <math>p&lt;0.002</math>, respectively).</li> <li>4. At 3 mo, improvements in DRS and LCF were seen (<math>p&lt;0.001</math> and <math>p=0.002</math>, respectively).</li> <li>5. At 12 mo (<math>n=5</math>) all patients demonstrated further improvements in spasticity and spasms, but this was non-significant compared to results at 3 mo.</li> <li>6. There were no differences in global outcomes (DRS and LCF) between patients in early ITB initiation group and those in late ITB initiation group.</li> </ol>
<a href="#">Hoarau et al. (2012a)</a> France Post-Test N=43	<p><b>Population:</b> TBI; Mean Age=23.3yr; Gender: Male=33, Female=10; Mean GCS score=4.6.</p> <p><b>Intervention:</b> After initial injury, participants who were started on Intrathecal Baclofen Therapy (IBT) to treat dysautonomia and hypertonia and were</p>	<ol style="list-style-type: none"> <li>1. At follow-up, 9 participants had died, 13 were severely disabled or in an unresponsive wakefulness syndrome and 21 had a good recovery of consciousness.</li> <li>2. Mean CRS-R score was 18.9 (Range 1-23), mean BI score was 50.1 (Range 0-100), 34.9% were</li> </ol>

Author/Year/ Country/Study Design/PEDro/N	Methods	Outcome
	included for evaluation of long-term outcomes (mean 10±0.6 yr post implantation). <b>Outcome Measure:</b> Coma Recovery Scale-Revised (CRS-R), Modified Ashworth Scale (MAS), Barthel Index (BI).	living at home, and mean MAS for upper limb was 1.6 (Range 0-4). 3. Most of the participants who had a positive recovery received IBT later than the other participants. 4. Complications occurred in 62.8% of patients; the most common being operative site infections (20.9%) and overdoses with profound flaccidity, sedation and vomiting (16.3%).
<a href="#">Horn et al.</a> (2010) USA Pre-Post N=28	<b>Population:</b> TBI=12, Hypoxic Encephalopathy=3, Stroke=13; Mean Age=35 yr; Gender: Male=12, Female=16; Mean Time Post Injury=45 mo. <b>Intervention:</b> The subjects received a 50 µg bolus of baclofen injected into the lumbar intrathecal space. <b>Outcome Measure:</b> Ashworth Scale, Video-based Motion Analysis Program.	1. The range of motion (ROM) increased in the ankle on both the more involved side (13±6 versus 15±7, p=0.008) and the less involved side (22±8 versus 24±8, p=0.031) from baseline to post-injection. 2. ROM improvement occurred most often at 4 and 6 hr after injection (p<0.05). 3. There was a significant correlation between the magnitude of change in ROM at the time of peak response and the magnitude of gait speed change (r=0.1, p<0.001). 4. Significant reductions in Ashworth scores compared to baseline (2.0±0.5) at 2 hr (1.6±0.4), 4 hr (1.4±0.4) and 6 hr (1.3±0.3) post-injection (all p<0.001).
<a href="#">Stokic et al.</a> (2005) USA Case Series N=30	<b>Population:</b> TBI=17, Anoxic=4, Stroke=9; Mean Age=31 yr; Gender: Male=17, Female=13; Mean Time Post Injury=3 yr. <b>Intervention:</b> Participants received a single 50 µg intrathecal baclofen bolus injection via a lumbar puncture. <b>Outcome Measure:</b> Ashworth Scale, H-Reflex from Soleus Muscle, F waves from Abductor Hallucis in Supine Position.	1. Ashworth score on the more involved side significantly decreased between baseline (2.4±0.7) and 4 (1.5±0.6) and 6 hr (1.4±0.6) post-injection (p<0.001). 2. Maximal individual change in Ashworth scores ranged from 0 to 2.6 points (mean 1.0±0.7). 3. H/M ratio significantly decreased bilaterally (p<0.001). 4. F-wave persistence significantly decreased on the more involved side (p<0.05) with no change in F/M ratio.
<a href="#">Francisco et al.</a> (2005) USA Case Series N=14	<b>Population:</b> Anoxic Encephalopathy=6, TBI=5, Stroke=3; Mean Age=35.9 yr; Gender: Male=6, Female=8. <b>Intervention:</b> Patients were surgically fitted with an infusion pump for continuous intrathecal baclofen delivery. This took place a mean of 5.62 mo (range 2-12 mo) post injury. Follow up occurred at a mean of 13.9 mo post pump implantation. <b>Outcome Measure:</b> Modified Ashworth Scale (MAS), Disability Rating Scale (DRS).	1. Participants received a mean daily intrathecal baclofen dose of 591.5 µg (93-2000.2µg). 2. From baseline to follow-up, the mean decrease in MAS scores for upper extremities was 1±1.4 (p<0.020) and lower extremities was 2.1±1.4 (p<0.001). 3. The changes in DRS scores were not significant.
<a href="#">Horn et al.</a> (2005) USA Pre-Post N=28	<b>Population:</b> TBI=12, Stroke=13, Hypoxic Encephalopathy=3; Mean Age=35 yr; Gender: Male=12, Female=16; Mean Time Post Injury=45 mo.	1. Mean change in hip and knee range of motion (ROM) during gait was less than ±2° after injection.



Author/Year/ Country/Study Design/PEDro/N	Methods	Outcome
	<p><b>Intervention:</b> Subjects received a single 50 µg intrathecal baclofen bolus injection via lumbar puncture.</p> <p><b>Outcome Measure:</b> Walking Performance, Ashworth scores.</p>	<ol style="list-style-type: none"> <li>2. ROM in ankles increased from baseline to post-injection on both the more involved (13° versus 15°, <math>p&lt;0.010</math>) and less involved side (22° versus 24°, <math>p&lt;0.050</math>).</li> <li>3. For all joints (<math>n=168</math>), ROM significantly improved in 42%, significantly worsened in 34%, and did not change in 24%.</li> <li>4. Significant reductions in Ashworth scores compared to baseline (<math>2.0\pm0.5</math>) at 2 hr (<math>1.6\pm0.4</math>), 4 hr (<math>1.4\pm0.4</math>) and 6 hr (<math>1.3\pm0.3</math>) post-injection (all <math>p&lt;0.001</math>).</li> </ol>
<p><a href="#">Dario et al.</a> (2002) Italy Pre-Post N=14</p>	<p><b>Population:</b> TBI=6, Anoxic ABI=8; Mean Age=38.8 yr; Gender: Male=10, Female=4; Mean Time Post Injury=36.7 mo.</p> <p><b>Intervention:</b> Patients received continuous intrathecal baclofen infusions through the implantation of a subcutaneous pump. Mean length of spasticity was 36.7 mo post injury.</p> <p><b>Outcome Measure:</b> Ashworth Scale (AS), Spasm Frequency Scale (SFS).</p>	<ol style="list-style-type: none"> <li>1. Between pre-operative through the last follow up, there was a significant decrease in AS scores in both lower (<math>4.3\pm0.5</math> versus <math>2.7\pm0.7</math>) and upper (<math>4.1\pm0.8</math> versus <math>2.3\pm0.9</math>) extremities (both <math>p&lt;0.05</math>).</li> <li>2. Significant reduction in SFS scores was found between preoperative and postoperative values (<math>2.5\pm0.5</math> versus <math>0.4\pm0.6</math>, <math>p&lt;0.001</math>).</li> <li>3. Mean daily dose of baclofen was 305 µg (range 90-510 µg).</li> </ol>
<p><a href="#">Francois</a> (2001) France Case Series N=4</p>	<p><b>Population:</b> TBI; Mean Age=19.5 yr; Gender: Male=1, Female=2, Unknown=1; Mean GCS=3.5.</p> <p><b>Intervention:</b> Patients received intrathecal baclofen infusions within 1 mo following injury onset.</p> <p><b>Outcome Measure:</b> Ashworth scores, Frequency and Intensity of Autonomic Disorders.</p>	<ol style="list-style-type: none"> <li>1. Reductions in spasticity, and lower limb Ashworth scores at 6 mo post intervention were reported in three of the four cases. In the last case, a substantial reduction in autonomic disorders and spasticity enabling passive physiotherapy was reported.</li> </ol>
<p><a href="#">Meythaler et al.</a> (1999) USA Pre-Post N=17</p>	<p><b>Population:</b> ABI; Mean Age=29 yr; Gender: Male=14, Female=3.</p> <p><b>Intervention:</b> Patients with spasticity and/or dystonia were surgically fitted with an infusion pump into the lower abdominal wall for continuous administration of intrathecal baclofen (100 µg/day). Patients were assessed at 1 yr.</p> <p><b>Outcome Measure:</b> Ashworth Rigidity Scale (ARS), Spasm Frequency Scale, Deep Tendon Reflex Score.</p>	<ol style="list-style-type: none"> <li>1. One year of intrathecal baclofen treatment (average dose: 302 µg/d) resulted in a decrease in ARS (mean 2.2 points), spasm frequency (mean 1.6 points), and reflex scores (mean 2.4 points) for the lower extremity (all <math>p&lt;0.0001</math>).</li> <li>2. For the upper extremity, the ARS, spasm frequency, and reflex scores decreased by a mean of 1.4, 1.0, and 1.2 points respectively (all <math>p&lt;0.0001</math>).</li> <li>3. No cognitive side effects were observed after 1 yr.</li> </ol>
<p><a href="#">Meythaler et al.</a> (1999) USA Pre-Post N=6</p>	<p><b>Population:</b> TBI=3, Stroke=3; Mean Age=50 yr; Gender: Male=2, Female=4.</p> <p><b>Intervention:</b> Patients were surgically fitted with a programmable infusion pump into the lower abdominal wall for continuous administration of baclofen using the same methodology as Meythaler et al. (1997).</p> <p><b>Outcome Measure:</b> Ashworth Rigidity Scale, Spasm Frequency Scale, Deep Tendon Reflex</p>	<ol style="list-style-type: none"> <li>1. Lower extremities showed a significant reduction in Ashworth scores (<math>p&lt;0.0001</math>), affected lower limb reflex score (<math>p=0.021</math>), normal side (<math>p=0.0051</math>), but not significant changes in affected lower limb spasm score (<math>p=0.500</math>).</li> <li>2. Upper extremities showed significant reductions in Ashworth scores on affected side (<math>p=0.0002</math>) but were not significant for Biceps Reflex score</li> </ol>

Author/Year/ Country/Study Design/PEDro/N	Methods	Outcome
	scores.	(affected and normal: $p=0.109$ and $p=0.068$ ), or spasm score (affected: $p=0.1797$ ). 3. No patients complained of subjective weakness on the normal side.
<a href="#">Meythaler et al.</a> (1997) USA Pre-Post N=12	<b>Population:</b> TBI=9, ABI=3; Mean Age=28 yr; Gender: Male=11, Female=1. <b>Intervention:</b> Patients received continuous intrathecal baclofen delivery for 3 mo via an implanted infusion pump-catheter system. <b>Outcome Measure:</b> Ashworth Rigidity Scale, Spasm Frequency Score, Deep Tendon Reflex Score.	1. For the lower extremity, Ashworth Scale Scores decreased by a mean of 1.4 points, spasm frequency by 1.5, and reflex scores by 2.5 (all $p<0.0001$ ). 2. For the upper extremity, the mean decrease in scores was 1.4 points for the Ashworth Scale ( $p=0.003$ ), 1.2 for spasm frequency ( $p=0.007$ ) and 1.0 for reflex ( $p=0.011$ ).
<a href="#">Becker et al.</a> (1997) Germany Case Series N=18	<b>Population:</b> TBI=9, Hypoxic Brain Injury=9; Mean Age=41yr; Gender: Male=13, Female=6; Mean Time Post Injury=11.6 mo. <b>Intervention:</b> Patients received continuous intrathecal baclofen infusion. <b>Outcome Measure:</b> Ashworth Scale, Spasm Frequency Scale.	1. In all patients spasticity was reduced. 2. Mean Ashworth scores reduced from 4.5 to 2.33, and the mean spasm frequency scores decreased from 2.16 to 0.94. 3. Reduction in spasticity led to a reduction in pain.

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

## Discussion

Meythaler et al. (1996) confirmed the effectiveness of intrathecal baclofen in decreasing upper and lower extremity spasticity in a randomized, double blinded, placebo controlled cross-over trial. In subsequent studies, the same investigators went on to demonstrate the effectiveness of intrathecal baclofen for decreasing spasticity for up to three months (Meythaler et al., 1997) and 1 year (J. M. Meythaler et al., 1999). Investigations carried out by other research groups have reported similar findings regarding the efficacy of intrathecal baclofen for the management of spasticity post ABI (Becker et al., 1997; Chow et al., 2015; Dario et al., 2002; Francisco et al., 2005; Hoarau et al., 2012b; Margetis et al., 2014; Posteraro et al., 2013; Stokic et al., 2005; Wang et al., 2016). However, a common limitation of these studies is the lack of a control group. Regardless, it appears that intrathecal baclofen is an effective treatment for spasticity. It should be noted, however, that some adverse effects, such as urinary hesitancy, were reported. Hoarau et al. (2012a) conducted a 10-year follow up of individuals with dysautonomia and hypertonia treated with intrathecal baclofen therapy. The study found that 62.8% of participants had some type of complication, with infections at the operative site being the most common (20.9%), followed by overdosed with profound flaccidity, sedation, and vomiting (16.3%) (Hoarau et al., 2012a).

Studies have also evaluated the functional consequences by assessing walking performance, gait speed, and range of motion following a bolus injection of intrathecal baclofen (Chow et al., 2015; Horn et al., 2010; Horn et al., 2005). Horn et al. (2005) found that although the injections produced changes in joint range of motion during gait, only ankles showed a significant result. Chow et al. (2015) similarly found an increase in ankle range of motion but found no significant differences in terms of gait speed, stride length, cadence, or stance. Future studies should be conducted using a prospective controlled trial or RCT study design that includes control groups to further establish the efficacy of intrathecal baclofen for the management of spasticity post ABI.



## Conclusions

*There is level 1b evidence that bolus intrathecal baclofen injections may produce short-term (up to six hours) reductions in upper and lower extremity spasticity compared to placebo following ABI.*

*There is level 4 evidence to suggest that prolonged intrathecal baclofen may result in longer-term (three months, and one year) reductions in spasticity in both the upper and lower extremities following an ABI.*

*There is conflicting level 4 evidence to suggest that intrathecal baclofen may result in short-term improvement of walking performance in ambulatory patients, particularly gait velocity, stride length, and step width, in individuals post ABI.*

**Bolus injections of intrathecal baclofen likely produce short-term reductions in upper and lower extremity spasticity and improvements in walking performance post ABI.**

**Prolonged intrathecal baclofen may reduce upper and lower extremity spasticity long-term post ABI.**

### 4.2.5.6 Casting

Spasticity frequently results in musculoskeletal contractures (Mayer et al., 1997) and has been estimated in one study to have an incidence as high as 84% in TBI patients (Yarkony & Sahgal, 1987). As with hand splinting, the theoretical premise for the effect of casting on hypertonia and joint mobility is based on different neurophysiological and biomechanical principles (Mortenson & Eng, 2003). Spasticity may be reduced by the effect of prolonged stretch, or possibly the effects of neutral warmth or prolonged pressure which may in turn reduce the cutaneous sensory input to the spinal cord. From a biomechanical perspective, muscle and connective tissues are likely elongated when immobilized in a stretched position, thus reducing the incidence on contractures (Mortenson & Eng, 2003) (Table 4.14).

Casting has been thought to reduce hypertonia and spasticity in individuals with an ABI. This is believed to be the result of reducing contractures by stretching the muscles of the immobilized limb (Pohl et al. 2002). Serial casting is a process in which the angle of the cast is changed periodically, with the objective of returning the joint to its original angle. However, despite the fact that serial casting has been utilized by physiotherapists for more than 40 years there is little empirical data to support its use in isolation. Conversely, evidence exists supporting the use of casting as a useful adjunct to other therapies for the management of spasticity and contracture in patients post TBI.

**Table 4.15 Casting Techniques for the Treatment of Spasticity Post ABI**

Author/Year/ Country/Study Design/PEDro Score/N	Methods	Outcome
<a href="#">Moseley et al.</a> (2008)	<b>Population:</b> TBI; <i>Positioning Group</i> (n=12): Gender: Male=11, Female=1; Mean Age=30.8 yr;	1. Stretching group received a mean of 13 hr of stretching during the intervention and the serial

Author/Year/ Country/Study Design/PEDro Score/N	Methods	Outcome
RCT PEDro=8 N=26	Median Time Post Injury=71 days; Median Glasgow Coma Scale (GCS) score=3. <i>Serial Casting Group (n=14)</i> : Gender: Male=12, Female=2; Mean Age=32.3 yr; Median Time Post Injury=59 days; Median GCS score=4.5. <b>Intervention:</b> Participants were randomized to one of two interventions for elbow flexion contracture: serial casting or passive stretch (positioning group). Those in the serial casting group had long arm synthetic casts applied for 2 wk with the elbow in a stretched position. Casts were changed to progress the stretch. After 2 wk, the cast was removed, and the participants underwent passive stretching 1 hr/wk for 4 wk. The second group had passive stretch applied to the elbow flexor muscles for 1 hr/day, 5 x/wk. <b>Outcome Measure:</b> Torque controlled passive elbow extension, Modified Tardieu Scale.	casting had stretch applied for a mean of 13.6 days. 2. Those in the serial casting group had a greater reduction in contracture in the short term: serial casting reduced contracture by a mean of 22° (p<0.001) when compared to the positioning group. The next day the mean reduction was only 11° for the casting group, and differences between groups were less (p=0.052). 3. At follow up assessment, there was no significant or clinically meaningful difference between groups (mean effect 2°, p=0.782). 4. When looking at spasticity the serial casting group had slightly lower spasticity than the stretching group (p<0.05).
<a href="#">Moseley</a> (1997) Australia RCT PEDro=4 N=9	<b>Population:</b> TBI; Mean Age=29.1 yr; Gender: Male=8; Female=1; Time Post Injury=72.2 days. <b>Intervention:</b> Subjects in the experimental group received a below-knee cast and stretched for 7 days. The control group did not receive a cast or stretching. Participants then received the other intervention. <b>Outcome Measure:</b> Passive ankle dorsiflexion (PAD) movement.	1. PAD movement increased (mean: 13.5°) during intervention compared to a decrease (mean: 1.9°) shown for the control condition (p<0.05). Mean difference between conditions was 15.4°.
<a href="#">Singer et al.</a> (2003) Australia Pre-Post N=9	<b>Population:</b> Stroke=3, Subarachnoid Hemorrhage=4, Intra-cerebral Hemorrhage=1, Diffuse Axonal Injury=1; Mean Age=30.7 yr; Gender: Male=6, Female=3; Mean Time Post Injury=3.9 mo. <b>Intervention:</b> A serial casting procedure addressing extensibility, passive resistance torque and stretch reflex response of the ankle was implemented. Casts were applied weekly, and continued until goal was reached or no measurable gain recorded. <b>Outcome Measure:</b> Maximal Passive Range of Motion, Transfer Dependency, and Rancho Los Amigos Levels of Cognitive Functioning (RLA).	1. Post-casting, all subjects had at least 10° dorsiflexion with the knee flexed and 6 had maximal passive range of motion of 10° dorsiflexion with knee extended. 2. Muscle extensibility and passive torque improved significantly (p<0.0001). Functional range was maintained in 8 subjects at 6mo follow-up. 3. All participants improved RLA scores by at least one point. 4. Significant improvements were noted for transfer dependency scores from initial to post intervention (p<0.0015). 2. Casting did lead to some tissue breakdown.
<a href="#">Pohl et al.</a> (2002) Germany Case Control N=105	<b>Population:</b> TBI=43, Stroke=19, Intracerebral Hemorrhage=19, Cerebral Hypoxia=11, Subarachnoid Hemorrhage=6, Other=7; Gender: Male=81, Female=24. <i>Control Group (n=56)</i> : Median Age=38.2 yr. <i>Intervention Group (n=49)</i> : Median Age=44.6 yr. <b>Intervention:</b> A stepwise reduction of fixed, flexed joint contracture via serial casting. Patients were treated with conventional casting	1. The median change interval was 6.9 days for 92 joints of 56 control group patients. 2. The median change interval was 2.7 days for 80 joints of 49 intervention group patients. 3. Mean casting time in the control and intervention group was 32.6±20.6 days and 9.3±5.6 days, respectively.

Author/Year/ Country/Study Design/PEDro Score/N	Methods	Outcome
	<p>changing intervals of 5-7 days (control) or 1-4 days (Intervention), to maximum possible extension (&lt;10% of extension deficit) or when extension deficit fails to reduce after two cast changes.</p> <p><b>Outcome Measure:</b> Maximum deficits of different joints (elbow, wrist, knee, ankles), Range of Motion (ROM), Number of Complications</p>	<p>4. ROM improved after casting and 1mo follow-up in both groups (<math>p&lt;0.001</math>) but no between group differences were found (<math>p=0.72</math>).</p> <p>5. Casting complications differed between groups 1 and 2 (29.3% versus 8.8%, <math>p=0.001</math>).</p>

### Discussion

In comparison to passive stretching, serial casting was beneficial in improving contracture of the elbow (Moseley et al. 2008). The improvement in contracture, however, was no longer significant after 4 weeks. In addition, the researchers noted a greater improvement in spasticity in the casting group when compared to the stretching group. The results from this study suggest that while serial casting may be effective initially, it does not appear to have long-term effects on contractures.

In order to evaluate the efficacy of lower extremity casting post ABI, Moseley (1997) used a randomized open cross-over design to compare one week of casting combined with stretching to a week of no therapy (control) for ankle plantar flexion contractures. The experimental group had a significantly improved range of passive ankle dorsiflexion whereas the control group tended to have overall deterioration of ankle range of motion (Moseley, 1997). In two separate studies, Singer et al. (2003) and Singer et al. (2003) also evaluated the efficacy of weekly casting and found casting to be effective in improving ankle movement. In addition, greater ankle mobility was shown to be associated with improved transfer independence (Singer et al., 2003). It should be noted, however, that casting can lead to tissue breakdown (Singer et al. 2003).

In a retrospective case comparison study, Pohl et al. (2002) compared short, one to four days casting to a longer duration, five to seven days casting, for both upper and lower extremity joints. Although improvements in range of motion were seen in each group immediately following the intervention and at a one-month follow-up, there was no significant difference found between groups. However, the discontinuation rate in the longer duration group due to complications was significantly higher than for the short casting interval group.

### Conclusions

***There is level 1b evidence that serial casting may improve contractures of the elbow initially, but not long-term, when compared to passive stretching in individuals with an ABI.***

***There is level 1b evidence that serial casting may be superior to passive stretching at improving spasticity of the elbow in individuals post ABI.***

***There is level 2 evidence that a below-knee casting and stretching protocol may increase passive ankle dorsiflexion in patients post ABI.***

*There is level 4 evidence that weekly below-knee casts may improve ankle range of motion, muscle extensibility, and passive torque in patients post ABI.*

*There is level 3 evidence that short duration (one to four days) and longer duration (five to seven days) serial casting may have similar effects on upper or lower extremity range of motion in individuals post ABI.*

Serial casting likely improves contractures and spasticity in individuals with an ABI compared to stretching; however, contracture improvement may not be maintained long-term.

Below-knee casting and stretching might increase passive ankle dorsiflexion in patients post ABI.

Serial below-knee casting may improve ankle range of motion and muscle extensibility in patients post TBI; however, this intervention may be associated with tissue breakdown.

Serial casting, whether for a short or long duration, might improve range of motion in individuals with an ABI. However, short duration casting may have a lower complication rate than long duration.

#### **4.2.5.7 Adjustable Orthosis**

Similar to casting, an adjustable pre-fabricated orthosis could potentially provide prolonged stretching of an ankle plantar flexion contracture. Advantages of the orthosis over a rigid cast include the ease of adjustability and the ability to remove it daily for short periods of time. A pre-post study by Grissom and Blanton (2001) examined six participants with mixed etiologies who received a 2% lidocaine block of the posterior tibial nerve and then wore an adjustable ankle-foot orthosis on the affected ankle for 23 hours per day for two weeks for plantarflexion contractures. Adjustments were attempted every two to three days to increase passive dorsiflexion range of motion. The group reported a significant mean gain in ankle dorsiflexion of 20.1° ( $p=0.0078$ ). Of concern, there was a relatively high complication rate of skin breakdown and pain that occurred with splinting (44%). Further, the only individual with a TBI dropped out as the orthosis was thought to agitate the individual (Grissom & Blanton, 2001). As a result, more research is needed with an ABI population before conclusions on adjustable orthoses can be made.

#### **4.2.5.8 Hand Splinting and Stretching**

Hand splinting and stretching is another way that patients may experience relief from spasticity and contracture post-ABI. The study below examines the effectiveness of combination splinting and stretching for spasticity relief (Table 14.16).

**Table 4.16 Hand Splinting and Stretching for the Treatment of Spasticity Post ABI**

Author/Year/ Country/Study Design/ PEDro Score/ N	Methods	Outcomes
<a href="#">Thibaut et al.</a> (2015) Belgium RCT PEDro=4 N=17	<p><b>Population:</b> TBI=7, Anoxia=5, Aneurysm=5; Mean Age=41 yr; Gender: Male=9, Female=8; Mean Time Post Injury=35 mo; Severity: Severe.</p> <p><b>Intervention:</b> Participants were randomized to receive one of the following exercise protocols on each of their upper limbs: manual stretching and control (no Intervention) (G1, n=8), soft splinting and control (G2, n=12), or soft splinting and manual stretching (G3, n=14). Each exercise was done for 30 min followed by a 60 min break. Outcomes were assessed before (T1) and after (T2) each protocol, and after each break (T3).</p> <p><b>Outcome Measures:</b> Modified Ashworth Scale (MAS), Modified Tardieu Scale (MTS), Range of Motion (ROM), and Hand Opening (HO).</p>	<ol style="list-style-type: none"> <li>1. In G1, there were no significant changes in MAS, MTS, ROM, or HO after stretching or after the control protocol.</li> <li>2. In G2, the mean MAS score of finger flexor muscles improved significantly after splinting from T1 to T2 (<math>p=0.014</math>) and the improvement was maintained at T3 (<math>p=0.022</math>). There was no significant change for the control.</li> <li>3. In G3, the mean MAS score of finger flexor muscles improved significantly after both splinting (<math>p=0.014</math>) and stretching (<math>p=0.022</math>) from T1 to T2, but neither improvement was maintained at T3.</li> <li>4. In G2, the mean HO score improved significantly after splinting from T1 to T2 (<math>p=0.009</math>), but the improvement was not maintained at T3. There was no significant change for the control.</li> <li>5. In G3, the mean HO score improved significantly after splinting (<math>p=0.005</math>) from T1 to T2, but the improvement was not maintained at T3. There was no significant change in mean HO score after stretching (<math>p=0.249</math>).</li> <li>6. In G3 and G2, there were no significant changes in MTS or ROM after the interventions.</li> </ol>

## Discussion

A randomized controlled trial (RCT) compared manual stretching, soft hand splinting, and manual stretching plus soft hand splinting to determine the optimal intervention (Thibaut et al., 2015). Results suggested that soft hand splinting for 30 minutes resulted in improved hand opening and reduced spasticity of the flexor finger muscles, however improvements in hand opening were not maintained after the break period. The hand splint was said to be feasible to use in daily care, as the splint was comfortable and easy to apply. There is a need to further research the effect of splinting in individuals with ABI as this practice is used in both acute and rehabilitation settings.

## Conclusions

*There is level 1b evidence that nocturnal hand splinting may not improve upper extremity range of motion or function compared to standard care in individuals post ABI.*

**Hand splinting combined with stretching may be an effective treatment for spasticity and range of motion.**

#### 4.2.5.9 Multimodal Interventions

Multimodal interventions can consist of combining two or more interventions or comparing different interventions to each other. The following studies use a multimodal approach to determining effective interventions for the treatment of spasticity post ABI.

**Table 4.17 Multimodal Interventions for the Treatment of Spasticity Post ABI.**

Author/Year/ Country/ Study design/ PEDro Score	Methods	Outcome
<a href="#">Leung et al.</a> (2014) Australia RCT PEDro=8 N <sub>Initial</sub> =35, N <sub>Final</sub> =32	<p><b>Population:</b> TBI; <i>Experimental Group (EG; n=17)</i>: Mean Age=38 yr; Gender: Male=14, Female=3; Mean Time Post Injury=140 days; Mean GCS=5. <i>Control Group (CG; n=18)</i>: Mean Age=38 yr; Gender: Male=15, Female=3; Mean Time Post Injury=83 days; Mean GCS=5.</p> <p><b>Intervention:</b> Participants were randomly allocated to either the EG or CG group. The EG group underwent a treatment of tilt table standing and electrical stimulation (30 min 5 days/wk) and splinting (12 hr 5days/wk) for a total of 6 wk. For the next 4 wk EG group participants underwent tilt table standing alone (30 min 3 days/wk). The CG group underwent tilt table standing (30 min 3 days/wk) for the full 10 wk. Measures were taken at baseline, 6 wk and 10 wk.</p> <p><b>Outcome Measure:</b> Passive ankle dorsiflexion, Functional Independence Measure (FIM).</p>	<ol style="list-style-type: none"> <li>1. The CG group had a greater range of motion for passive ankle dorsiflexion than the EG group at 6 wk (3 degrees) and 10 wk (-1 degree).</li> <li>2. The EG group had a greater mean reduction in spasticity (1 point) at 6 wk; however, the effect disappeared at 10 wk.</li> <li>3. There was no between group differences in walking speed.</li> <li>4. There were no differences between groups for tolerance to treatment, perceived treatment benefit, perceived treatment worth, and willingness to continue with treatment.</li> </ol>
<a href="#">Lorentzen et al.</a> (2012) Denmark RCT-Crossover PEDro=6 N=10	<p><b>Population:</b> TBI=6, Stroke=2, Subarachnoid Hemorrhage=1, Post-Operative Hemorrhage=1; Mean Age=31.5 yr; Gender: Male=6, Female=4; Mean Time Post Injury=3.6 mo.</p> <p><b>Intervention:</b> Participants received either Neural Tension Technique (NTT) intervention or the random passive movement (RPM) treatment on knee joints. The NTT and RPM treatments lasted for 20 min, with clinical tests conducted immediately before and after each intervention.</p> <p><b>Outcome measure:</b> Modified Ashworth Scale (MAS), Range of Motion (ROM).</p>	<ol style="list-style-type: none"> <li>1. The blinded reviewers found no significant change on the MAS for knee flexors after the NTT (Mean change=0.4–0.6, p=0.10–0.31) or the RPM (Mean change=0.4–0.5, p=0.1–0.3). No significant between group differences were found (p=0.12–0.71).</li> <li>2. No significant between or within group differences were found based on the MAS for knee extensors after the intervention.</li> <li>3. The blinded reviewers found no significant difference in ROM after RPM (p=0.13) but did for NTT (p&lt;0.05). No significant between group differences for ROM were found (p&gt;0.32).</li> </ol>
<a href="#">Verplancke et al.</a> (2005) UK RCT PEDro=4 N=35	<p><b>Population:</b> TBI=20, Neurosurgery=11, Anoxia=4; Gender: Male=25, Female=10. <i>Group 1 (n=11)</i>: Median Age=40 yr; Mean Time Post Injury=9.3 days, Mean Glasgow Coma Scale (GCS) score 4.3. <i>Group 2 (n=12)</i>: Median Age=33.5 yr; Mean Time Post Injury=13.25 days; Mean GCS score=4.7.</p>	<ol style="list-style-type: none"> <li>1. Eighty-eight percent of patients developed spasticity within 14 days of injury.</li> <li>2. Mean change in angle of passive ankle dorsiflexion was 4.59° in controls, 11.69° in group 2 and 13.59° in group 3.</li> </ol>

Author/Year/ Country/ Study design/ PEDro Score	Methods	Outcome
	<p>Group 3 (n=12): Median Age=41.5 yr; Mean Time Post Injury=10.6 days; Mean GCS score=5.2.</p> <p><b>Intervention:</b> Participants entered one of three groups: group 1 received a physical intervention (controls), group 2 received casting plus injections of saline (4 ml), and group 3 received casting with botulinum toxin (100 units per leg) into the gastrocnemius and soleus muscles. Patients were re-cast if a 10° change in dorsiflexion occurred.</p> <p><b>Outcome Measure:</b> Calf contracture, Modified Ashworth Scale (MAS), Passive Range of Motion.</p>	<p>3. There were significant improvements in MAS scores in treated groups (group 2, <math>p&lt;0.03</math>; group 3, <math>p=0.04</math>) but not controls (<math>p&gt;0.05</math>).</p>

For the lower extremity, Verplancke et al. (2005) found that active prophylaxis of leg spasticity using casting is beneficial; however, there was no difference in outcomes between those casted with or without Botulinum toxin. This indicates that BTX may not be beneficial when paired with casting (Verplancke et al., 2005). Future studies, with a larger sample size, are needed to examine this further.

Electrical stimulation was then studied as a multimodal intervention, combined with standing on a tilt table, and splinting for ankle contractures (Leung et al., 2014). This RCT found improvements in passive ankle dorsiflexion that favoured the control group; however, neither group reached values of clinical significance. Leung et al. (2014) did find a significant reduction in spasticity favouring the intervention group at week 6 but it no longer existed by week 10. Of note, 10 participants had issues with adhering to the tilt table procedure due to fainting, fatigue, or behavioural issues. In addition, due to the fact that the experimental group received a combination of 3 treatments (tilt table, electrical stimulation, and casting) while the control group only underwent tilt table treatment, it is unclear which intervention was responsible for the short-term reduction in spasticity in the experimental group.

In a RCT by Lorentzen et al. (2012), participants received either neural tension technique (NTT) treatment or random passive movement (RPM) therapy on knee joints. No significant changes in spasticity were observed between groups in the knee flexor or extensor muscles. Furthermore, range of motion may be improved to the same effect by NTT and RPM therapies Hirose et al. (2013).

## Conclusions

***There is level 1b evidence that electrical stimulation in combination with tilt table standing and splinting may decrease spasticity at 6 weeks post intervention compared to tilt table standing alone in patients with an ABI.***

***There is level 2 evidence that botulinum toxin combined with casting may not be more effective than botulinum toxin injections alone in improving leg spasticity in individuals with an ABI.***



*There is level 1b evidence that neural tension technique may not be more effective than random passive movement in improving lower extremity spasticity and range of motion in individuals with an ABI.*

**Botulinum toxin injections in combination with casting may be as effective as casting alone at reducing leg spasticity in patients post ABI.**

**Electrical stimulation in combination with tilt table standing and splinting may acutely improve spasticity (6 weeks) in patients post ABI.**

**Neural tension technique may be just as effective as random passive movement for improving lower extremity spasticity post ABI.**

### 4.3 Visual Dysfunction

Dysfunctions of the visual system are quite common following TBI (Morton, 2004). The overall incidence of cranial nerve injury in individuals hospitalized following TBI has been reported to be 19% (Bontke et al., 1993). It is a relatively new concept that the visual system can respond to treatments directed towards visual-perceptual and/or visual motor skills in individuals with acquired neurological damage. The visual system is highly integrated with many functions other than sight, as it also acts as a primary sensory receptor for motor, social, cognitive, communicative, and emotive tasks. Improvements in visual-perceptual and visual-motor disorders can increase function in all the aforementioned areas and can enhance maximal functional recovery. Consequently, it is necessary to direct a fair amount of attention to visual system disorders in individuals with TBI and this aspect should be considered an essential part of any rehabilitation program (Morton, 2004). In a review conducted by Riggs et al. (2007), the authors noted that visual rehabilitation studies have primarily involved stroke patients and have largely neglected the TBI population. Their review indicates that visual neglect disorders resulting from a stroke and brain injury show improvement after treatment with prisms, visuomotor feedback training, and patching interventions. Moreover, a recent review by Berger et al. (2016) examined specific interventions for improving occupational performance in adults with visual impairments as a result of TBI. Results indicate that there is limited evidence on the effectiveness of vision therapy for oculomotor dysfunction, however there is sufficient evidence to support vision therapy as a method to improve visual field deficits in patients with TBI (Berger et al., 2016) (Table 4.17).

**Table 4.18 Interventions for the Treatment of Visual Dysfunction Post ABI**

Author/Year/ Country/ Study design/ PEDro Score	Methods	Outcome
<a href="#">Kasten et al.</a> (2000) Germany RCT PEDro=5 N=32	<b>Population:</b> Vascular Disease=9, ABI=23; Mean Age=51.1 yr; Gender: Male=20, Female=12; Mean Time Post Injury=6.8 yr. <b>Intervention:</b> Participants were randomly assigned to either the Control Group (foveal fixation training only - FixTrain) or Restitution Group (PC-based training	1. The restitution group showed an increase in PeriMa and TAP-2000 after training ( $p<0.01$ and $p<0.04$ , respectively). 2. The restitution group had non-significant improvements in PeriForm and PeriColor ( $p=0.06$ and $p=0.12$ , respectively) within the defective area of the visual field.



Author/Year/ Country/ Study design/ PEDro Score	Methods	Outcome
	<p>program – Visure, SeeTrain). Both groups trained for 1 hr/day at home for <math>\geq 150</math> hr over a 6 mo period.</p> <p><b>Outcome Measure:</b> High-Resolution Campimetry (PeriMa), Conventional Perimetry (TAP-2000), Pattern Recognition (PeriForm), Colour Discrimination (PeriColor).</p>	<ol style="list-style-type: none"> <li>There was a correlation between PeriMa and PeriForm (<math>r=0.67</math>, <math>p&lt;0.05</math>) and PeriForm and PeriColor (<math>r=0.37</math>, <math>p&lt;0.05</math>) for improved color and form perception.</li> <li>The PeriMa, PeriForm, and PeriColor all demonstrated a shift of the visual field border in the direction of the blind area for subjects in the restitution group.</li> </ol>
<a href="#">Kasten et al. (1998)</a> Germany RCT PEDro=7 N=38	<p><b>Population:</b> Stroke=10, ABI=28; Mean Age=51.5 yr; Gender: Male=24, Female=14; Mean Time Post Injury=7.0 mo.</p> <p><b>Intervention:</b> Participants were randomly assigned to either the Restitution Group (visual restitution training (VRT)) or the Control Group (fixation training program which required eye movement toward stimuli within the foveal region). Both groups completed 150 hr of training over 6 mo at home in a darkened room.</p> <p><b>Outcome Measure:</b> High-Resolution Perimetry (HRP), Response Frequency, Area of Absolute Defect, Tübinger Automatic Perimeter 2000 (TAP).</p>	<ol style="list-style-type: none"> <li>Performance on HRP showed improved ability to perceive visual stimuli above detection threshold in the VRT group post-training (post-chiasmic: <math>p&lt;0.05</math>, optic nerve: <math>p&lt;0.01</math>).</li> <li>The VRT group demonstrated a higher response frequency to stimuli than the control group (<math>p&lt;0.05</math>).</li> <li>TAP scores showed a decrease in the area of absolute defect for subjects in the VRT group with optic nerve injuries (<math>p&lt;0.01</math>).</li> <li>Subjects with optic nerve damage benefitted most from VRT; 72.2% of subjects who received VRT reported subjective improvement while only 16.6% of the control subjects did so (<math>p&lt;0.03</math>).</li> </ol>
<a href="#">Conrad et al. (2016)</a> USA Pre-Post N <sub>Initial</sub> =19, N <sub>Final</sub> =13	<p><b>Population:</b> TBI=15, Stroke=3, Organic Brain Syndrome=1; Mean Age=45.2 yr; Gender: Male=12, Female=7; Time Post Injury=2.2 yr.</p> <p><b>Intervention:</b> Participants were prescribed home-based computer vergence therapy using software provided (5 days/wk for 12 wk). Participants were assessed at baseline, 4, 8 and 12 wk.</p> <p><b>Outcome Measure:</b> Negative Fusional Vergence, Positive Fusional Vergence, Near Point of Convergence, Vergence Facility, Convergence Insufficiency Symptom Survey (CISS).</p>	<ol style="list-style-type: none"> <li>Negative fusional vergence showed significant improvement over 12 wk in blur (<math>p=0.037</math>), break (<math>p=0.003</math>) and recovery (<math>p=0.006</math>).</li> <li>Positive fusional vergence showed significant improvement over 12 wk in blur, break and recovery (<math>p&lt;0.0001</math>).</li> <li>Near point of convergence showed significant improvement over 12 wk in break (<math>p=0.002</math>) and recovery (<math>p&lt;0.001</math>).</li> <li>Vergence facility showed a significant improvement from baseline to 12 wk (<math>p&lt;0.0001</math>).</li> <li>CISS scores improved significantly from baseline to 12wk (<math>p=0.0001</math>).</li> </ol>
<a href="#">Doble et al. (2010)</a> USA Pre-Post N=43	<p><b>Population:</b> TBI; Mean Age=44 yr; Gender: Male=12, Female=31; Mean Time Post Injury=3.6 yr.</p> <p><b>Intervention:</b> Patients were given individualized prismatic spectacle lenses.</p> <p><b>Outcome Measure:</b> Vertical Heterophoria Symptom Questionnaire (VHS-Q).</p>	<ol style="list-style-type: none"> <li>The mean VHS-Q score at baseline was <math>34.8 \pm 16.1</math> (scale ranges 0-75 points).</li> <li>The mean difference in VHS-Q scores pre to post intervention was <math>16.7 \pm 12.8</math> (<math>p&lt;0.01</math>).</li> </ol>
<a href="#">Ciuffreda et al. (2006)</a> USA PCT N=14	<p><b>Population:</b> TBI=9, Stroke=5; Mean Age=48.4 yr; Gender: Male=9, Female=5; Mean Time Post Injury=2.4 yr.</p> <p><b>Intervention:</b> Patients with oculomotor-based dysfunction received reading-related</p>	<ol style="list-style-type: none"> <li>Significant improvements were found for each of the five questions on the reading rating scale (<math>p&lt;0.01</math>).</li> <li>Simulated reading saccade ratio showed significant improvements for ML (TI: <math>p&lt;0.05</math>)</li> </ol>

Author/Year/ Country/ Study design/ PEDro Score	Methods	Outcome
	<p>rehabilitation. Participants were assigned to either Visual (V) Feedback Training (modes included normal internal oculomotor visual feedback in isolation - T1 for 4 weeks) or combined Visual and Auditory (V+A) Feedback (concurrent with external oculomotor auditory feedback - T2 for 4wk) with a cross-over design. Participants underwent single-line (SL) and multiple-line (ML) simulated reading, and basic versional tracking (fixation, saccade, and pursuit) 2 x/wk for an 8 wk period.</p> <p><b>Outcome Measure:</b> Simulated Reading, Visagraph, Basic Versional Eye Movements, Reading Rating Scale.</p>	<p>and SL (T1: <math>p&lt;0.01</math>; T2: <math>p&lt;0.01</math>) training compared to pre-training levels</p> <ol style="list-style-type: none"> <li>3. The TBI subgroup had more improvements in the simulated reading and Visagraph.</li> <li>4. There was a trend (<math>0.05&lt;p&lt;0.10</math>) for greater reading improvement in V+A Feedback training.</li> </ol>
<p><a href="#">Padula et al. (1994)</a> USA Pre-Post N=20</p>	<p><b>Population:</b> TBI=10, Healthy Control=10; Age Range=22-46 yr; Gender: Male=8, Female=12.</p> <p><b>Intervention:</b> Visual evoked potentials (VEP) were performed using Nicolet Compact Four Electrodiagnostic System and a Visual Stimulator over three trials. During the baseline trial, subjects were tested without bi-nasal occluders and base-in prisms. In the experimental trial, subjects were tested with bi-nasal occluders and two diopters of base-in prisms. In the last phase, the bi-nasal occluders and prisms were removed and the subjects were re-evaluated.</p> <p><b>Outcome Measure:</b> Visual Evoked Potential (VEP).</p>	<ol style="list-style-type: none"> <li>1. The use of base-in prisms and bi-nasal occluders produced a large increase in VEP amplitude in individuals with TBI (<math>p&lt;0.01</math>).</li> <li>2. Using base-in prisms and bi-nasal occluders resulted in a significantly larger increase in VEP amplitude in individuals with TBI compared with the healthy controls (mean difference between groups 1.78, <math>p&lt;0.01</math>).</li> </ol>

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

## Discussion

A small pre-post study reported that visual dysfunction post ABI can be corrected with base-in prisms, as they affect the ambient visual process by increasing the effectiveness of binocular cortical cells (Padula et al., 1994). Base-in prisms and bi-nasal occluders incorporated within the lenses of both eyes increase the amplitude of visual-evoked potentials (Padula et al., 1994). A different group suggested that prismatic spectacle lenses are also effective in reducing symptoms related to vertical heterophoria and concussion, as they reduce the faulty vertical alignment signal generated by the brain injury (Doble et al., 2010).

Kasten et al. (1998) found that individuals with optic nerve or post-chiasmic injury associated with ABI who complete computer-based Visual restitution training (VRT) experience visual field enlargement and increased light detection. Furthermore, detection training has shown improvements in visual detection, as well as improvements in other visual functions such as shape and color recognition (Kasten et al., 2000). Recently, Conrad et al. (2016) studied a home-based computer vergence therapy program used to improve binocular visual dysfunction after ABI. Participants underwent home-based visual vergence therapy five days a week for 12 weeks. Negative vergence, positive vergence, near point convergence

and vergence facility all showed significant improvements over the 12 week intervention period (Conrad et al., 2016).

When the reading dysfunction post ABI is a result of sensory-based hemifield deficits or neuromotor deficits, saccadic oculomotor rehabilitation can lead to improvements in eye movements which are required for accurate reading (Ciuffreda et al., 2006). Repetitive oculomotor conditioning reduces the cognitive and attentional load of reading and results in a structural and systematic approach to reading. The benefits of oculomotor rehabilitation were observed in other activities of daily living such as concentration and visual scanning. Most importantly, reducing visual deficits in patients post TBI may facilitate their involvement in other therapies and contribute to overall recovery (Ciuffreda et al., 2006).

### **Conclusions**

***There is level 1b evidence that computer-based restitution training may be effective in improving the vision of those who sustain a TBI compared to visual fixation training.***

***There is level 2 evidence that saccade visual tracking compared to fixation and pursuit tracking may improve single-line and multi-line reading post ABI.***

***There is level 4 evidence showing that base-in prisms and bi-nasal occluders can be effective in treating ambient vision disturbances resulting from an ABI.***

***There is level 4 evidence that prismatic spectacle lenses may be effective in reducing symptom burden in patients with vertical heterophoria and post-concussive symptoms post injury.***

***There is level 4 evidence that rehabilitation programs directed at improving visual function can improve functional outcomes such as reading in patients post ABI.***

**Computer based restitution training and rehabilitation programs directed at improving visual function likely improve the vision of those who sustain a TBI.**

**Base-in prisms and bi-nasal occluders may be effective in treating ambient vision disturbances.**

**Saccadic oculomotor rehabilitation may improve eye movements and reading in patients post ABI.**

### **4.4 Vestibular Dysfunction**

Vestibular dysfunction is commonly overlooked when diagnosing an individual with TBI. Vertigo, balance problems, visual complaints (double vision, blurriness), and nausea are possible symptoms of vestibular injury. The most common persisting vestibular symptom after TBI is positional vertigo, or vertigo caused by head movement. Vertigo is caused by dysfunction of the vestibular nerve or the labyrinth (Shepard & Telian, 1995) and the inability of the central nervous system to effectively compensate for the dysfunction (Gurr & Moffat, 2001). Provoked vertigo manifests as either unilateral peripheral hypofunction, bilateral peripheral hypofunction, or benign paroxysmal positional vertigo (BPPV) (Godbout, 1997).

Although it is common for spontaneous resolution of vertigo to occur within 6 months of onset, recovery in the TBI population is constricted due to the frequent combination of central and peripheral vestibular structure injury. Vestibular rehabilitation following TBI is therefore needed to promote vestibular adaptation and recovery. Techniques which are typically used in vestibular rehabilitation are gaze stability exercises, vestibulo-ocular reflex gain adaptation, substitution exercises, habituation techniques, and static and dynamic balance and gait exercises (Scherer & Schubert, 2009). The optimal recovery of vestibular dysfunction is thought to be based on selecting the appropriate vestibular exercises for a specific individual and progressing gradually through the assigned exercises while increasing difficulty and intensity (Wee, 2002). Current literature includes a variety of interventions for vestibular rehabilitation (Table 4.18).

**Table 4.19 Interventions for the Treatment of Vestibular Dysfunction Post ABI**

Author/Year/ Country/Study Design/PEDro Score/N	Methods	Outcome
<a href="#">Naguib &amp; Madian</a> (2014) Egypt RCT PEDro=5 N=60	<p><b>Population:</b> TBI; Mean Age=30 yr; <i>Group 1 (n=20):</i> Gender: Male=14, Female=6; Severity: Mild=8, Moderate=7, Severe=5. <i>Group 2 (n=20):</i> Gender: Male=14, Female=6; Severity: Mild=8, Moderate=8, Severe=4. <i>Group 3 (n=20):</i> Gender: Male=15, Female=5; Severity: Mild=6, Moderate=8, Severe=6.</p> <p><b>Intervention:</b> Participants were randomized to receive betahistine dihydrochloride (48 mg/d, Group 1), a vestibular rehabilitation program (Group 2), or both (Group 3) as treatment for a balance disorder. Outcomes were assessed via videonystagmography at baseline, 1 and 2 wk, and then every month until recovery.</p> <p><b>Outcome Measures:</b> Recovery time.</p>	<ol style="list-style-type: none"> <li>Group 3 showed the earliest recovery time: complete recovery within 2 months.</li> <li>For Group 2, 80% had complete recovery within 2 months and 20% within 3 months.</li> <li>For Group 1, 85% had complete recovery within 2-3 months, and 15% in more than 3 months.</li> <li>Mean recovery time was significantly longer in Group 1 (62.1d) than in Group 2 (37.6d) and Group 3 (34.4d; <math>p&lt;0.050</math>), but there was no significant difference between Group 2 and Group 3 (<math>p&gt;0.05</math>).</li> </ol>
<a href="#">Peirone et al.</a> (2014) Italy RCT PEDro=6 N=16	<p><b>Population:</b> TBI=7, Stroke=7, Other=2; Mean Age=40.5 yr; Gender: Male=9, Female=7; Mean Time Post Injury=14.3 mo.</p> <p><b>Intervention:</b> Participants were randomized into a control (n=8) or intervention group (n=8). Both groups received standard physiotherapy in 50min sessions (3 x/wk for 7 wk). The intervention group also performed an individualized dual-task home-based programme (6 days/wk for 7 wk).</p> <p><b>Outcome Measure:</b> Balance Evaluation System Test (BEST), Activities-Specific Balance Confidence Scale, Goal Attainment Scaling (GAS).</p>	<ol style="list-style-type: none"> <li>Post-intervention scores differed significantly between groups on the BEST, with the intervention group improving more (<math>p=0.008</math>).</li> <li>There were no significant between group differences on the Activities-specific Balance Confidence Scale (<math>p=0.110</math>), or the GAS (<math>p=0.093</math>).</li> <li>The control group made significant improvements on the BEST (mean change=<math>5.5\pm3.53</math>, <math>p=0.020</math>) and the GAS (mean change=<math>16.28\pm6.58</math>, <math>p=0.010</math>).</li> <li>The intervention group made significant improvements from pre to post intervention on the BEST (mean change=<math>17.87\pm6.05</math>, <math>p=0.014</math>), the Activities-Specific Balance Confidence scale (mean change=<math>25.25\pm25.51</math>, <math>p=0.01</math>) and the GAS (mean change=<math>19.37\pm9.03</math>, <math>p=0.02</math>).</li> </ol>

Author/Year/ Country/Study Design/PEDro Score/N	Methods	Outcome
<a href="#">Motin et al.</a> (2005) Israel Post-Test N=10	<p><b>Population:</b> Severe TBI; Mean Age=43 yr; Gender: Male=8, Female=2; Mean Time Post Injury=67 d.</p> <p><b>Intervention:</b> Patients underwent a particle reposition maneuver. The examiner performed the Dix-Hallpike test to the affected side such that nystagmus and vertigo were elicited; this position was maintained for 1-2 min. The patient's head was then rotated 90° to the opposite side and held for ~ 30 sec. The subject was then asked to turn their head another 90° to the unaffected side. This position was maintained for another 1-2 min and then the subject was assisted to sit-up.</p> <p><b>Outcome Measure:</b> Improvements in Positional Nystagmus.</p>	<ol style="list-style-type: none"> <li>1. Six of 10 subjects had resolved positional nystagmus and vertigo following a single particle repositioning maneuver.</li> <li>2. Nine of 14 (64%) affected ears had resolved positional nystagmus and vertigo following a single particle repositioning maneuver.</li> <li>3. The other four subjects needed between 3 and 6 repeated treatments until their symptoms were completely resolved.</li> </ol>
<a href="#">Dault and Duga</a> (2002) Canada PCT N=8	<p><b>Population:</b> TBI=8; Mean Age=29.6 yr; Gender: Male=6, Female=2; Mean Time Post Injury=44.4 mo.</p> <p><b>Intervention:</b> Participants completed an individualized 12 wk specific training program (STP) combining aerobic dance, and slide and step training for 30 min, 3 x/wk compared to traditional muscular training (TMT) for 60 min, 2 x/wk for 12 wk.</p> <p><b>Outcome Measure:</b> Clinical Test for Sensory Interaction in Balance (CTSIB), Jumping Jack movement.</p>	<ol style="list-style-type: none"> <li>1. Over time, all of the participants' performance of the exercises improved.</li> <li>2. The analysis of balance revealed a significant difference between pre- and post-training sway area for the STP group (<math>p&lt;0.05</math>) but not for the TMT group.</li> </ol>
<a href="#">Gurr and Moffat</a> (2001) UK Pre-Post N=41	<p><b>Population:</b> TBI; Mean Age=44.1 yr; Gender: Male=28, Female=41; Mean Time Post Injury=78.7 mo.</p> <p><b>Intervention:</b> Therapy consisted of a behavioral exposure program to movements and activities that provoked vertigo and anxiety in order to assist compensation of vestibular dysfunction and habituation to physical anxiety symptoms.</p> <p><b>Outcome Measure:</b> Vertigo Symptom Scale (VSS), Vertigo Rating scale (VRS), Vertigo Handicap Questionnaire (VHQ), Sway-Monitor Assessment.</p>	<ol style="list-style-type: none"> <li>1. At the end of therapy, participants' vertigo symptoms and somatic anxiety (VSS) had significantly decreased from pre-test to post-test (both <math>p&lt;0.01</math>).</li> <li>2. Significant reductions in VRS scores were shown from pre-test to post-test, and post-test to follow up (both <math>p&lt;0.01</math>).</li> <li>3. Patients were able to perform exercises significantly faster (<math>p&lt;0.01</math>) and with significant lower rating of dizziness (<math>p&lt;0.01</math>) after the intervention.</li> <li>4. Post-test levels of postural sway on the sway monitor (ability to balance on an unstable surface with eyes open) had significantly improved compared to pre-test levels (<math>p=0.008</math>).</li> <li>5. Vertigo handicap levels (VHQ scores) significantly decreased from pre to post intervention (<math>p&lt;0.01</math>).</li> </ol>

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

## Discussion

Patients with TBI suffering from BPPV should be specifically treated with repositioning maneuvers until complete resolution (Motin et al., 2005). Vestibular rehabilitation, alone or in combination with pharmacological treatment (i.e., betahistine dihydrochloride), as a treatment for balance disorders post TBI has been shown to significantly reduce recovery time when compared to pharmacological management alone (Naguib & Madian, 2014).

In a small sample of adults, aerobic dancing and slide-and-step training improved balance and coordination in patients many years following TBI, suggesting that long-term improvement of vestibular dysfunction is possible with the appropriate program (Dault & Dugas, 2002). Further, Gurr and Moffat (2001) added a cognitive aspect to vestibular rehabilitation. The authors attempted to restructure the maladaptive thoughts and belief patterns associated with the symptoms of provoked vertigo. This multidimensional psychological approach was effective in improving vertigo symptoms, independence, emotional distress, physical flexibility and postural stability (Gurr & Moffat, 2001).

In terms of more familiar therapy interventions for balance, one study compared standard physiotherapy and standard therapy in addition to a home-based rehabilitation program (Peirone et al., 2014). Both groups showed significant improvements on the Goal Attainment Scaling and the Balance Evaluation System Test. However, when comparing these interventions, those receiving home-based rehabilitation made significantly greater improvements on the Balance Evaluation System Test (Peirone et al., 2014). Despite these findings, this study was underpowered and further investigation is needed before definitive conclusions are made.

### **Conclusions**

***There is level 4 evidence that vestibular rehabilitation programs, such as a behavioural exposure program, may improve symptoms of vertigo in patients after TBI.***

***There is level 2 evidence that vestibular rehabilitation programs, alone or in combination with betahistine dihydrochloride, can improve recovery time for balance disorders in individuals with an ABI compared to betahistine dihydrochloride alone.***

***There is level 2 evidence to that using a combined aerobic dancing and slide and step training program may reduce balance and coordination deficits post TBI.***

**Combined aerobic dance and slide and step programs may improve balance and coordination post TBI.**

**A vestibular rehabilitation program may improve symptoms of vertigo in patients following TBI.**

### **4.5 Pain Post TBI**

Pain is defined as “an unpleasant sensory and emotional experience associated with actual or potential tissue damage...” (p.210) (International Association for the Study of Pain, 1994); the relationship between pain and tissue damage is not constant nor uniform. Pain following an injury or surgery can be either acute or chronic, and often lasts for months or years post injury. Acute pain is usually associated with defined tissue damage or a pathological process, and although it usually occurs at the time of injury



as a one-time event, it may reoccur as a series of time limited events (Gould, 2007). Chronic pain is usually defined as pain that continues for more than three to six months and is often not as well associated with tissue damage or a pathological process. Using a separate classification, pain can also be defined as subacute (pain between the end of the first month post injury and six months post injury) and recurrent acute (pain that persists over an extended period of time but recurs as isolated pain episodes).

Until very recently, there has been very little information in the literature regarding the prevalence, etiology, assessment, and treatment of pain post TBI (Zasler et al., 2011). This may be the result of pain syndromes being overlooked in patients with a TBI for a number of reasons (Gellman et al., 1996). Multiple etiologies including orthopedic injuries, burns, organ injuries, or central or peripheral nervous system injuries can result in acute or chronic pain in those recovering from a TBI (Ivanhoe & Hartman, 2004). A lack of recognition or diagnosis of pain can lead to an increase in aggression and agitation, or an inability to participate or benefit from rehabilitation (Ivanhoe & Hartman, 2004; Sherman et al., 2006). In individuals who have sustained a moderate or severe TBI, the diagnosis of pain is often made through the combination of symptoms described by the patient and information provided by family members. Pain post TBI can evolve from episodic pain to daily pain with an increasing negative impact over time; pain ultimately impacts participation in rehabilitation and thereby slows recovery (Branca & Lake, 2004).

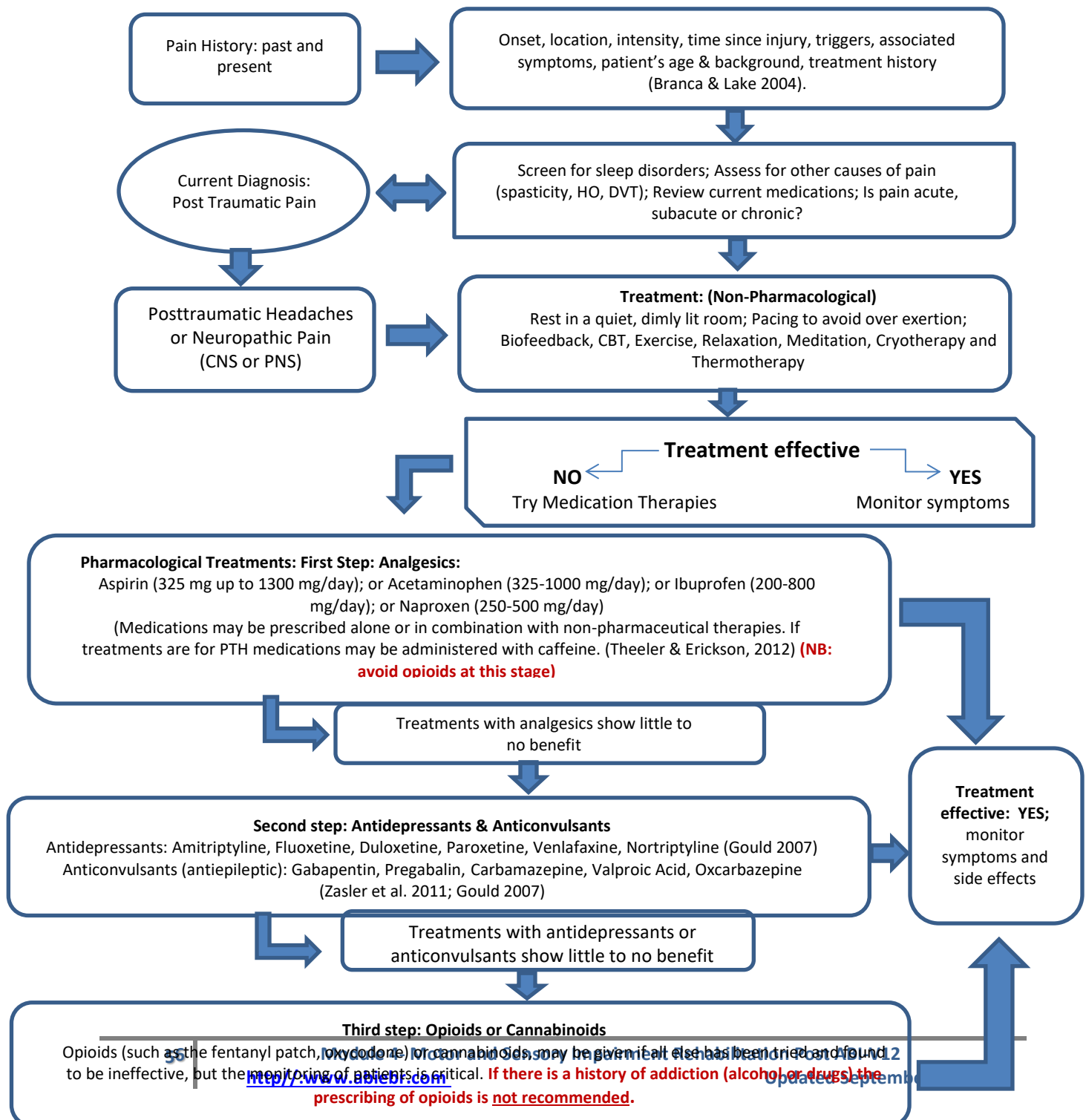
Pain is believed to be more common immediately post injury (acute pain) and it is widely accepted that this pain will resolve as the damaged tissue recovers (Uomoto & Esselman, 1993). The focus is on management of symptoms over a relatively short defined period of time and on assisting the healing of damaged structures. Chronic pain by its very nature may not resolve, or is very slow to resolve, and often manifests itself as post traumatic headaches (PTH), neck and shoulder pain, back pain, peripheral nerve injury, heterotopic ossification, and pain related to spasticity (Hoffman et al., 2007; Lahz & Bryant, 1996; Ofek & Defrin, 2007). In a study conducted by Lahz and Bryant (1996), chronic pain was reported by 52% of those who were diagnosed with a moderate to severe TBI and 58% of those diagnosed with a mild TBI. Of those reporting pain, over 80% reported experiencing pain on a daily basis (Lahz & Bryant, 1996). Comparable rates were given by Hoffman et al. (2007) who examined a bodily pain scale one year post TBI. Of the 146 individuals who participated, 74% of participants reported experiencing pain and 55% of those reported that pain interfered with a variety of daily activities. Higher rates of pain were also related to gender, lower Functional Independence Measure scores, higher rates of depressive symptoms at baseline and again at one year post injury, and lower scores on the Community Integration Scale. Those who were injured in acts of violence reported experiencing greater pain (Hoffman et al., 2007). Pain is significantly associated with depression, with one study reporting rates of pain and depression as 70% and 31%, respectively and 34% and 22%, respectively at one year follow-up (Sullivan-Singh et al., 2014). Pain related to orthopedic injuries, spasticity, or heterotopic ossification will not be covered in this section. For a more detailed discussion on spasticity and treatments post ABI please see section 4.2.5 in this module and a detailed discussion on heterotopic ossification post ABI is available in Module 11. Due to the complexities of pain, we have decided to focus on pain post TBI specifically. The diagnosis of pain post TBI is an important part of an individual's recovery.

Problems associated with pain include a delay in cognitive recovery, sleep disorders, fatigue, elevated levels of anxiety, depression, and post-traumatic stress disorder (Dobscha et al., 2009; Hoffman et al., 2007). Cognitive deficits associated with TBI may prevent individuals from using adaptive pain coping strategies that are critical to the management of chronic pain. When treating pain post TBI, it is important for clinicians to identify the causes of pain, not just the symptoms (Zasler et al., 2011). To reduce the impact on cognitive recovery, treatment plans should take into consideration the



medications the patient is already receiving, as well as the location, type, and frequency of the pain. It should be acknowledged that in many cases the pain generator persists in which case pain can only be managed. Treatment for pain often involves an interdisciplinary approach (Branca & Lake, 2004). To increase the likelihood of compliance with treatments, a good working relationship between physicians and the patient is needed. Overall, more research is needed to assess the effectiveness and efficacy of these treatments in the TBI population. For a summary of these findings please see Figure 1.

**Figure 1: Treatment for Sub-acute/Chronic Pain Post Traumatic Brain Injury**



#### **4.5.1 Assessing Pain Post TBI**

Pain itself is both complex and subjective; thus, self-reports are vital to any treatment plan. Descriptive details related to the intensity, the length of time the pain is felt, the location, and what exacerbates or relieves the pain are vital in developing an individualized treatment strategy (Zasler et al., 2011). Currently there are a number of tools and assessments used by physicians and therapists to assist in diagnosing and measuring pain. Amongst these is the numeric rating scale (NRS), the visual analogue scale (VAS), the Headache Diary, the Headache Disability Inventory, and the McGill Pain Questionnaire.

##### **4.5.1.1 Visual Analog Scale**

The Visual Analog Scale (VAS) is available at no cost and has been used with many adult populations to assess pain intensity. The scale is a horizontal or vertical line, usually 10 centimeters in length and is completed by the respondent. Two verbal descriptors, no pain and the worst pain imaginable, anchor the scale (Zasler et al., 2011). Generally, those completing the scale are asked to describe the pain within the past 24 hours. A ruler is used to the distance between the no pain anchor and the line that the respondent has drawn. Higher scores indicate greater pain (Hawker et al., 2011).

##### **4.5.1.2 Numeric Rating Scale**

The Numeric Rating Scale (NRS) is also commonly used to assess pain. Here the patient rates the pain they are experiencing on a scale of 0 to 10. The NRS, like the VAS, is anchored with no pain and the worst pain imaginable. Individuals are generally asked to report on pain intensity within the past 24 hours. This scale can be administered verbally or graphically. A higher score indicates higher levels of pain (Hawker et al., 2011). A 30% reduction on the NRS is clinically important and has been found to be equivalent to ratings of moderate relief or much improved (Farrar et al., 2001).

##### **4.5.1.3 McGill Pain Questionnaire**

The McGill pain questionnaire, designed for adults with chronic pain, measures the sensory, affective, and evaluative aspects of pain and pain intensity (Hawker et al., 2011). This paper and pencil questionnaire is administered by an interviewer with the respondent present. The respondent is asked questions and must select one word from a list presented to them which best describes their present pain. If the pain an individual is feeling cannot be described by the words presented to them, then no word is selected. Scores are based on the number of words selected with higher scores indicating greater levels of pain.

##### **4.5.1.4 Headache Disability Inventory**

The headache disability inventory was designed to measure the impact of headaches on activities of daily living (Jacobson et al., 1994). This self-report scale consists of 25 items designed to probe the functional and emotional impact of headaches in a patient's life. The scale has been found to be easy to complete - items are measured as either a yes or a no - and simple to score and interpret (Jacobson et al., 1994).

##### **4.5.1.5 Headache Diary**

Those who are asked to maintain a headache diary are required to log their headaches on a daily basis. Recorded in the diary is the time of day the headache begins, the intensity of the pain, any medication

being taken, and any triggers for the headache. Additionally, individuals are asked to record alcohol consumed, periods of fasting, foods eaten, sleep patterns, weather, stress levels, emotional ups and downs, and for women, date of their menstrual cycle (Arnstein, 2004). A variety of headache diaries or headache calendars are currently available. These diaries potentially allow the patient to gain better control over their pain by identifying its potential causes, and provide feedback regarding treatment efficacy (Branca & Lake, 2004).

#### **4.5.2 Pain Syndromes Post ABI**

While the pain an individual with an ABI experiences can vary, there are several defined pain syndromes that are common post ABI. Defining the pain someone experiences as a specific pain syndrome can be valuable in determining the ideal treatment method.

##### **4.5.2.1 Neuropathic Pain**

Neuropathic pain is initiated or caused by a primary lesion or by dysfunction of the nervous system (International Association for the Study of Pain, 1994). Peripheral nervous system pain can result from mechanical trauma, metabolic disease, neurotoxic chemicals, infection, or tumor (Costigan et al., 2009; O'Connor & Dworkin, 2009). On the other hand, central nervous system pain may be associated with spinal cord injury, stroke, TBI or multiple sclerosis (Costigan et al., 2009). Neuropathic pain can result from compression of peripheral nerves when patients are immobilized for long periods of time and diagnosis is often based on careful medical evaluation (careful history, physical and neurological exams, MRI findings, blood and serologic tests) (Dworkin et al., 2003). Despite clear diagnostic guidelines, treatment remains a challenge, as even effective treatments offer only partial pain relief (Finnerup et al., 2005). Common interventions for the treatment of neuropathic pain include pharmacological agents, such as: amitriptyline, pregabalin, gabapentin, duloxetine, carbamazepine, lidocaine, and opioids (Waszkielewicz et al., 2011). When considering treatments, it is important to keep in mind the safety and efficacy of these drugs; particularly how they interact with other medications and how they will impact neurological recovery post TBI. More specific information on interventions for neuropathic pain is discussed in sections 4.5.4.1 (anticonvulsants) and 4.5.4.5 (cannabinoids).

##### **4.5.2.2 Central Pain Syndromes**

Zaslar et al. (2012) have defined central pain as “pain associated with lesions of the central nervous system” (p 967). Treatments are limited in number and efficacy (Nicholson, 2004); however, the goal is often a reduction in pain and not complete relief. For example, studies have examined the effects of lidocaine and IV morphine in alleviating central pain syndromes, but the results have been mixed (Attal et al., 2000; Attal et al., 2002).

##### **4.5.2.3 Post Traumatic Headaches**

Damage to the skull, brain tissue, or cervical spine can result in PTHs that can be serious and incapacitating (Watanabe et al., 2012). According to Elkind (1989), a headache is a common and dominant symptom of TBI, with approximately 44% of those who sustain a closed head injury developing PTHs. Head, neck, and shoulder pain usually begins within the first 24 to 48 hours post injury, however PTH may appear immediately after the injury and subside, or occur days, weeks or months following the injury (Young, 2001). Factors that may lead to PTHs include chronic muscle contraction, chronic and diffuse muscle strain, and anxiety (Hillier et al., 1997). Visual or vestibular system complications may also result in headache syndromes. Studies have found that a PTH often resolves itself within the first 6 to 12 months of injury; however, in 18-33% of the TBI population headaches persist longer than one year (Lew et al., 2006; Seifert & Evans, 2010). Estimating the number of

individuals who develop PTH is difficult as there is lack of consistency in how PTH is defined which may reflect limited understanding of its pathophysiology.

Previously, studies looking at the incidence of PTH reported that those who sustained a mild TBI were more likely to report problems with headaches than those who sustained moderate to severe TBIs (Couch & Bearss, 2001; Gurr & Coetzer, 2005; Uomoto & Esselman, 1993). However, more recent studies have found that individuals with moderate or severe TBIs report experiencing headaches even at one year post TBI (Hoffman et al., 2011; Hoffman et al., 2007; Lainez & Pesquera, 2011). In a survey of 485 individuals, Hoffman and colleagues (2011) found the prevalence of headaches during the first year of recovery post TBI was 40%, regardless of the severity of injury. Lucas (2011) found that almost 60% of respondents who reported experiencing headaches, also reported experiencing migraines or probable migraines. Other headaches reported were tension type headaches, cervicogenic headaches, or unclassifiable headaches. Despite what is known about PTH, there remains a need for further epidemiological, clinical, and pathophysiological studies (Lainez & Pesquera, 2011). Studies evaluating interventions for post traumatic headache can be found in the following sections: Biofeedback to Manage Post Traumatic Headache (section 4.5.3.1), Cognitive Behavioural Theory (section 4.5.3.2), Manual Therapy (section 4.5.3.4), and Cryotherapy and Thermotherapy (section 4.5.3.6).

#### **4.5.3 Non-Pharmacological Interventions for Pain and Post Traumatic Headache**

Early detection and treatment of pain is regarded as crucial to reduce its impact and help individuals develop appropriate approaches to dealing with their pain (Ivanhoe & Parrilla, 2002). As mentioned previously, deciding on a treatment for both general pain and PTHs may be challenging due to the many underlying factors of TBI and the fact that some pain conditions are only partially responsive to treatment. Given that pharmacological interventions may worsen cognitive deficits post TBI, non-pharmacological interventions should be incorporated into the treatment plan.

Non-pharmacological interventions for both chronic pain and PTH may include: biofeedback, cold and heat packs, massage therapy, acupuncture, and exercise (Medina, 1992). Biofeedback, relaxation, meditation, and CBT are considered the gold standard of behavioural treatments for pain (Branca & Lake, 2004). In a recent review of manual treatments for migraines, massage therapy, physiotherapy, relaxation, and chiropractic spinal manipulative therapy were found to be just as effective as propranolol and topiramate at reducing symptoms (Cassidy et al., 2014). Physiotherapy exercises have also been suggested to treat pain; however, unless the pain is controlled, caution is recommended when using these exercises to prevent aggravating the painful structures further (Medina, 1992). Lifestyle changes are also suggested to prevent the onset of PTH, such as getting enough sleep and daily exercise.

##### **4.5.3.1 Biofeedback to Manage Post Traumatic Headache**

According to a study by Mullally et al. (2009), biofeedback therapy was not found to significantly reduce the number and severity of headaches in individuals who were diagnosed with migraines or tension type headaches. However, several earlier studies found more positive results. A study by Ham and Packard (1996) studied 40 individuals who sustained a mild TBI and were experiencing post traumatic headaches. Subjects participated in biofeedback sessions and were placed on anti-depressants and anti-inflammatory or non-narcotic analgesics. Participants began treatment on average 12.7 months post injury, although in half of the participants PTHs occurred immediately post injury. Biofeedback was reported to help 93% of participants to some degree with those who waited longer to begin biofeedback therapy found it less successful. Individuals who had more sessions and began treatment sooner found

the sessions to be very beneficial. Unfortunately, the research on this topic for the moderate to severe TBI population is limited.

In a study by Tatrow et al. (2003), PTHs were targeted for six weeks in 14 mild TBI individuals. The first four sessions consisted of progressive muscle relaxation, with biofeedback (thermal and EMG) being introduced in the fifth session. Participants learned to relax and control muscle tension, and relaxation ratings were on average 8.6 out of 10. Improvements in PTH were shown for most participants and the treatment also lowered post-concussion syndrome checklist scores significantly in the treatment group (Tatrow et al., 2003).

#### 4.5.3.2 Cognitive Behavioural Therapy

Cognitive behavioural therapy (CBT) has been used to assist individuals in managing their pain. It is considered a diverse set of problem-specific interventions and incorporates physical, psychological, and behavioural approaches to managing pain (Roth & Pilling, 2008). With CBT the individual is taught to use self-regulation and self-control, and to take responsibility for one's lifestyle (Martelli, 2012). This therapy has been used to help patients cope with the pain, depression, and anxiety associated with chronic headaches (Gurr & Coetzer, 2005; Wetherell et al., 2011). Despite the extensive use of CBT, there are not many studies evaluating its efficacy in treating pain post ABI (Table 4.20).

**Table 4.20 Cognitive Behavioural Therapy for Pain Management Post ABI**

Author/Year/ Country/Study Design/N	Methods	Outcomes
<a href="#">Gurr and Coetzer</a> (2005) UK Pre-Post N <sub>Initial</sub> =41, N <sub>Final</sub> =20	<p><b>Population:</b> TBI; Mean Age=44.05 yr; Gender: Male=28, Female=13; Mean Time Post Injury=78.7 mo.</p> <p><b>Intervention:</b> The CBT program consisted of 3 weekly relaxation group sessions, which were followed by six 30 min individual therapy sessions. Psychological intervention included: progressive muscle relaxation-combined with the use of imagery, psycho-education tailored to the individual, cognitive behavioural strategies, lifestyle management, and maintenance and relapse.</p> <p><b>Outcome Measure:</b> Structured Diagnostic Interview, Headache Disability Inventory, Headache Needs Assessment (HANA), Nottingham Health Profile (NHP), Chronic Pain Index (CPI).</p>	<ol style="list-style-type: none"> <li>Twenty-four participants had tension-type headaches, 7 migraines, 4 had both of the previous types, 3 had tension-type and soft tissue/scar pain, and 3 had other types.</li> <li>Headaches occurred a mean 14 days per month and the mean length was 10.46 hr.</li> <li>Following the intervention, headache intensity (CPI) and frequency decreased significantly (<math>p=0.004</math>).</li> <li>Headache disability, according to results on the HDI and HANA, were significantly reduced (<math>p=0.001</math> and <math>p=0.02</math> respectively).</li> <li>According to the NHP, pain was reduced but this was not significant.</li> <li>Emotional well-being as measured by the HDI-emotion and the NHP-emotion subscales was also significantly improved (<math>p&lt;0.05</math>).</li> </ol>

#### Discussion

Gurr and Coetzer (2005) studied twenty participants who had sustained a mild to severe TBI and who had completed a CBT program for PTH. The CBT program consisted of progressive muscle relations, psycho-education, cognitive behavioral strategies, lifestyle management and maintenance, and relapse prevention. After the intervention, headache intensity and frequency, and disability significantly decreased, while emotional wellbeing increased. Results from the Nottingham Health Profile pain scale found no significant differences in pain pre and post intervention.

#### Conclusions

*There is level 4 evidence that cognitive behavioural therapy may improve post traumatic headache intensity and frequency but not pain, in those who have sustained a mild to severe TBI.*

**Cognitive behavioural therapy may be useful in managing post-traumatic headaches; however, may not be useful for headache-associated pain.**

#### **4.5.3.3 Relaxation Training**

Relaxation training, or progressive muscle relaxation, is a possible treatment for chronic pain and PTHs. Individuals are taught how to breathe from the diaphragm and how to tense and relax various muscles. Through such techniques, the muscle tension triggers associated with headaches can be better controlled (Arnstein, 2004). Over time, patients may be able to reduce the number of headaches or prevent the pain from worsening.

#### **4.5.3.4 Manual Therapy**

Manual therapy refers to more hands-on types of therapy such as massage therapy, traction, mobilization, and physical therapy. The purpose of these therapies is to reduce muscle tension, increase muscle length, enhance circulation, and increase mobility in the joints (Gould, 2007). The results from an earlier study indicated that manual therapy was more effective than cold packs in reducing pain associated with PTHs (Jensen et al., 1990).

Massage therapy involves either deep tissue massage or a lighter massage technique. Massage therapy has been shown to increase oxygenation and blood flow to the muscles being treated as well as to reduce pain (D'Arcy, 2011). Physical therapy involves the patients and a physical therapist working together to identify the areas where pain is being experienced. Therapy may involve stretching and or strengthening exercises, ultrasound to the affected areas, or the application of hot and cold packs. Physical therapy for both pain and chronic daily headaches focuses on the upper body, including the upper back, neck, and face (Sherman et al., 2006).

In an earlier study, Medina (1992) investigated the treatment of PTHs in 20 patients post TBI or spinal cord injury through individualized therapeutic sessions each lasting 30 minutes. Patients received educational sessions, biofeedback training, electromyographic biofeedback, or physical therapy sessions, and were placed on appropriate medication to treat the pain. The combination therapies were effective as 17 patients were able to return to work and 19 patients reported a decrease in PTH intensity.

#### **4.5.3.5 Acupuncture**

Acupuncture, one of the oldest forms of physical therapies, is a non-medicinal intervention involving a certified acupuncturist selecting specific points on the body to insert acupuncture needles. The points of insertion differ in every individual. Although research indicates that there is some benefit to acupuncture therapy, there is a lack of strong evidence supporting its effectiveness with the TBI population (Gould, 2007; Wong et al., 2012).



#### 4.5.3.6 Cryotherapy and Thermotherapy

Heating and cooling therapy can provide relief to TBI patients who suffer chronic headaches and neck pain. Specifically, cryotherapy involves the application of cold to relieve pain while thermotherapy involves the application of heat to relieve pain (Pangarkar & Lee, 2011). Both therapies are typically used in conjunction with other treatments.

**Table 4.21 Cold Therapy for Pain Management Post ABI**

Author/Year/ Country/Study Design/PEDro Score/N	Methods	Outcomes
<a href="#">Jensen et al.</a> (1990) Denmark RCT PEDro=4 N=19	<p><b>Population:</b> TBI; Mean Age=31.6 yr; Gender: Male=7, Female=12; Mean Time Post Injury=359 d.</p> <p><b>Intervention:</b> Patients were randomized to one of two groups: the manual therapy group (n=10) or the cold pack group (n=9). Those in the manual therapy group received soft passive movements of the joint at the outer range of motion. Cold pack therapy involved placing the cold pack under the neck and shoulders of the individual. Each intervention period lasted 15 to 20 min. Interventions were given over a 12 wk period.</p> <p><b>Outcome Measure:</b> Pain Schedules, Intensity of Headache on Visual Analogue Scale.</p>	<ol style="list-style-type: none"> <li>1. The pain index of those in the manual therapy group declined after the two treatments while remaining relatively constant in the cold pack group.</li> <li>2. The manual therapy group reported the greatest reduction in pain at 5 wk.</li> <li>3. Reduction in pain was significantly different (<math>p&lt;0.05</math>) between the two groups at 5 wk, with the manual therapy group reporting significantly less pain than the cold pack group.</li> <li>4. Pain reduction for the manual therapy group decreased by 84% at 6 wk.</li> </ol>

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

#### Discussion

Jensen et al. (1990) compared manual therapy to cold pack therapy for the treatment of PTH pain in 19 participants with head injury. Those in the manual therapy group reported a significantly greater reduction in pain following the intervention when compared to the cold pack group. The pain index for all participants was also correlated with the frequency of associated symptoms (dizziness, visual disturbances, and ear symptoms) and the use of analgesics.

#### Conclusions

*There is level 2 evidence that cold therapy may not be as effective as manual therapy for reducing post traumatic headache pain in individuals post ABI.*

**Cold therapy is likely not as effective as manual therapy at reducing post traumatic headache pain in patients post TBI.**

#### 4.5.4 Pharmacological Management of Pain and Post Traumatic Headache

There are a variety of medications used in the treatment of chronic pain post ABI and PTH. Aspirin or aspirin compounds, acetaminophen, and ibuprofen are often the first lines of treatment prescribed for chronic pain; however, adjuvant medications such as anticonvulsants, antidepressants, benzodiazepines,



bisphosphonates, local anesthetics, antispasmodic agents, and topical agents may also be prescribed (Gould, 2007; Khan et al., 2011). In some cases, the prescription of opioids may be considered.

#### 4.5.4.1 Anticonvulsants

The administration of anticonvulsants to treat pain post brain injury has been a common practice since the 1960's. It is thought that epilepsy and pain share a common pathogenesis, thus allowing anticonvulsant medications to be used in pain management, particularly neuropathic pain that is either peripheral or central in origin (Dickinson et al., 2000; Zasler et al., 2011). It has also been noted that the use of anticonvulsant medication seems to produce fewer adverse events (Gould, 2007).

Anticonvulsants currently used to treat pain include carbamazepine, oxcarbamazepine, lamotrigine, gabapentin, pregabalin, and topiramate; however, there are limited studies investigating their effectiveness either in isolation or in combination with other medications. Table 4.22 summarizes several antiepileptic medications that are used to treat pain post ABI.

**Table 4.22 Antiepileptic Medications to Treat Pain Post TBI (Gould, 2007; Guay, 2003; Zasler et al., 2011)**

Antiepileptic Medication	Typical Dose; Dose Range	Adverse Events (partial list)
Carbamazepine (Tegretol®)	200 mg q 8 hr; 100-1600 mg/day	Drowsiness, bone marrow suppression, kidney stones
Valproic acid (Depekene®)	250 mg q 8 hr; 600-2400 mg/day	Drowsiness, headache, sleepiness, agitation, mood swings, memory loss
Phenytoin (Dilantin®)	100 mg q 8 hr; 200-600 mg/day	Double vision, imbalance, slurred speech
Gabapentine (Neurontin®)	600 mg q 8hr; 200-3600 mg/day	Drowsiness, dizziness
Clonazepam (Klonopin®)	0.5 mg q 8 hr; 2-7 mg/day	Drowsiness, disequilibrium, abnormal behavior
Oxcarbazepine (Tripeptal®)	300-600 mg q 12 hr; 150-1800 mg/day	Drowsiness, lightheadedness, dizziness
Lamotrigine (Lamictal®)	50-100 mg q 12hr; 50-200 mg/day	Rash, fatigue, stomach upset
Topiramate	25 mg q 12 hr; 200-400 mg/day	Ataxia, impaired concentration, confusion, dizziness, fatigue, speech disturbances, language problems.
Pregabalin (Lyrica®)	300-450 mg/day; 150-600 mg/day	Drowsiness, dizziness
Levetiracetam (Keppra®)	250-500 mg q 12 hr; 250-1500 mg/day	Drowsiness, dizziness

#### Valporic Acid and Divalproex Sodium

Valporic acid and divalproex sodium are antiepileptic medications that have been used to reduce pain; however, there are no clinical trials demonstrating its efficacy post moderate to severe TBI. A retrospective study was identified that investigated the effectiveness of divalproex sodium on PTH in a mild TBI population (Packard, 2000). The dosing was individualized, however the starting dose was 250 mg per day and was increased by 250 mg as needed. Results showed that 60% of those on divalproex found mild to moderate improvement in chronic daily PTH. Further, 19 of 28 participants who had chronic daily headaches reported mild to moderate improvement. Additionally, six participants were headache free after a single month of treatment. The authors suggest that divalproex is effective as it works by activating the inhibitory neurotransmitter  $\gamma$  aminobutyric acid (GABA). The authors noted that although PTHs that persist for more than one year are considered permanent, it is still possible to treat them effectively, allowing the patient to increase their activity level and perhaps reduce their dependence on other analgesics.

#### Pregabalin and Gabapentin

Pregabalin and gabapentin work by blocking neuronal calcium channels and have become widely used for the treatment of neuropathic pain; in particular peripheral neuropathic pain. Gabapentin requires a

longer time to reach a therapeutic dose compared to pregabalin, which can provide a faster response to pain as it has 90% bioavailability. Furthermore, pregabalin does not appear to have a negative effect on other medications that an individual may be taking (D'Arcy, 2011; Vranken et al., 2011). Gabapentin has been found to be very effective in the treatment of neuropathic pain in the elderly and is considered the first line of defense with this population (Guay, 2003). Again, despite these medications being administered to individuals post TBI, there is limited literature supporting their effectiveness in this population.

In a RCT by Vranken et al. (2008), the effects of pregabalin on pain in those with either an ABI or spinal cord injury were examined. The intervention group received a flexible dose of pregabalin, starting at 150 mg per day. If the pain relief was insufficient, the dose of pregabalin was increased to 300 mg, then 600 mg if needed. The control group received a placebo. Participants were administered the medication or placebo twice a day over the span of four weeks. Results indicate those in the pregabalin group reported a significant decrease in pain intensity, measured by a VAS, compared to the control group ( $p < 0.010$ ). When using the Pain Disability Index, no significant between-group differences were noted at the end of the intervention. Of note, those in the pregabalin group reported few side effects, indicating that the medication was well tolerated (Vranken et al., 2008).

### **Carbamazepine and Oxcarbazepine**

Carbamazepine was once considered the drug of first choice for treating neuropathic pain (Gould, 2007). Despite its success, there has been considerable concern regarding the adverse effects that many individuals experience while on the medication. If administering carbamazepine, the monitoring of such adverse events is strongly recommended (Backonja, 2003). Oxcarbazepine has been found to be effective in the treatment of neuropathic pain and has also been reported to have an improved side effect profile compared to carbamazepine (Nasreddine & Beydoun, 2007). In patients experiencing painful diabetic neuropathy, oxcarbazepine resulted in decreased VAS pain scores; however, changes were not always statistically significant. When administered to patients with the pain of trigeminal neuralgia, oxcarbazepine was found to be effective in reducing pain (Nasreddine & Beydoun, 2007).

### **Lamotrigine**

Lamotrigine, a newer anticonvulsant medication, has also been found to aid in the treatment of neuropathic pain often associated with diabetic neuropathy, spinal cord injury pain, phantom limb pain, postoperative and traumatic neuropathic pain, and cancer related neuropathy (Ettinger & Argoff, 2007; Wiffen et al., 2011).

### **Topiramate**

Topiramate has been found to be successful in treating migraine and cluster headaches (Ettinger & Argoff, 2007). Although topiramate is believed to be effective in treating pain, it has been noted that any preexisting cognitive impairments (language, attentions, cognitive functioning, or memory) due to the brain injury itself may be exacerbated by topiramate (Tang et al., 2007).

#### **4.5.4.2 Antidepressants**

Among the antidepressant medications available, tricyclic antidepressants are the most commonly used for the treatment of pain; in particular neuropathic pain (Gironda et al., 2009; Gordon & Love, 2004; Guindon et al., 2007). Medications such as fluoxetine, sertraline, paroxetine, or citalopram work best at controlling pain when there is an underlying primary problem such as depression. Tricyclic antidepressants used to treat pain include amitriptyline, nortriptyline, desipramine, doxepin, and imipramine (Gould, 2007); however, the mechanism of action for these medications in the treatment of

pain is not yet fully understood. Currently, there is no evidence to support the administration of antidepressants to treat pain or PTH; Table 4.23 summarizes several antidepressants that are used to treat pain post ABI.

**Table 4.23 Antidepressants to Treat Pain Post ABI (Gould, 2007; Zasler et al., 2011)**

Antidepressants	Typical Dose; Dose Range	Adverse Events
Amitriptyline (Elavil®)	75 mg qhs; 10-150 mg/day	Drowsiness, dry mouth, weight gain, constipation, seizures, cardiac toxicity, urinary retention
Venlafaxine (Effexor®)	37.5 mg od; 150-225 mg/day	High blood pressure, weight loss, dry mouth, impotence, tremor
Nortriptyline (Pamelor®)	75 mg qhs; 10-150 mg/day	Drowsiness, dry mouth, weight gain, constipation, seizures, cardiac toxicity, urinary retention
Desipramine (Norpramin®)	75 mg qhs; 50-200 mg/day	Drowsiness, dry mouth, weight gain, constipation, seizures, cardiac toxicity, urinary retention
Duloxetine (Cymbalta®)	60 mg qd; 3-120 mg/day	Insomnia, nausea, dizziness, fatigue, constipation
Fluoxetine (Prozac®)	20 mg qd; 5-60 mg/day	Anxiety, nervousness, insomnia, tremor, chest pain, diarrhea
Paroxetine (Paxil®)	20-40 mg qd; 20-50 mg/day	Drowsiness, dizziness, insomnia, headache

### **Amitriptyline**

Amitriptyline is often used to treat headaches in those who suffer from migraines and has been found to be very effective in reducing symptoms (Pringsheim et al., 2012). Along with treating migraines, amitriptyline has been used to treat pain in patients since 1964 when its efficacy was first demonstrated (Adelman et al., 2000). In addition, amitriptyline is used to treat pain in those who have suffered a stroke or spinal cord injury, and in those who have been diagnosed with dementia, multiple sclerosis, fibromyalgia, or chronic diabetic peripheral neuropathic pain. The effectiveness of amitriptyline in the treatment of pain post TBI is not well studied.

### **Venlafaxine**

Venlafaxine, a serotonin and norepinephrine reuptake inhibitor, has been shown to be a safe and effective medication in the treatment of depression and anxiety, as well as various pain syndromes including: neuropathic pain, headaches, fibromyalgia, painful peripheral diabetic neuropathy, and post-mastectomy pain (Dharmshaktu et al., 2012). Venlafaxine, amitriptyline, and propranolol are the most prescribed medications in the treatment of migraine-related pain, and chronic tension type headaches (Adelman et al., 2000).

#### **4.5.4.3 Topical Analgesics**

Pain that is described as localized and superficial has been treated effectively with topical treatments. Topical analgesics include menthol/methylsalicylates, capsaicin, and anesthetics; however, as with the previously discussed medications, there is no clinical evidence to support the use of topical analgesics. Menthol has been shown to be somewhat effective as it releases a cooling sensation over the painful area (Pangarkar & Lee, 2011). Capsaicin cream has been found to cause a burning sensation, so it is strongly recommended to apply the cream only to where the pain is located. Despite this, capsaicin cream has been found to decrease neck pain (Pangarkar & Lee, 2011). The lidocaine patch is applied to the painful area and worn for typically 12 hours. The patch tends to be well tolerated by most (D'Arcy, 2011). More studies are required to determine the efficacy of topical treatments; Table 4.24 summarizes several topical anesthetics that are used to treat pain post ABI.

**Table 4.24 Topical Anesthetics to Treat Pain Post TBI (Gould, 2007)**

Medication	Typical Dose	Adverse Events
Capsaicin (Zostrix®, Axsain®)	0.025-0.075% 3-4 times daily	Burning, skin irritation
Lidocaine 5% (Lidoderm®)	1-4 patches 12 hours per day	Skin irritation

#### 4.5.4.4 Opioids

The use of opioids to manage non-cancer pain has been on the rise for the past several decades (Martelli, 2012). It is believed that neuropathic pain can be relieved by the administration of opioids, provided a balance exists between the optimal dose and any unmanageable side-effects (Dellemijn, 1999). A decrease in libido, sedation, mental dullness, difficulties concentrating, and a higher risk for developing osteoporosis have been reported when taking opioids (Ersek et al., 2004; Haanpaa et al., 2010). The risk of exacerbating cognitive impairments of patients with TBI is one of the reasons for clinicians' hesitancy to administer opioids for pain management. Although opioid use within a TBI population has been discussed in the literature, few studies investigate its efficacy for reducing pain within this population. When opioids are administered it has been suggested to start with a low dose and titrate up (Gallagher et al., 2006). Unfortunately with narcotics there is no recommended therapeutic dose (Khan et al., 2011); although with musculoskeletal complaints it is recommended that the dose not exceed 120-200 mg/day morphine equivalent dose (Haanpaa et al., 2010). Moreover, with opioids, because the risk of physical dependency and addiction is problematic, patients should be screened for addiction and dependency risk factors. Psychological problems and a history of substance abuse are considered the two most common risk factors for opioid misuse and addiction.

Franceschi et al. (2008) administered oxycodone to a group of polytrauma patients, five of which had mild head injury, admitted to an emergency department suffering from acute pain. Main pain sites for the group were the chest, neck, lower back, leg, heel, pelvis, upper arm, and shoulder. Oxycodone (10 mg twice per day for three days given orally) was found to significantly reduce pain for 14 of the 15 patients. One patient required an increase in medication (20 mg twice per day) to achieve pain relief. Overall the medication was well tolerated by patients with some reporting mild side effects (light headaches, constipation and nausea) (Franceschi et al., 2008). Oxycodone has been found to be successful in reducing pain; however, it remains unclear as to whether this medication would be effective and well tolerated in those who sustain a moderate or severe ABI.

#### 4.5.4.5 Cannabinoids

Cannabis sativa has been used to treat pain for centuries. However the use of cannabis and its derivatives to treat pain had fallen out of favour in the mid 1940's to the mid 1990's, possibly due to the suspected risk of addiction, abuse, dependence, cognitive effects, and other adverse medical and psychiatric effects (Aggarwal, 2013; Greenwell, 2012). The utility of the medication is gaining increasing recognition as physicians and other health care professionals increase their knowledge regarding the efficacy and safety of cannabinoid based medications (Aggarwal, 2013). Cannabis is generally administered through either inhalation, ingestion, or topically, with the method of delivery determining the rate at which the drug begins to take effect. According to Aggarwal (2013), the use of cannabinoids can result in muscle relaxation, anti-inflammatory effects, and neuroprotection in ischemia and hypoxia. Currently, cannabinoids are used to treat cancer pain, pain associated with multiple sclerosis, human immunodeficiency virus, fibromyalgia, and rheumatoid arthritis. Although many studies have looked at the benefits of using cannabinoids to treat chronic pain, the results of many of these studies were inconclusive (Greenwell, 2012).

A study by Ware et al. (2010) examined the effects of cannabis at different potencies (0%, 2.5%, 6% and 9.4%) in individuals with post-traumatic or postsurgical neuropathic pain. Pain intensity was found to be significantly lower on 9.4% tetrahydrocannabinol cannabis than on 0% tetrahydrocannabinol ( $p=0.023$ ). Further, when 9.4% tetrahydrocannabinol cannabis was compared to taking a placebo, patients experienced more drowsiness and fewer periods of wakefulness. Results from Ware et al. (2010) suggest cannabis is effective in the treatment of neuropathic pain. Due to the addictive properties of this group, cannabinoids should only be administered if there is no history of alcohol or drug addiction. Once on these medications, monitoring of patients is paramount.

#### **4.6 Conclusions**

Overall, a wide variety of interventions exist for sensory and motor rehabilitation post ABI. For motor impairment specifically a variety of interventions such as splinting, constraint induced movement therapy, and exercise programs have been shown to be effective for the remediation of motor deficits post ABI.

More pharmacological based interventions exist for the treatment of spasticity in general, compared to other areas of motor rehabilitation. The spasticity studies presented here present multiple therapeutic options as well as compare their efficacy in ABI specific populations. It is important to keep in mind that some of the pharmacological interventions discussed have a longer history of investigation than others, such as botulinum toxin injections, while newer pharmacological interventions may want to be interpreted with more care.

Ultimately the appropriate interventions should be agreed upon by the care-team with what is in the best interest of the patient, as well as discussing realistic expectations for recovery.

#### 4.7 Summary

*There is level 4 evidence that constraint induced movement therapy (CIMT) or modified CIMT may improve upper extremity function in individuals post ABI.*

*There is level 1b evidence that nocturnal hand splinting may not improve upper extremity range of motion or function compared to standard care in individuals post ABI.*

*There is level 4 evidence that soft hand splinting, but not manual therapy, may improve hand opening in individuals post ABI.*

*There is level 2 evidence that functional retraining activities may be more effective than tabletop fine motor control retraining activities for improving fine motor function in the dominant hand in individuals post ABI.*

*There is level 4 evidence that visual feedback-based grip force training may improve tracking accuracy and transfer tasks in individuals post ABI.*

*There is level 2 evidence that gesture recognition biofeedback may improve fine motor function compared to standard repetitive training without feedback in individuals post ABI.*

*There is level 2 evidence that virtual reality training may improve neurobehavioral functioning as well as reaching accuracy and movements post-ABI.*

*There is level 2 evidence that body weight supported treadmill training may not improve ambulation or mobility compared to conventional gait training in individuals post ABI.*

*There is level 1b evidence that physical therapy with partial weight-bearing gait training may not improve ambulation, mobility, or balance compared to standard physical therapy in individuals post ABI.*

*There is level 2 evidence that robotic assisted body weight supported treadmill training may not improve ambulation or gait velocity compared to manually assisted treadmill training in individuals post ABI.*

*There is level 1b evidence that sit-to-stand training combined with usual rehabilitation may improve motor performance in sit-to-stand tasks compared to usual rehabilitation in individuals post ABI.*

*There is level 2 evidence that electrical muscle stimulation with passive exercise may reduce lower extremity muscle atrophy compared to passive exercise in individuals post ABI.*

*There is level 4 evidence that Intensive Mobility Training may improve ambulation and mobility in individuals post ABI.*

*There is level 1b evidence that virtual reality training compared to balance training may not be more effective for improving lower extremity function post-ABI. However, virtual reality training was shown to improve function independently.*

*There is level 4 evidence that visual feedback may reduce weight-bearing asymmetry in the lower extremities post-ABI.*

*There is level 1b evidence that virtual reality-based training may not improve balance and gait compared to standard physical therapy in individuals post ABI.*

*There is level 4 evidence that virtual reality therapy may improve balance, gait, and functional reaching in individuals post ABI.*

*There is level 1b evidence that participating in an exercise program at a fitness-center compared to home-based exercise program may lead to greater program adherence but not significantly different motor results in individuals post-ABI.*

*There is level 2 evidence that aerobic training compared to vocational rehabilitation may be more effective at improving co-ordination, strength, flexibility, and endurance in individuals post-ABI.*

*There is level 1b evidence that exercise programs may improve FIM scores, but not balance or mobility compared to relaxation training in individuals post-ABI.*

*There is level 4 evidence that multimodal exercise programs may improve gait and mobility in individuals post-ABI.*

*There is level 3 evidence that a home-based exercise program may improve stability to the level of healthy controls, but may not improve motor control, mobility, or dual-task performance in individuals post-ABI.*

*There is level 2 evidence that aerobic dance training compared to musculature training may improve sensory interaction and balance post-ABI.*

*There is level 4 evidence that botulinum toxin type A injections may be effective in the management of localized spasticity following ABI.*

*There is level 1b evidence that receiving botulinum toxin type A through a single motor point or multisite distributed injections are similar at reducing spasticity in individuals with an ABI.*

*There is level 4 evidence that phenol nerve blocks may reduce contractures and spasticity at the elbow, wrist, and finger flexors for up to five months post injection in individuals post ABI.*

*There is level 4 evidence that electrical stimulation may be effective for decreasing lower extremity spasticity for six or more hours, lasting up to 24 hours, in individuals post ABI.*

*There is level 4 evidence that oral baclofen may improve lower extremity spasticity, but not upper extremity spasticity, in individuals post ABI.*

*There is level 1b evidence that bolus intrathecal baclofen injections may produce short-term (up to six hours) reductions in upper and lower extremity spasticity compared to placebo following ABI.*



*There is level 4 evidence to suggest that prolonged intrathecal baclofen may result in longer-term (three months, and one year) reductions in spasticity in both the upper and lower extremities following an ABI.*

*There is conflicting level 4 evidence to suggest that intrathecal baclofen may result in short-term improvement of walking performance in ambulatory patients, particularly gait velocity, stride length, and step width, in individuals post ABI.*

*There is level 1b evidence that serial casting may improve contractures of the elbow initially, but not long-term, when compared to passive stretching in individuals with an ABI.*

*There is level 1b evidence that serial casting may be superior to passive stretching at improving spasticity of the elbow in individuals post ABI.*

*There is level 2 evidence that a below-knee casting and stretching protocol may increase passive ankle dorsiflexion in patients post ABI.*

*There is level 4 evidence that weekly below-knee casts may improve ankle range of motion, muscle extensibility, and passive torque in patients post ABI.*

*There is level 3 evidence that short duration (one to four days) and longer duration (five to seven days) serial casting may have similar effects on upper or lower extremity range of motion in individuals post ABI.*

*There is level 1b evidence that nocturnal hand splinting may not improve upper extremity range of motion or function compared to standard care in individuals post ABI.*

*There is level 1b evidence that electrical stimulation in combination with tilt table standing and splinting may decrease spasticity at 6 weeks post intervention compared to tilt table standing alone in patients with an ABI.*

*There is level 2 evidence that botulinum toxin combined with casting may not be more effective than botulinum toxin injections alone in improving leg spasticity in individuals with an ABI.*

*There is level 1b evidence that neural tension technique may not be more effective than random passive movement in improving lower extremity spasticity and range of motion in individuals with an ABI.*

*There is level 1b evidence that computer-based restitution training may be effective in improving the vision of those who sustain a TBI compared to visual fixation training.*

*There is level 2 evidence that saccade visual tracking compared to fixation and pursuit tracking may improve single-line and multi-line reading post ABI.*

*There is level 4 evidence showing that base-in prisms and bi-nasal occluders can be effective in treating ambient vision disturbances resulting from an ABI.*

*There is level 4 evidence that prismatic spectacle lenses may be effective in reducing symptom burden in patients with vertical heterophoria and post-concussive symptoms post injury.*

*There is level 4 evidence that rehabilitation programs directed at improving visual function can improve functional outcomes such as reading in patients post ABI.*

*There is level 4 evidence that vestibular rehabilitation programs, such as a behavioural exposure program, may improve symptoms of vertigo in patients after TBI.*

*There is level 2 evidence that vestibular rehabilitation programs, alone or in combination with betahistine dihydrochloride, can improve recovery time for balance disorders in individuals with an ABI compared to betahistine dihydrochloride alone.*

*There is level 2 evidence to that using a combined aerobic dancing and slide and step training program may reduce balance and coordination deficits post TBI.*

*There is level 4 evidence that cognitive behavioural therapy may improve post traumatic headache intensity and frequency but not pain, in those who have sustained a mild to severe TBI.*

*There is level 2 evidence that cold therapy may not be as effective as manual therapy for reducing post traumatic headache pain in individuals post ABI.*

## 4.8 References

## References

- Adelman, L. C., Adelman, J. U., Von Seggern, R., & Mannix, L. K. (2000). Venlafaxine extended release (XR) for the prophylaxis of migraine and tension-type headache: A retrospective study in a clinical setting. *Headache*, 40(7), 572-580.
- Aggarwal, S. K. (2013). Cannabinergic pain medicine: a concise clinical primer and survey of randomized-controlled trial results. *Clin J Pain*, 29(2), 162-171.
- Arnstein, P. (2004). Chronic neuropathic pain: issues in patient education. *Pain Manag Nurs*, 5(4 Suppl 1), 34-41.
- Attal, N., Gaude, V., Brasseur, L., Dupuy, M., Guirimand, F., Parker, F., & Bouhassira, D. (2000). Intravenous lidocaine in central pain: a double-blind, placebo-controlled, psychophysical study. *Neurology*, 54(3), 564-574.
- Attal, N., Guirimand, F., Brasseur, L., Gaude, V., Chauvin, M., & Bouhassira, D. (2002). Effects of IV morphine in central pain: a randomized placebo-controlled study. *Neurology*, 58(4), 554-563.
- Backonja, M. (2003). Anticonvulsants for the treatment of neuropathic pain syndromes. *Curr Pain Headache Rep*, 7(1), 39-42.
- Bateman, A., Culpan, F. J., Pickering, A. D., Powell, J. H., Scott, O. M., & Greenwood, R. J. (2001). The effect of aerobic training on rehabilitation outcomes after recent severe brain injury: a randomized controlled evaluation. *Arch Phys Med Rehabil*, 82(2), 174-182.
- Becker, R., Alberti, O., & Bauer, B. L. (1997). Continuous intrathecal baclofen infusion in severe spasticity after traumatic or hypoxic brain injury. *Journal of Neurology*, 244(3), 160-166.
- Bellon, K., Kolakowsky-Hayner, S., Wright, J., Huie, H., Toda, K., Bushnik, T., & Englander, J. (2015). A home-based walking study to ameliorate perceived stress and depressive symptoms in people with a traumatic brain injury. *Brain Inj*, 29(3), 313-319.
- Berger, S., Kaldenberg, J., Selmane, R., & Carlo, S. (2016). Effectiveness of Interventions to Address Visual and Visual-Perceptual Impairments to Improve Occupational Performance in Adults With Traumatic Brain Injury: A Systematic Review. *Am J Occup Ther*, 70(3), 7003180010p7003180011-7003180017.
- Boake, C., Francisco, G. E., Ivanhoe, C. B., & Kothari, S. (2000). Brain injury rehabilitation. In B. RL (Ed.), *Physical medicine and rehabilitation* (pp. 1073-1116). Toronto: Saunders Company.
- Bontke, C. F., Lehmkuhl, D. I., Englander, J., Mann, N., Ragnarsson, K. T., Zasler, N. D., Graves, D. E., Thoi, L. I., & Jung, C. (1993). Medical complications and associated injuries of persons treated in the traumatic brain injury model systems programs. *The Journal of Head Trauma Rehabilitation*, 8(2), 34-46.
- Branca, B., & Lake, A. E. (2004). Psychological and neuropsychological integration in multidisciplinary pain management after TBI. *J Head Trauma Rehabil*, 19(1), 40-57.
- Brown, T. H., Mount, J., Rouland, B. L., Kautz, K. A., Barnes, R. M., & Kim, J. (2005). Body weight-supported treadmill training versus conventional gait training for people with chronic traumatic brain injury. *J Head Trauma Rehabil*, 20(5), 402-415.

- Bushbacher, R. M., & Porter, C. D. (2000). Deconditioning, conditioning, and the benefits of exercise. In Braddom RL (Ed.), *Physical Medicine and Rehabilitation* (pp. 702-726). Toronto: Saunders Company.
- Canning, C. G., Shepherd, R. B., Carr, J. H., Alison, J. A., Wade, L., & White, A. (2003). A randomized controlled trial of the effects of intensive sit-to-stand training after recent traumatic brain injury on sit-to-stand performance. *Clin Rehabil*, 17(4), 355-362.
- Cassidy, J. D., Boyle, E., & Carroll, L. J. (2014). Population-based, inception cohort study of the incidence, course, and prognosis of mild traumatic brain injury after motor vehicle collisions. *Arch Phys Med Rehabil*, 95(3 SUPPL), S278-S285.
- Charrette, A. L., Lorenz, L. S., Fong, J., O'Neil-Pirozzi, T. M., Lamson, K., Demore-Taber, M., & Lilley, R. (2016). Pilot study of intensive exercise on endurance, advanced mobility and gait speed in adults with chronic severe acquired brain injury. *Brain Inj*, 1-7.
- Chow, J. W., Yablon, S. A., & Stokic, D. S. (2015). Effect of intrathecal baclofen bolus injection on ankle muscle activation during gait in patients with acquired brain injury. *Neurorehabil Neural Repair*, 29(2), 163-173.
- Ciuffreda, K. J., Han, Y., Kapoor, N., & Ficarra, A. P. (2006). Oculomotor rehabilitation for reading in acquired brain injury. *NeuroRehabilitation*, 21(1), 9-21.
- Clark, R. A., Williams, G., Fini, N., Moore, L., & Bryant, A. L. (2012). Coordination of dynamic balance during gait training in people with acquired brain injury. *Arch Phys Med Rehabil*, 93(4), 636-640.
- Clemenzi, A., Formisano, R., Matteis, M., Gallinacci, L., Cochi, G., Savina, P., & Cicinelli, P. (2012). Care management of spasticity with botulinum toxin-A in patients with severe acquired brain injury: A 1-year follow-up prospective study. *Brain Injury*, 26(7-8), 979-983.
- Conrad, J. S., Mitchell, G. L., & Kulp, M. T. (2016). Vision Therapy for Binocular Dysfunction Post Brain Injury. *Optom Vis Sci*.
- Costigan, M., Scholz, J., & Woolf, C. J. (2009). Neuropathic pain: a maladaptive response of the nervous system to damage. *Annu Rev Neurosci*, 32, 1-32.
- Couch, J. R., & Bearss, C. (2001). Chronic daily headache in the posttrauma syndrome: relation to extent of head injury. *Headache*, 41(6), 559-564.
- Cuthbert, J. P., Staniszewski, K., Hays, K., Gerber, D., Natale, A., & O'Dell, D. (2014). Virtual reality-based therapy for the treatment of balance deficits in patients receiving inpatient rehabilitation for traumatic brain injury. *Brain Injury*, 28(2), 181-188.
- D'Arcy, Y. (2011). *Compact Clinical Guide to Chronic Pain Management: An Evidence-Based Approach for Nurses*: Spencer Publishing Company.
- Damiano, D., Zampieri, C., Ge, J., Acevedo, A., & Durney, J. (2016). Effects of a rapid-resisted elliptical training program on motor, cognitive and neurobehavioral functioning in adults with chronic traumatic brain injury. *Experimental Brain Research*, 234(8), 2245-2252.
- Dario, A., Di Stefano, M. G., Grossi, A., Casagrande, F., & Bono, G. (2002). Long-term intrathecal Baclofen infusion in supraspinal spasticity of adulthood. *Acta Neurol Scand*, 105(2), 83-87.
- Dault, M. C., & Dugas, C. (2002). Evaluation of a specific balance and coordination programme for individuals with a traumatic brain injury. *Brain Inj*, 16(3), 231-244.

- DelleMijn, P. (1999). Are opioids effective in relieving neuropathic pain? *Pain*, 80(3), 453-462.
- Dettmers, C., Teske, U., Hamzei, F., Uswatte, G., Taub, E., & Weiller, C. (2005). Distributed form of constraint-induced movement therapy improves functional outcome and quality of life after stroke. *Arch Phys Med Rehabil*, 86(2), 204-209.
- Dharmshaktu, P., Tayal, V., & Kalra, B. S. (2012). Efficacy of antidepressants as analgesics: a review. *J Clin Pharmacol*, 52(1), 6-17.
- Dickinson, K., Bunn, F., Wentz, R., Edwards, P., & Roberts, I. (2000). Size and quality of randomised controlled trials in head injury: review of published studies. *Bmj*, 320(7245), 1308-1311.
- Doble, J. E., Feinberg, D. L., Rosner, M. S., & Rosner, A. J. (2010). Identification of binocular vision dysfunction (vertical heterophoria) in traumatic brain injury patients and effects of individualized prismatic spectacle lenses in the treatment of postconcussive symptoms: a retrospective analysis. *Pm r*, 2(4), 244-253.
- Dobscha, S. K., Clark, M. E., Morasco, B. J., Freeman, M., Campbell, R., & Helfand, M. (2009). Systematic review of the literature on pain in patients with polytrauma including traumatic brain injury. *Pain Med*, 10(7), 1200-1217.
- Driver, S., Rees, K., O'Connor, J., & Lox, C. (2006). Aquatics, health-promoting self-care behaviours and adults with brain injuries. *Brain Inj*, 20(2), 133-141.
- Dworkin, R. H., Backonja, M., Rowbotham, M. C., Allen, R. R., Argoff, C. R., Bennett, G. J., Bushnell, M. C., Farrar, J. T., Galer, B. S., Haythornthwaite, J. A., Hewitt, D. J., Loeser, J. D., Max, M. B., Saltarelli, M., Schmader, K. E., Stein, C., Thompson, D., Turk, D. C., Wallace, M. S., Watkins, L. R., & Weinstein, S. M. (2003). Advances in neuropathic pain: diagnosis, mechanisms, and treatment recommendations. *Arch Neurol*, 60(11), 1524-1534.
- Elkind, A. H. (1989). Headache and head trauma. *Clin J Pain*, 5(1), 77-87.
- Ersek, M., Cherrier, M. M., Overman, S. S., & Irving, G. A. (2004). The cognitive effects of opioids. *Pain Manag Nurs*, 5(2), 75-93.
- Esquenazi, A., Lee, S., Packel, A. T., & Braitman, L. (2013). A Randomized Comparative Study of Manually Assisted Versus Robotic-Assisted Body Weight Supported Treadmill Training in Persons With a Traumatic Brain Injury. *PM and R*, 5(4), 280-290.
- Ettinger, A. B., & Argoff, C. E. (2007). Use of antiepileptic drugs for nonepileptic conditions: psychiatric disorders and chronic pain. *Neurotherapeutics*, 4(1), 75-83.
- Farrar, J. T., Young, J. P., Jr., LaMoreaux, L., Werth, J. L., & Poole, R. M. (2001). Clinical importance of changes in chronic pain intensity measured on an 11-point numerical pain rating scale. *Pain*, 94(2), 149-158.
- Finnerup, N. B., Otto, M., McQuay, H. J., Jensen, T. S., & Sindrup, S. H. (2005). Algorithm for neuropathic pain treatment: an evidence based proposal. *Pain*, 118(3), 289-305.
- Foo, J., Paterson, K., Williams, G., & Clark, R. (2013). Low-cost evaluation and real-time feedback of static and dynamic weight bearing asymmetry in patients undergoing in-patient physiotherapy rehabilitation for neurological conditions. *Journal of NeuroEngineering and Rehabilitation*, 10(1).
- Franceschi, F., Marini, M., Ursella, S., Carbone, L., Candelli, M., Pignataro, G., Gabrielli, M., Santarelli, L., Ojetti, V., Giupponi, B., Fiore, V., Barbaro, F., De Marco, G., Buccelletti, F.,

- Mancini, F., Roccarina, D., Gigante, G., Gasbarrini, A., Gasbarrini, G., & Silveri, N. G. (2008). Use of oxycodone in polytrauma patients: the "Gemelli" experience. *Eur Rev Med Pharmacol Sci*, 12(2), 123-126.
- Francisco, G. E., Hu, M. M., Boake, C., & Ivanhoe, C. B. (2005). Efficacy of early use of intrathecal baclofen therapy for treating spastic hypertonia due to acquired brain injury. *Brain Inj*, 19(5), 359-364.
- Francois, B., Vacher, P., Roustan, J., Salle, J. Y., Vidal, J., Moreau, J. J., & Vignon, P. (2001). Intrathecal baclofen after traumatic brain injury: early treatment using a new technique to prevent spasticity. *J Trauma*, 50(1), 158-161.
- Gallagher, R., Drance, E., & Higginbotham, S. (2006). Finding the person behind the pain: chronic pain management in a patient with traumatic brain injury. *J Am Med Dir Assoc*, 7(7), 432-434.
- Garland, D. E., Lilling, M., & Keenan, M. A. (1984). Percutaneous phenol blocks to motor points of spastic forearm muscles in head-injured adults. *Arch Phys Med Rehabil*, 65(5), 243-245.
- Gellman, H., Keenan, M. A. E., & Botte, M. J. (1996). Recognition and Management of Upper Extremity Pain Syndromes in the Patient with Brain Injury. *The Journal of Head Trauma Rehabilitation*, 11(4), 23-30.
- Gironda, R. J., Clark, M. E., Ruff, R. L., Chait, S., Craine, M., Walker, R., & Scholten, J. (2009). Traumatic brain injury, polytrauma, and pain: challenges and treatment strategies for the polytrauma rehabilitation. *Rehabil Psychol*, 54(3), 247-258.
- Godbout, A. (1997). Structured habituation training for movement provoked vertigo after severe traumatic brain injury: a single-case experiment. *Brain Inj*, 11(9), 629-641.
- Gordon, D. B., & Love, G. (2004). Pharmacologic management of neuropathic pain. *Pain Manag Nurs*, 5(4 Suppl 1), 19-33.
- Gordon, W. A., Sliwinski, M., Echo, J., McLoughlin, M., Sheerer, M. S., & Meili, T. E. (1998). The benefits of exercise in individuals with traumatic brain injury: a retrospective study. *J Head Trauma Rehabil*, 13(4), 58-67.
- Gormley, M. E., Jr., O'Brien, C. F., & Yablon, S. A. (1997). A clinical overview of treatment decisions in the management of spasticity. *Muscle Nerve Suppl*, 6, S14-20.
- Gould, H. J. I. (2007). Pain Defined Understanding Pain: What it is, Why it happens and How it's managed. In S. LM (Ed.), *American Academy of Neurology* (1 ed., pp. 1-8).
- Gracies, Elovic, E., McGuire, J., & Simpson, D. M. (1997a). Traditional pharmacological treatments for spasticity. Part I: Local treatments. *Muscle Nerve Suppl*, 6, S61-91.
- Gracies, Nance, P., Elovic, E., McGuire, J., & Simpson, D. M. (1997b). Traditional pharmacological treatments for spasticity. Part II: General and regional treatments. *Muscle Nerve Suppl*, 6, S92-120.
- Greenwell, G. T. (2012). Medical marijuana use for chronic pain: risks and benefits. *J Pain Palliat Care Pharmacother*, 26(1), 68-69.
- Gregory, C. M., & Bickel, C. S. (2005). Recruitment patterns in human skeletal muscle during electrical stimulation. *Phys Ther*, 85(4), 358-364.



- Grissom, S. P., & Blanton, S. (2001). Treatment of upper motoneuron plantarflexion contractures by using an adjustable ankle-foot orthosis. *Arch Phys Med Rehabil*, 82(2), 270-273.
- Grotta, J. C., Noser, E. A., Ro, T., Boake, C., Levin, H., Aronowski, J., & Schallert, T. (2004). Constraint-induced movement therapy. *Stroke*, 35(11 Suppl 1), 2699-2701.
- Guay, D. R. (2003). Oxcarbazepine, topiramate, zonisamide, and levetiracetam: potential use in neuropathic pain. *Am J Geriatr Pharmacother*, 1(1), 18-37.
- Guindon, J., Walczak, J. S., & Beaulieu, P. (2007). Recent advances in the pharmacological management of pain. *Drugs*, 67(15), 2121-2133.
- Gurr, B., & Coetzer, B. R. (2005). The effectiveness of cognitive-behavioural therapy for post-traumatic headaches. *Brain Inj*, 19(7), 481-491.
- Gurr, B., & Moffat, N. (2001). Psychological consequences of vertigo and the effectiveness of vestibular rehabilitation for brain injury patients. *Brain Inj*, 15(5), 387-400.
- Haanpaa, M. L., Gourlay, G. K., Kent, J. L., Miaskowski, C., Raja, S. N., Schmader, K. E., & Wells, C. D. (2010). Treatment considerations for patients with neuropathic pain and other medical comorbidities. *Mayo Clin Proc*, 85(3 Suppl), S15-25.
- Halstead, L. S., Seager, S. W., Houston, J. M., Whitesell, K., Dennis, M., & Nance, P. W. (1993). Relief of spasticity in SCI men and women using rectal probe electrostimulation. *Paraplegia*, 31(11), 715-721.
- Ham, L. P., & Packard, R. C. (1996). A retrospective, follow-up study of biofeedback-assisted relaxation therapy in patients with posttraumatic headache. *Biofeedback Self Regul*, 21(2), 93-104.
- Hassett, L. M., Moseley, A. M., Tate, R. L., Harmer, A. R., Fairbairn, T. J., & Leung, J. (2009). Efficacy of a fitness centre-based exercise programme compared with a home-based exercise programme in traumatic brain injury: a randomized controlled trial. *J Rehabil Med*, 41(4), 247-255.
- Hassett, L. M., Moseley, A. M., Whiteside, B., Barry, S., & Jones, T. (2012). Circuit class therapy can provide a fitness training stimulus for adults with severe traumatic brain injury: a randomised trial within an observational study. *J Physiother*, 58(2), 105-112.
- Hassett, L. M., Tate, R. L., Moseley, A. M., & Gillett, L. E. (2011). Injury severity, age and pre-injury exercise history predict adherence to a home-based exercise programme in adults with traumatic brain injury. *Brain Inj*, 25(7-8), 698-706.
- Hawker, G. A., Mian, S., Kendzerska, T., & French, M. (2011). Measures of adult pain: Visual Analog Scale for Pain (VAS Pain), Numeric Rating Scale for Pain (NRS Pain), McGill Pain Questionnaire (MPQ), Short-Form McGill Pain Questionnaire (SF-MPQ), Chronic Pain Grade Scale (CPGS), Short Form-36 Bodily Pain Scale (SF-36 BPS), and Measure of Intermittent and Constant Osteoarthritis Pain (ICOAP). *Arthritis Care Res (Hoboken)*, 63 Suppl 11, S240-252.
- Hillier, S. L., Sharpe, M. H., & Metzger, J. (1997). Outcomes 5 years post-traumatic brain injury (with further reference to neurophysical impairment and disability). *Brain Inj*, 11(9), 661-675.
- Hirose, T., Shiozaki, T., Shimizu, K., Mouri, T., Noguchi, K., Ohnishi, M., & Shimazu, T. (2013). The effect of electrical muscle stimulation on the prevention of disuse muscle atrophy in



- patients with consciousness disturbance in the intensive care unit. *Journal of Critical Care*, 28(4), 536.e531-537.
- Hoarau, X., Richer, E., Dehail, P., & Cuny, E. (2012a). A 10-year follow-up study of patients with severe traumatic brain injury and dysautonomia treated with intrathecal baclofen therapy. *Brain Injury*, 26(7-8), 927-940.
- Hoarau, X., Richer, E., Dehail, P., & Cuny, E. (2012b). Comparison of long-term outcomes of patients with severe traumatic or hypoxic brain injuries treated with intrathecal baclofen therapy for dysautonomia. *Brain Injury*, 26(12), 1451-1463.
- Hoffman, J. M., Bell, K. R., Powell, J. M., Behr, J., Dunn, E. C., Dikmen, S., & Bombardier, C. H. (2010). A randomized controlled trial of exercise to improve mood after traumatic brain injury. *Pm r*, 2(10), 911-919.
- Hoffman, J. M., Lucas, S., Dikmen, S., Braden, C. A., Brown, A. W., Brunner, R., Diaz-Arrastia, R., Walker, W. C., Watanabe, T. K., & Bell, K. R. (2011). Natural history of headache after traumatic brain injury. *J Neurotrauma*, 28(9), 1719-1725.
- Hoffman, J. M., Pagulayan, K. F., Zawaideh, N., Dikmen, S., Temkin, N., & Bell, K. R. (2007). Understanding pain after traumatic brain injury: impact on community participation. *Am J Phys Med Rehabil*, 86(12), 962-969.
- Horn, T. S., Yablon, S. A., Chow, J. W., Lee, J. E., & Stokic, D. S. (2010). Effect of intrathecal baclofen bolus injection on lower extremity joint range of motion during gait in patients with acquired brain injury. *Arch Phys Med Rehabil*, 91(1), 30-34.
- Horn, T. S., Yablon, S. A., & Stokic, D. S. (2005). Effect of intrathecal baclofen bolus injection on temporospatial gait characteristics in patients with acquired brain injury. *Arch Phys Med Rehabil*, 86(6), 1127-1133.
- International Association for the Study of Pain. (1994). Part III Pain Terms: A Current List with Definitions and Notes on Usage. In H. Merskey & N. Bogduk (Eds.), *Classification of Chronic Pain: Descriptions of Chronic Pain Syndromes and Definitions of Pain Terms* (2 ed.). Seattle: IASP Press.
- Intiso, D., Simone, V., Di Rienzo, F., Iarossi, A., Pazienza, L., Santamato, A., Maruzzi, G., & Basciani, M. (2014). High doses of a new botulinum toxin type A (NT-201) in adult patients with severe spasticity following brain injury and cerebral palsy. *NeuroRehabilitation*, 34(3), 515-522.
- Ivanhoe, C. B., & Hartman, E. T. (2004). Clinical caveats on medical assessment and treatment of pain after TBI. *J Head Trauma Rehabil*, 19(1), 29-39.
- Ivanhoe, C. B., & Parrilla, Z. M. (2002). Pain Management in Traumatic Brain Injury. In G. M. Monga TN (Ed.), *Pain Management in Rehabilitation* (pp. 101-125). New York, NY: Demos Medical Publishing.
- Jacobson, G. P., Ramadan, N. M., Aggarwal, S. K., & Newman, C. W. (1994). The Henry Ford Hospital Headache Disability Inventory (HDI). *Neurology*, 44(5), 837-842.
- Jankovic, J., & Brin, M. F. (1991). Therapeutic uses of botulinum toxin. *N Engl J Med*, 324(17), 1186-1194.
- Jensen, O. K., Nielsen, F. F., & Vosmar, L. (1990). An open study comparing manual therapy with the use of cold packs in the treatment of post-traumatic headache. *Cephalalgia*, 10(5), 241-250.

- Kasten, E., Poggel, D. A., & Sabel, B. A. (2000). Computer-based training of stimulus detection improves color and simple pattern recognition in the defective field of hemianopic subjects. *J Cogn Neurosci*, 12(6), 1001-1012.
- Kasten, E., Wust, S., Behrens-Baumann, W., & Sabel, B. A. (1998). Computer-based training for the treatment of partial blindness. *Nat Med*, 4(9), 1083-1087.
- Katz, & Campagnolo, D. I. (1993). Pharmacological management of spasticity. In K. RT (Ed.), *Spasticity: State of the Art Reviews; Physical Medicine and Rehabilitation* (Vol. 8, pp. 473-480). Philadelphia: Hanley and Belfus.
- Katz, Dewald, J. P., & Schmit, B. D. (2000). Spasticity. In B. RL (Ed.), *Physical medicine and rehabilitation*. (pp. 592-615). Toronto: Saunders Company.
- Keenan, M. A., Tomas, E. S., Stone, L., & Gersten, L. M. (1990). Percutaneous phenol block of the musculocutaneous nerve to control elbow flexor spasticity. *J Hand Surg Am*, 15(2), 340-346.
- Khan, M. I., Walsh, D., & Brito-Dellan, N. (2011). Opioid and adjuvant analgesics: compared and contrasted. *Am J Hosp Palliat Care*, 28(5), 378-383.
- Kriz, G., Hermsdorfer, J., Marquardt, C., & Mai, N. (1995). Feedback-based training of grip force control in patients with brain damage. *Arch Phys Med Rehabil*, 76(7), 653-659.
- Lahz, S., & Bryant, R. A. (1996). Incidence of chronic pain following traumatic brain injury. *Arch Phys Med Rehabil*, 77(9), 889-891.
- Lainez, M. J., & Pesquera, B. L. (2011). Headache after trauma: physiological considerations. *Curr Pain Headache Rep*, 15(6), 467-473.
- Lance, J. W. (1980). The control of muscle tone, reflexes, and movement: Robert Wartenberg Lecture. *Neurology*, 30(12), 1303-1313.
- Lannin, N. A., Horsley, S. A., Herbert, R., McCluskey, A., & Cusick, A. (2003). Splinting the hand in the functional position after brain impairment: a randomized, controlled trial. *Arch Phys Med Rehabil*, 84(2), 297-302.
- Leung, J., Harvey, L. A., Moseley, A. M., Whiteside, B., Simpson, M., & Stroud, K. (2014). Standing with electrical stimulation and splinting is no better than standing alone for management of ankle plantarflexion contractures in people with traumatic brain injury: a randomised trial. *J Physiother*, 60(4), 201-208.
- Lew, H. L., Lin, P. H., Fuh, J. L., Wang, S. J., Clark, D. J., & Walker, W. C. (2006). Characteristics and treatment of headache after traumatic brain injury: a focused review. *Am J Phys Med Rehabil*, 85(7), 619-627.
- Linacre, J. M., Heinemann, A. W., Wright, B. D., Granger, C. V., & Hamilton, B. B. (1994). The structure and stability of the Functional Independence Measure. *Arch Phys Med Rehabil*, 75(2), 127-132.
- Lorentzen, J., Nielsen, D., Holm, K., Baagøe, S., Grey, M. J., & Nielsen, J. B. (2012). Neural tension technique is no different from random passive movements in reducing spasticity in patients with traumatic brain injury. *Disabil Rehabil*, 34(23), 1978-1985.
- Lucas, S. (2011). Headache management in concussion and mild traumatic brain injury. *Pm r*, 3(10 Suppl 2), S406-412.

- Margetis, K., Korfiatis, S. I., Gatzonis, S., Boutos, N., Stranjalis, G., Boviatsis, E., & Sakas, D. E. (2014). Intrathecal baclofen associated with improvement of consciousness disorders in spasticity patients. *Neuromodulation*, 17(7), 699-704: discussion 704.
- Martelli, M. F., Nicholson, K., & Zasler N.D. (2012). Psychological Assessment and Management of Post Traumatic Pain. In N. D. Zasler, Katz, D.I. & Zafonte, R.D (Ed.), *Brain Injury Medicine: Principles and Practice* (2 ed., pp. 974-987).
- Mayer. (1997). Clinicophysiology concepts of spasticity and motor dysfunction in adults with an upper motoneuron lesion. *Muscle Nerve Suppl*, 6, S1-13.
- Mayer, Esquenazi, A., & Childers, M. K. (1997). Common patterns of clinical motor dysfunction. *Muscle Nerve Suppl*, 6, S21-35.
- Mayer, Esquenazi, A., & Keenan, M. A. (1996). Analysis and management of spasticity, contracture and impaired motor control. In Z. N. Horn LJ (Ed.), *Medical rehabilitation of traumatic brain injury* (pp. 411-458). Toronto: Mosby.
- Mayer, N. H., Whyte, J., Wannstedt, G., & Ellis, C. A. (2008). Comparative impact of 2 botulinum toxin injection techniques for elbow flexor hypertonia. *Arch Phys Med Rehabil*, 89(5), 982-987.
- McDowell, I. (2006). *Measuring Health. A Guide to Rating Scales and Questionnaires*. New York: Oxford University Press.
- Medina, J. L. (1992). Efficacy of an individualized outpatient program in the treatment of chronic post-traumatic headache. *Headache*, 32(4), 180-183.
- Meythaler, Guin-Renfroe, S., & Hadley, M. N. (1999). Continuously infused intrathecal baclofen for spastic/dystonic hemiplegia: a preliminary report. *Am J Phys Med Rehabil*, 78(3), 247-254.
- Meythaler, J. M., Clayton, W., Davis, L. K., Guin-Renfroe, S., & Brunner, R. C. (2004). Orally delivered baclofen to control spastic hypertonia in acquired brain injury. *J Head Trauma Rehabil*, 19(2), 101-108.
- Meythaler, J. M., DeVivo, M. J., & Hadley, M. (1996). Prospective study on the use of bolus intrathecal baclofen for spastic hypertonia due to acquired brain injury. *Arch Phys Med Rehabil*, 77(5), 461-466.
- Meythaler, J. M., Guin-Renfroe, S., Grabb, P., & Hadley, M. N. (1999). Long-term continuously infused intrathecal baclofen for spastic-dystonic hypertonia in traumatic brain injury: 1-year experience. *Arch Phys Med Rehabil*, 80(1), 13-19.
- Meythaler, J. M., Guin-Renfroe, S., Johnson, A., & Brunner, R. M. (2001). Prospective assessment of tizanidine for spasticity due to acquired brain injury. *Arch Phys Med Rehabil*, 82(9), 1155-1163.
- Meythaler, J. M., McCary, A., & Hadley, M. N. (1997). Prospective assessment of continuous intrathecal infusion of baclofen for spasticity caused by acquired brain injury: a preliminary report. *J Neurosurg*, 87(3), 415-419.
- Mortenson, P. A., & Eng, J. J. (2003). The use of casts in the management of joint mobility and hypertonia following brain injury in adults: a systematic review. *Phys Ther*, 83(7), 648-658.

- Morton, R. (2004). Visual Dysfunction Following Traumatic Brain Injury. In A. MJ (Ed.), *Traumatic Brain Injury - Rehabilitative Treatment and Case Management*. (pp. 183-207). Boca Raton, Florida: CRC Press;.
- Moseley, A. M. (1997). The effect of casting combined with stretching on passive ankle dorsiflexion in adults with traumatic head injuries. *Phys Ther*, 77(3), 240-247; discussion 248-259.
- Moseley, A. M., Hassett, L. M., Leung, J., Clare, J. S., Herbert, R. D., & Harvey, L. A. (2008). Serial casting versus positioning for the treatment of elbow contractures in adults with traumatic brain injury: a randomized controlled trial. *Clin Rehabil*, 22(5), 406-417.
- Moseley, A. M., Herbert, R. D., Sherrington, C., & Maher, C. G. (2002). Evidence for physiotherapy practice: a survey of the Physiotherapy Evidence Database (PEDro). *Aust J Physiother*, 48(1), 43-49.
- Motin, M., Keren, O., Groswasser, Z., & Gordon, C. R. (2005). Benign paroxysmal positional vertigo as the cause of dizziness in patients after severe traumatic brain injury: diagnosis and treatment. *Brain Inj*, 19(9), 693-697.
- Mullally, W. J., Hall, K., & Goldstein, R. (2009). Efficacy of biofeedback in the treatment of migraine and tension type headaches. *Pain Physician*, 12(6), 1005-1011.
- Mumford, N., Duckworth, J., Thomas, P. R., Shum, D., Williams, G., & Wilson, P. H. (2012). Upper-limb virtual rehabilitation for traumatic brain injury: a preliminary within-group evaluation of the elements system. *Brain Injury*, 26(2), 166-176.
- Naguib, M. B., & Madian, Y. T. (2014). Betahistine Dihydrochloride With and Without Early Vestibular Rehabilitation for the Management of Patients With Balance Disorders Following Head Trauma: A Preliminary Randomized Clinical Trial. *Journal of Chiropractic Medicine*, 13(1), 14-20.
- Nasreddine, W., & Beydoun, A. (2007). Oxcarbazepine in neuropathic pain. *Expert Opin Investig Drugs*, 16(10), 1615-1625.
- Neistadt, M. E. (1994). The effects of different treatment activities on functional fine motor coordination in adults with brain injury. *Am J Occup Ther*, 48(10), 877-882.
- Nicholson, B. D. (2004). Evaluation and treatment of central pain syndromes. *Neurology*, 62(5 Suppl 2), S30-36.
- O'Connor, A. B., & Dworkin, R. H. (2009). Treatment of neuropathic pain: an overview of recent guidelines. *Am J Med*, 122(10 Suppl), S22-32.
- Ofek, H., & Defrin, R. (2007). The characteristics of chronic central pain after traumatic brain injury. *Pain*, 131(3), 330-340.
- Packard, R. C. (2000). Treatment of chronic daily posttraumatic headache with divalproex sodium. *Headache*, 40(9), 736-739.
- Padula, W. V., Argyris, S., & Ray, J. (1994). Visual evoked potentials (VEP) evaluating treatment for post-trauma vision syndrome (PTVS) in patients with traumatic brain injuries (TBI). *Brain Inj*, 8(2), 125-133.
- Page, S., & Levine, P. (2003). Forced use after TBI: promoting plasticity and function through practice. *Brain Inj*, 17(8), 675-684.
- Pangarkar, S., & Lee, P. C. (2011). Conservative treatment for neck pain: medications, physical therapy, and exercise. *Phys Med Rehabil Clin N Am*, 22(3), 503-520, ix.

- Peirone, E., Gorla, P. F., & Anselmino, A. (2014). A dual-task home-based rehabilitation programme for improving balance control in patients with acquired brain injury: a single-blind, randomized controlled pilot study. *Clinical Rehabilitation*, 28(4), 329-338.
- Peters, D. M., Jain, S., Liuzzo, D. M., Middleton, A., Greene, J., Blanck, E., Sun, S., Raman, R., & Fritz, S. L. (2014). Individuals with chronic traumatic brain injury improve walking speed and mobility with intensive mobility training. *Arch Phys Med Rehabil*, 95(8), 1454-1460.
- Pohl, M., Ruckriem, S., Mehrholz, J., Ritschel, C., Strik, H., & Pause, M. R. (2002). Effectiveness of serial casting in patients with severe cerebral spasticity: a comparison study. *Arch Phys Med Rehabil*, 83(6), 784-790.
- Posteraro, F., Calandriello, B., Galli, R., Logi, F., Iardella, L., & Bordi, L. (2013). Timing of intrathecal baclofen therapy in persons with acquired brain injury: influence on outcome. *Brain Inj*, 27(13-14), 1671-1675.
- Pringsheim, T., Davenport, W., Mackie, G., Worthington, I., Aube, M., Christie, S. N., Gladstone, J., & Becker, W. J. (2012). Canadian Headache Society guideline for migraine prophylaxis. *Can J Neurol Sci*, 39(2 Suppl 2), S1-S9.
- Riggs, R. V., Andrews, K., Roberts, P., & Gilewski, M. (2007). Visual deficit interventions in adult stroke and brain injury: a systematic review. *Am J Phys Med Rehabil*, 86(10), 853-860.
- Roth, A. D., & Pilling, S. (2008). Using an Evidence-Based Methodology to Identify the Competences Required to Deliver Effective Cognitive and Behavioural Therapy for Depression and Anxiety Disorders. *Behavioural and Cognitive Psychotherapy*, 36(2), 129-147.
- Schafer, A. Y., & Ustinova, K. I. (2013). Does use of a virtual environment change reaching while standing in patients with traumatic brain injury? *Journal of NeuroEngineering and Rehabilitation*, 10(1).
- Scherer, M. R., & Schubert, M. C. (2009). Traumatic brain injury and vestibular pathology as a comorbidity after blast exposure. *Phys Ther*, 89(9), 980-992.
- Seib, T. P., Price, R., Reyes, M. R., & Lehmann, J. F. (1994). The quantitative measurement of spasticity: effect of cutaneous electrical stimulation. *Arch Phys Med Rehabil*, 75(7), 746-750.
- Seif-Naraghi, A. H., & Herman, R. M. (1999). A novel method for locomotion training. *J Head Trauma Rehabil*, 14(2), 146-162.
- Seifert, T. D., & Evans, R. W. (2010). Posttraumatic headache: a review. *Curr Pain Headache Rep*, 14(4), 292-298.
- Shaw, S. E., Morris, D. M., Uswatte, G., McKay, S., Meythaler, J. M., & Taub, E. (2005). Constraint-induced movement therapy for recovery of upper-limb function following traumatic brain injury. *J Rehabil Res Dev*, 42(6), 769-778.
- Shepard, N. T., & Telian, S. A. (1995). Programmatic vestibular rehabilitation. *Otolaryngol Head Neck Surg*, 112(1), 173-182.
- Sherman, K. B., Goldberg, M., & Bell, K. R. (2006). Traumatic brain injury and pain. *Phys Med Rehabil Clin N Am*, 17(2), 473-490, viii.
- Sietsema, J. M., Nelson, D. L., Mulder, R. M., Mervau-Scheidel, D., & White, B. E. (1993). The use of a game to promote arm reach in persons with traumatic brain injury. *Am J Occup Ther*, 47(1), 19-24.



- Singer, Jegasothy, G. M., Singer, K. P., & Allison, G. T. (2003). Evaluation of serial casting to correct equinovarus deformity of the ankle after acquired brain injury in adults. *Arch Phys Med Rehabil*, 84(4), 483-491.
- Singer, B. J., Singer, K. P., & Allison, G. T. (2003). Evaluation of extensibility, passive torque and stretch reflex responses in triceps surae muscles following serial casting to correct spastic equinovarus deformity. *Brain Inj*, 17(4), 309-324.
- Stokic, D. S., Yablon, S. A., & Hayes, A. (2005). Comparison of clinical and neurophysiologic responses to intrathecal baclofen bolus administration in moderate-to-severe spasticity after acquired brain injury. *Arch Phys Med Rehabil*, 86(9), 1801-1806.
- Sullivan-Singh, S. J., Sawyer, K., Ehde, D. M., Bell, K. R., Temkin, N., Dikmen, S., Williams, R. M., & Hoffman, J. M. (2014). Comorbidity of pain and depression among persons with traumatic brain injury. *Arch Phys Med Rehabil*, 95(6), 1100-1105.
- Tang, V., Warden, J., Cullen, N., & Rutledge, E. (2007). Topiramate in traumatic brain injury: adverse effects on cognitive function. *J Head Trauma Rehabil*, 22(6), 409-410.
- Tatrow, K., Blanchard, E. B., & Silverman, D. J. (2003). Posttraumatic headache: an exploratory treatment study. *Appl Psychophysiol Biofeedback*, 28(4), 267-278.
- Theeler, B. J., & Erickson, J. C. (2012). Posttraumatic headache in military personnel and veterans of the iraq and afghanistan conflicts. *Curr Treat Options Neurol*, 14(1), 36-49.
- Thibaut, A., Deltombe, T., Wannez, S., Gosseries, O., Ziegler, E., Dieni, C., Deroy, M., & Laureys, S. (2015). Impact of soft splints on upper limb spasticity in chronic patients with disorders of consciousness: A randomized, single-blind, controlled trial. *Brain Inj*, 29(7-8), 830-836.
- Uomoto, J. M., & Esselman, P. C. (1993). Traumatic brain injury and chronic pain: differential types and rates by head injury severity. *Arch Phys Med Rehabil*, 74(1), 61-64.
- Ustinova, K. I., Chernikova, L. A., Dull, A., & Perkins, J. (2015). Physical therapy for correcting postural and coordination deficits in patients with mild-to-moderate traumatic brain injury. *Physiother Theory Pract*, 31(1), 1-7.
- Ustinova, K. I., Perkins, J., Leonard, W. A., & Hausbeck, C. J. (2014). Virtual reality game-based therapy for treatment of postural and co-ordination abnormalities secondary to TBI: A pilot study. *Brain Injury*, 28(4), 486-495.
- Verplancke, D., Snape, S., Salisbury, C. F., Jones, P. W., & Ward, A. B. (2005). A randomized controlled trial of botulinum toxin on lower limb spasticity following acute acquired severe brain injury. *Clin Rehabil*, 19(2), 117-125.
- Vranken, J. H., Dijkgraaf, M. G., Kruis, M. R., van der Vegt, M. H., Hollmann, M. W., & Heesen, M. (2008). Pregabalin in patients with central neuropathic pain: a randomized, double-blind, placebo-controlled trial of a flexible-dose regimen. *Pain*, 136(1-2), 150-157.
- Vranken, J. H., Hollmann, M. W., van der Vegt, M. H., Kruis, M. R., Heesen, M., Vos, K., Pijl, A. J., & Dijkgraaf, M. G. (2011). Duloxetine in patients with central neuropathic pain caused by spinal cord injury or stroke: a randomized, double-blind, placebo-controlled trial. *Pain*, 152(2), 267-273.
- Wang, Z. M., Law, J. H., King, N. K., Rajeswaran, D. K., Soh, S., Rao, J. P., Ng, W. H., & Chua, K. S. (2016). Treatment of severe, disabling spasticity with continuous intrathecal baclofen

- therapy following acquired brain injury: the experience of a tertiary institution in Singapore. *Singapore Med J*, 57(1), 8-12.
- Ware, M. A., Wang, T., Shapiro, S., Robinson, A., Ducruet, T., Huynh, T., Gamsa, A., Bennett, G. J., & Collet, J. P. (2010). Smoked cannabis for chronic neuropathic pain: a randomized controlled trial. *CMAJ*, 182(14), E694-701.
- Waszkielewicz, A. M., Gunia, A., Sloczynska, K., & Marona, H. (2011). Evaluation of anticonvulsants for possible use in neuropathic pain. *Curr Med Chem*, 18(28), 4344-4358.
- Watanabe, T. K., Bell, K. R., Walker, W. C., & Schomer, K. (2012). Systematic review of interventions for post-traumatic headache. *Pm r*, 4(2), 129-140.
- Wee, S. K. (2002). Vestibular rehabilitation for a patient with severe traumatic brain injury: a case study. *Physiotherapy Singapore*, 5, 59-65.
- Wetherell, J. L., Afari, N., Rutledge, T., Sorrell, J. T., Stoddard, J. A., Petkus, A. J., Solomon, B. C., Lehman, D. H., Liu, L., Lang, A. J., & Atkinson, J. H. (2011). A randomized, controlled trial of acceptance and commitment therapy and cognitive-behavioral therapy for chronic pain. *Pain*, 152(9), 2098-2107.
- Wiffen, P. J., Derry, S., & Moore, R. A. (2011). Lamotrigine for acute and chronic pain. *Cochrane Database Syst Rev*(2), Cd006044.
- Wilson, D. J., Powell, M., Gorham, J. L., & Childers, M. K. (2006). Ambulation training with and without partial weightbearing after traumatic brain injury: results of a randomized, controlled trial. *Am J Phys Med Rehabil*, 85(1), 68-74.
- Wong, V., Cheuk, D. K., Lee, S., & Chu, V. (2012). Accupuncture for acute management and rehabilitation of traumatic brain injury. *Eur J Phys Rehabil Med*, 48(1), 71-86.
- Yablon, S. A., Agana, B. T., Ivanhoe, C. B., & Boake, C. (1996). Botulinum toxin in severe upper extremity spasticity among patients with traumatic brain injury: an open-labeled trial. *Neurology*, 47(4), 939-944.
- Yarkony, G. M., & Sahgal, V. (1987). Contractures. A major complication of craniocerebral trauma. *Clin Orthop Relat Res*(219), 93-96.
- Young, W. B., Packard, R.C. & Ramadan. (2001). Headaches associated with head trauma. In S. D. Silverstein, Lipton, R.B. & Dalessio, D.J. (Ed.), *Wolfe's Headache and other Head Pain* (7 ed., pp. 325-348).
- Yungher, D., & Craelius, W. (2012). Improving fine motor function after brain injury using gesture recognition biofeedback. *Disability and Rehabilitation: Assistive Technology*, 7(6), 464-468.
- Zasler, N. D., Martelli, M. F., & Nicholson, K. (2011). Chronic Pain. In M. T. Silver JM, Yudofsky SC (Ed.), *Textbook of Traumatic Brain Injury* (pp. 375-396). Washington, DC: American Psychiatric Publishing, Inc.