ERABI
EVIDENCE-BASED REVIEW
of moderate to severe
ACQUIRED BRAIN INJURY

Clinical Guidebook

6. Motor and Sensory Dysfunction Following Acquired Brain Injury

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Motor and Sensory Dysfunction Following ABI

By the end of this chapter you should know:

- The motor and sensory dysfunctions that individuals should always be screened for.
- How to identify the classic presentation of upper motor neuron syndrome.
- Common assessments for evaluating motor deficits, sensory deficits, and pain following an ABI.
- Multiple management strategies for upper and lower extremities.
- Which interventions are supported by clinical practice guidelines.
- Non-pharmacological and pharmacological interventions for pain management.

6.1 Introduction to Motor and Sensory Deficits

This chapter will cover the common motor and sensory impairments following acquired brain injury (ABI), such as pain syndromes. Motor challenges following an ABI may be primary or secondary. Compared to mild brain injury, moderate to severe brain injuries may result in prolonged periods of immobility during recovery and contribute to motor deficits observed during rehabilitation. Within 4-6 weeks resulting immobility can have significant effects on the body, including up to 40% muscle loss, decreases in bone density, and joint contracture (Bell & Shenouda, 2013). Unlike motor deficits, vestibular deficits are rarely caused by secondary injury (Shepard et al., 2013), this is discussed further in section 6.2.2 (Clinical presentation of sensory deficits). Where relevant, strategies for the management of motor and sensory deficits will be discussed along with recommended interventions.

As the regions of the brain responsible for the control of motor and sensory responses (Figure 6.1) are located in the cortex there is a high association between traumatic brain injuries (TBIs) and motor and sensory deficits.

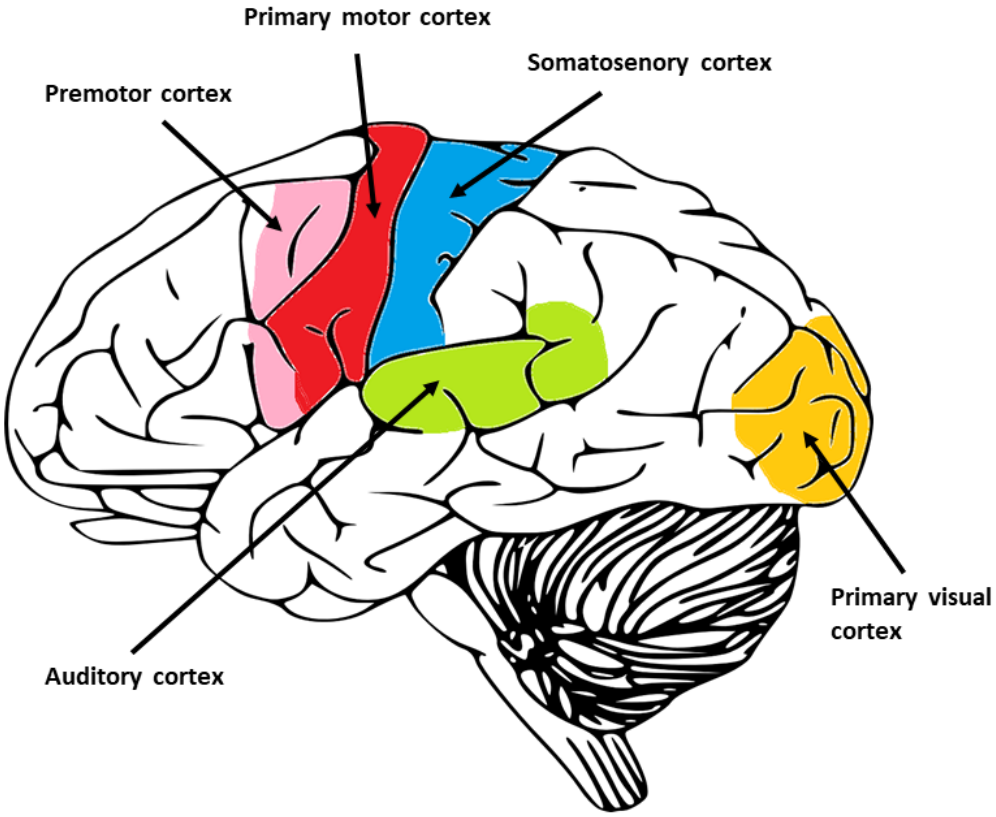

Figure 6.1 A sagittal view of the brain highlighting the areas of the cortex which are responsible for specific sensory and motor functions.

It is the physical domain that is typically 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 more common because both patients and family members are more likely to recognize and acknowledge physical impairments, in contrast to cognitive and behavioural impairments. Further, with amelioration of physical deficits, there is a decrease in burden of care of need for assistance for the patient within the home setting.

6.2 Clinical Presentation

6.2.1 Motor Dysfunction

Common motor deficits which may be observed following ABI include, upper motor neuron syndrome (UMNS) associated with spasticity, joint contractures, and muscle atrophy (Bell & Shenouda, 2013; Mayer et al., 1997). The primary cause of motor impairment and movement dysfunction post ABI is UMNS (Mayer, 1997). Common signs and symptoms associated with UMNS are presented in Table 6.1. UMNS occurs when motor neurons originating in the premotor or primary motor cortex of the brain (Figure 6.1) are damaged and unable to communicate with efferent motor neurons due to signal disruptions. Upper

motor neurons, by definition, originate in the brain and can connect synaptically to lower motor neurons and then muscle tissue via the spinal cord (Figure 6.2). One way to check for the presence of UMN lesions is to see if the Babinski sign/reflex in present (Figure 6.3) for lower extremity and the Hoffman reflex for the upper extremity. Common patterns of upper motor neuron deformity are presented in Table 6.2. These synergy patterns present after brain injury and can foster long term complications for patients including joint contracture, pain and can also contribute to skin breakdown. Intervention is often necessary early on to offset the effects of spasticity.

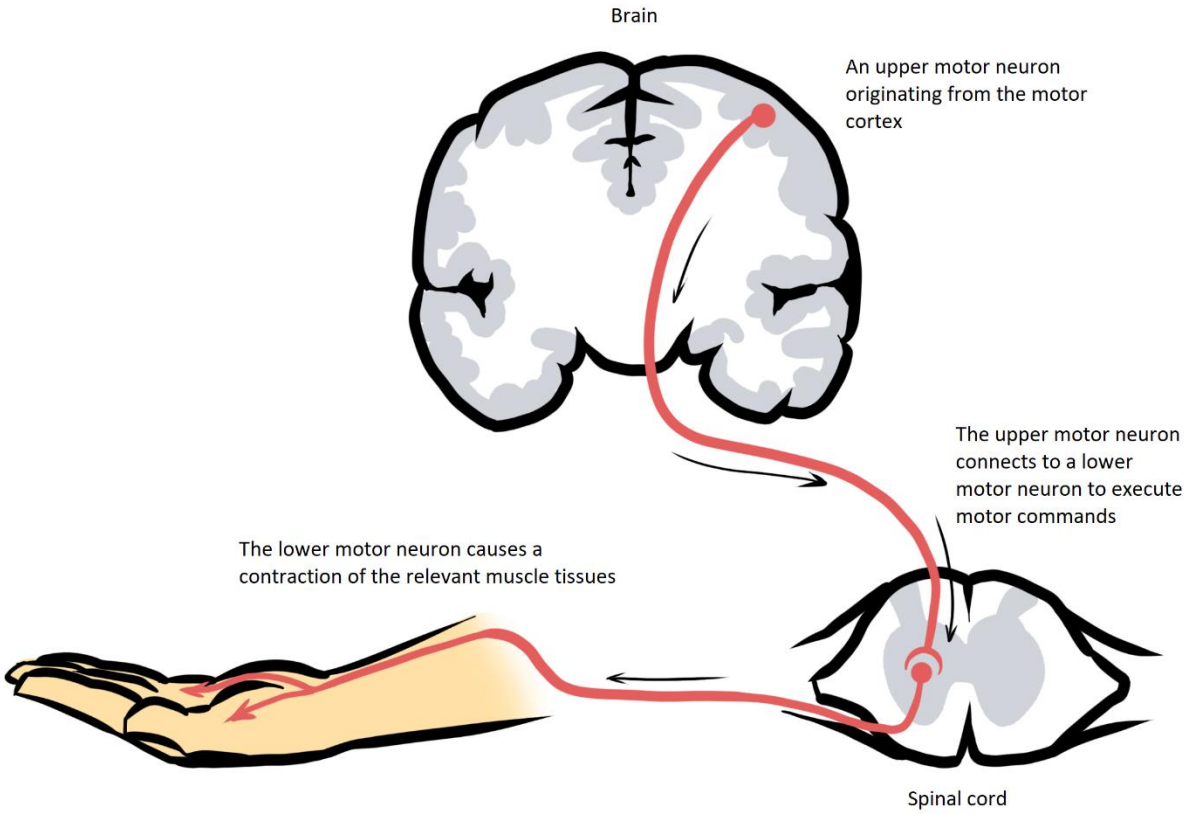

Figure 6.2 The pathway of upper motor neurons from the cortex to the muscles.

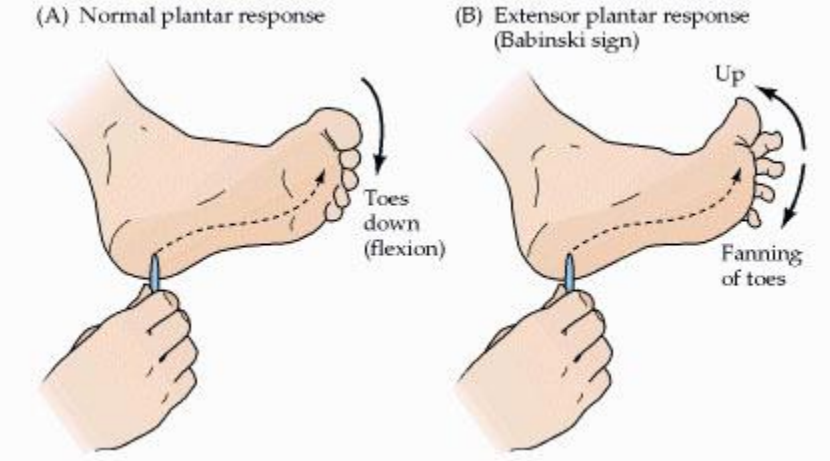

Figure 6.3 Testing for the Babinski Sign (abnormal) response and the presence of upper motor neuron lesions.

Table 6.1 Signs and symptoms associated with upper motor neuron deficits and lesions.

Signs of Upper Motor Neuron Lesions	Description
Spasticity	Spasticity is an inappropriate increase in the frequency and intensity of muscle contractions and can interfere with conscious movement and gait.
Hypertonicity	Hypertonicity is an increase in muscle tone and tension at rest resulting in reduced mobility in the affected limbs (Evans & Cameron, 2017).
Hyperreflexia	Hyperreflexia is defined by over-responsive and over-reactive reflexes, including twitching or spastic movements (Little et al., 1999).
Disuse atrophy	Disuse atrophy refers to the breakdown of muscle tissues and overall muscle wasting with disuse.
Muscle weakness	Muscle weakness is defined as a lack of muscle strength and neuromuscular fatigue whereby signal disruptions limit the activation of muscles.

Joint contractures are another area of motor deficits which can be caused by an ABI. Joint contractures are changes in the connective tissues (muscles, tendons, ligaments, etc.) that result in the loss of passive range of motion in the affected joints (Bell & Shenouda, 2013). In animal models it has been seen that the immobilization of limbs results in an increase in connective tissues to the point where adjacent muscle and collagen fibers become indistinguishable (Bell & Shenouda, 2013; Booth, 1978; Järvinen et al., 2002; Martti Kvist Timo Hurme Pekka Kannus Teppo JÃ¤rvinen Vesa-Matti Maunu Laszlo Jozsa Markku, 1995). A series of risk factors have been found to be associated with the development of joint contractures including: spasticity, weakness, prolonged immobility, edema, diabetes mellitus, and older age (Bell & Shenouda, 2013; Singer et al., 2004; Yarkony & Sahgal, 1987). Prevention of joint contractures can be done by treating other motor deficits in a timely and efficient manner.

Table 6.2 Common Patterns of Upper Motor Neuron Deformity (adapted from Mayer(2013)).

Upper Limb	Lower Limb
Abducted/internally rotated shoulder	Flexed hip
Flexed elbow	Scissoring thighs
Pronated forearm	Stiff knee
Bent wrist	Flexed knee
Clenched fist	Equinovarus foot with curl or claw toes
Thumb-in-palm	Valgus foot
Intrinsic plus hand	Hitchhiker hyperextended great toe

Muscle atrophy and weakness is more likely to be debilitating in the lower, weight-bearing limbs of the body following an ABI (Bell & Shenouda, 2013; LeBlanc et al., 1992). Starting as early as the fifth day of bed rest, an increase of nitrogen in the urine can be seen; this signals the occurrence of protein degradation and is an indicator of muscle atrophy (Deitrick et al., 1948; Goldspink et al., 1986; Thomason & Booth, 1989). Muscles which work against gravity (such as the knee extensors) are disproportionately affected by immobilization (Bell & Shenouda, 2013). Similar to joint contractures, prolonged periods of immobilization increase the extent of muscle atrophy, 30 days of bedrest has seen a decrease in up to 20% of knee extensor muscle bulk (Adams et al., 2003; Bell & Shenouda, 2013; Dudley et al., 1989), and significant reductions in muscle endurance as well (Tesch et al., 1991).

The predicted functional outcome of an individual by the clinical team will influence the intensity and frequency of treatment (Bonds et al., 2015). However, a recent study of critical care nurses, trauma/critical care physicians, and neurosurgeons found that there was low accuracy (25%) in predicting long term negative functional outcomes for individuals with moderate to severe brain injuries (Bonds et al., 2015). In non-specialized clinicians this value dropped to 16% (Bonds et al., 2015). Overall, clinicians overestimated the likelihood of long term bad outcomes.

6.2.2 Sensory Deficits

Sensory deficits following a traumatic brain injury can include touch (light touch, pain, position, vibration), visual, auditory, and vestibular challenges. Vision is a critical function which guides the movement attempted by the body, therefore when one's vision is compromised corresponding motor challenges may also arise.

Visual Deficits

Over 23 different visual problems have been reported following a moderate to severe traumatic brain injury, among them are visual acuity, photophobia, pupillary function, motion perception, reading ability, and visual hallucinations (Armstrong, 2018). Many individuals with a brain injury also tend to exhibit more than one visual complication post-injury (Armstrong, 2018; Master et al., 2016; Schlageter et al., 1993). Common visual deficits following an ABI are presented in Table 6.3.

Table 6.3 Common visual deficits following ABI.

Deficit	Description	References
Visual acuity	Usually referring to the clarity of one's vision. Visual acuity loss occurs in approximately 13% of individuals and can also be persistent. Individuals with blast exposure ABIs typically have poorer long-term visual acuity.	(Armstrong, 2018; Lemke et al., 2013; Pradat-Diehl et al., 1999; Wasserman et al., 2015)
Photophobia	Light sensitivity is common following an ABI with high rates of comorbidity with post-traumatic headaches. Exposure to light can result in pain, and discomfort. Photophobia may be present for multiple reasons such as too much light entering the eye or excessive electric impulses to the optic nerve.	(Armstrong, 2018; Digre & Brennan, 2012; Hazin et al., 2009; Magone et al., 2014)
Stereopsis	Stereopsis refers to depth perception. Significant proportions of individuals have shown deficits in stereopsis following an ABI. Some studies have suggested that challenges in depth perception may be the result of high level cortical injury.	(Armstrong, 2018; Ciuffreda et al., 2012; Schlageter et al., 1993)
Visual field loss	Visual field loss or hemianopia has been found to occur in 60% of individuals with a brain injury as a result of a motor vehicle accident and concurrent damage to the occipital lobe. With 53% of these patients having multiple lesion sites in the occipital lobe. Individuals with blast injuries have shown a reduced incidence of hemianopia (36%). Visual field loss can be complete or partial and can become worse over time.	(Bruce et al., 2006; Lemke et al., 2016; Walsh et al., 2015)
Pupillary function	Pupillary response in either the acute or chronic phase of an ABI is typically measured by the neurological pupil index (NPi). Acute pupil reactivity can be used to inform on other potential concerns such as intracranial pressure. NPi scores are recommended as part of the initial clinical examination and a return to normal NPi scores suggests that pupillary function may improve over time.	(Armstrong, 2018; Chen et al., 2014)
Eye movement	Eye movements involve tracking fixed and moving objects, and are used during everyday tasks such as reading and driving. In those with blast injuries eye movement disorders have been reported as high as 90%. FMRI studies have implicated damage (resulting in signal reduction) to the superior colliculus, oculomotor and nucleus abducens in eye movement disorders for those with chronic TBI. Eye movement disorders can result in blurred vision, convergence insufficiency, and eye strain.	(Alvarez et al., 2012; Samadani et al., 2015; Tyler et al., 2015)

Auditory Deficits

Auditory deficits can arise via two main mechanisms, a direct injury to the area of the brain responsible for auditory processing or damage to the physical structure of the ear or inner ear (Figure 6.4). Both of these injuries impact functional hearing. Individuals can often be misdiagnosed as unresponsive or under-responsive due to hearing loss (Coelho & Hoffer, 2013). Conductive hearing loss (CHL) and traumatic sensorineural hearing (SNHL) loss are both associated with mechanical damage to the structures of the ear itself. CHL occurs when the inner ear is unable to receive information and energy from the middle or outer ear structures, typically this does not affect the clarity of sound only the volume at which it is heard (Coelho & Hoffer, 2013). SNHL occurs further down the hearing pathway and occurs when the inner ear is not able to convert mechanical energy into neural energy, this also results in a decrease in hearing volume but can also result in a decrease in hearing clarity (Coelho & Hoffer, 2013).

Although these are not the only two types of hearing loss which can occur following an ABI, they are the two most commonly associated with mechanical damage to the primary structures of the ear. Auditory impairments should also be suspected if subsequent auditory structures in the hearing pathway are believed to be damaged, such as the brain stem, midbrain, and cerebral cortex. Other common symptoms of auditory deficits are tinnitus, hyperacusis, and perilymph fistulas. Temporal bone fractures are another injury which can cause significant damage to the inner ear. In the case of temporal bone fractures individuals may experience a labyrinthine concussion, which refers to the presence of hearing loss as well as vertigo. Overall two factors influence the recovery of auditory deficits, the location of the trauma along the hearing pathway and the mechanism by which the injury was sustained; with a poorer long term prognosis for those having blast injuries.

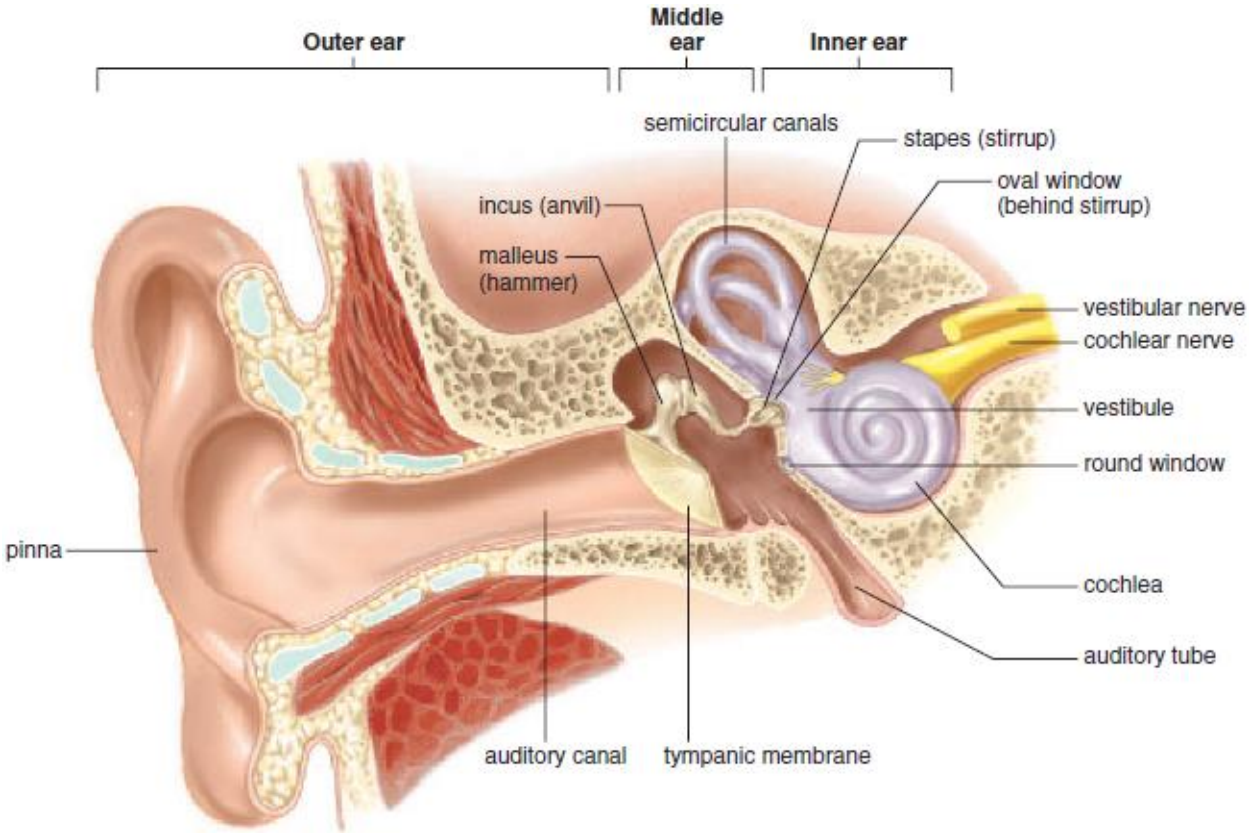

Figure 6.4 Anatomy of the outer, middle, and inner ear.

Vestibular Deficits

Clinical Tip!

Any individual with complaints of vertigo following an ABI should have a Dix-Hallpike maneuver performed.

In the human body, no single structure is responsible for balance (Shepard et al., 2013). Causes of balance problems following traumatic brain injury can be put into two categories: peripheral vestibular origin or central nervous system origin. Currently, between 23%-81% of individuals report balance problems following an ABI (Alsalaheen et al., 2013; Herdman & Clendaniel, 2014; Maskell et al., 2006; Peterson, 2010). Vestibular challenges due to peripheral origins refers to damage inflicted upon the structures of the inner ear and other peripheral structures contributing to vestibular functions. The most common cause of dizziness following an TBI is benign paroxysmal positional vertigo, which occurs when damage to the inner ear (Figure 6.4) results in the semicircular ducts becoming sensitive to gravity. Individuals report short periods of dizziness, light headedness, and falling during daily movement of the head in everyday tasks (Shepard et al., 2013). Unfortunately, there are large variations in both the time these symptoms are experienced as well as predicted recovery. Temporal bone fractures are another injury which can cause significant damage to the inner ear. In the case of temporal bone fractures individuals may experience a labyrinthine concussion, which refers to the presence of hearing loss as well as vertigo. The last major type of peripheral complication resulting in vestibular dysfunction are perilymphatic fistulas. This occurs when the barrier between the middle and inner ear becomes inappropriately connected disrupting signal transmission; this occurs most commonly at the oval window behind the stapes and less frequently at the round window below (Shepard et al., 2013).

Central nervous system causes of vertigo and dizziness can range from post-traumatic seizures to anxiety disorders (Herdman & Clendaniel, 2014; Shepard et al., 2013; Staab, 2000) and occur when damage to the central nervous system is responsible for incidences of vertigo or dizziness. If it is determined that vestibular dysfunction is the result of another existing medical complication such as post-traumatic seizures, that disorder should be addressed first to see if the vestibular dysfunction resolves with the treatment of the underlying condition. Corresponding to this, the nature of the observed vestibular dysfunction will be indicative of the lesion location (Herdman & Clendaniel, 2014).

6.2.3 Pain Syndromes Following ABI

Specific pain syndromes have been identified that have been known to occur more frequently in those with an ABI (Table 6.4). In cases where an individual has cognitive-communication challenges, presents with agitation, and/or displays of non-verbal signs of pain (recoiling from stimuli) pain should be screened for in order to avoid unnecessary suffering (INESSS-ONF, 2015). Pain syndromes can be challenging as pain may manifest itself in a variety of ways following an ABI. Particularly in relation to polytrauma, pain is a common comorbidity for patients. There may associated fractures and soft tissue injury in addition to pain secondary to effects on the central and peripheral nervous systems. Central pain syndrome can be accompanied by hemisensory impairment and typically localizes to one side of the body. Spasticity secondary to central nervous system injury can also be a significant cause of pain. Muscle spasm or spasticity can be a cause of increased pain in limbs with injury/ fracture or pre-existing problems such as degenerative joint disease. Neuropathic pain secondary to brachial or lumbosacral plexopathies or peripheral nerve injury can be a common type of pain syndrome experienced by brain injury patients.

Ultimately, for patients to progress in recovery and rehabilitation post ABI, focus on pain management is an important element of the treatment plan.

Table 6.4 Pain syndromes associated with ABI

Pain Syndrome	Description and clinical presentation
Neuropathic pain	Caused by primary damage or dysfunction to the peripheral nervous system. Can result in chronic symptoms in the form of pain, burning, numbness or tingling. Pain can change in intensity and frequency from day to day (Zasler, 2013).
Central pain syndrome	Caused by damage to or dysfunction of the central nervous system and is usually chronic. Common symptoms include burning and numbness, which can become more severe with touch or colder temperatures. Central pain syndromes may impact large areas of the body, or small areas, such as only the hands and feet. Numbness and occasional sharp pains may also occur (National Institute of Neurological Disorders and Stroke, 2019).
Post-traumatic headaches (PTH)	PTH is the most commonly reported physical complaint following an ABI (Yamaguchi, 1992) and can develop up to months following the primary injury (Young, 2001). PTH should not be viewed as a singular condition, rather it should be regarded as something that may be generated through multiple sources (Horn et al., 2013). The primary types of PTH are tension-type, migraine like, cluster like, and cervicogenic like (Defrin, 2014). PTH do not necessarily indicate a serious underlying issue and are often considered an expected part of recovery. However, if PTH are accompanied by other significant symptoms further investigation is warranted.

6.3 Assessments and Outcomes for Motor and Sensory Deficits

Although there are many measures available to help assess the status of motor and sensory deficits, six common measures are discussed here to help elucidate the assessment of different areas of motor and sensory deficits. Specific considerations with each measure are discussed when relevant.

6.3.1 Berg Balance Scale

The Berg Balance Scale (Berg, 1989) is a discrete measure of mobility and balance and can be found here. The assessment requires an individual to complete instructed tasks (such as unsupported standing, looking behind, and standing on one foot) while maintaining balance. The Berg Balance Scale has shown high inter-rater reliability (r=.98), and high test-retest reliability (r=.98) (Berg et al., 1995). This measure has also shown high concurrent validity between other functional measures (Whitney et al., 2003). The Berg Balance Scale should be used when there is an evident balance impairment and can be used to identify balance consistent with independent mobility. This scale while useful, does have the limitation of a ceiling effect where even with a perfect score (56/56), there may still be higher level balance deficits that can have implications for those returning to active lifestyles or work settings requiring normal balance. In this instance, one would then use a standardized measure such as the community mobility and balance test (Balasubramanian, 2015).

The equipment required to complete the test is a stopwatch, a ruler (30cm) and a chair. This test should be administered by an occupational therapist or physiotherapist. Individuals are scored on each task between 0-4 (0 being poor, and 4 being excellent) for a maximum score of 56. Berg et al. (1992) established that a score ≤45 indicates an increased risk of falling and poor balance. Furthermore, a significant change in functioning is considered true when there is a change of ≥4 if the initial score was between 45-56, ≥5 if the initial score is between 35-44, ≥7 if the initial score is between 25-34, and ≥5 if the initial score is between 0-24 (Donoghue & Stokes, 2009).

6.3.2 Community Balance and Mobility Scale

The Community Balance and Mobility Scale (CB&M) is designed to measure stability, balance, postural control, mobility, and walking ability (Howe et al., 2006). The CB&M and administration instructions are available here. Each of the 13 tasks involved in the assessment are graded on a nominal scale from 0 to 5, for a possible total score of 96. Individuals must have some level of ambulation in order to participate in this assessment. The function of this assessment is to determine whether or not an individual has the mobility and functionality necessary to participate in the community. This assessment requires the use of an 8-meter track and a flight of stairs with an approximate completion time between 20-30 minutes. It has been noted that although administering the CB&M does not require specific training individuals should still be familiar with the assessment for ease of administration (Howe et al., 2006). When examining specific scores on the CB&M it can be useful to compare to them to normative ranges of healthy individuals (Figure 6.5).

Age Group	N	Mean*	SD	95% CI
20-29	24	88.71	3.53	87.2 - 90.2
30-39	27	86.33	5.78	84.1 - 88.6
40-49	23	84.35	4.03	82.6 - 86.1
50-59**	26	77.43	6.55	75.0 - 79.9
60-69**	17	64.94	8.22	60.7 - 69.2
70-79**	4	49.75	6.95	38.7- 60.8

*CB&M scored out of 96

**Significant difference from the group in the previous decade.

Figure 6.5 Normative score ranges on the CB&M taken from Zbarsky et al. (2010).

6.3.3 Visual Analog Scale (Pain)

The Visual Analog Scale (VAS) (Figure 6.6) is a subjective measure of pain. The individual in question marks along the 10 cm spectrum the location that they feel best describes their pain intensity. Once this is complete a ruler is used to measure the distance from the “no pain” location (0mm) to the indicated mark to measure the intensity of pain. The following thresholds are typically used; no pain (0-4mm), mild pain (5-44mm), moderate pain (45-74mm), and severe pain (75-100) (Aun et al., 1986). It should be noted that individuals need sufficient visual function to complete this assessment as it has not been found to be effective explicitly using auditory cues.

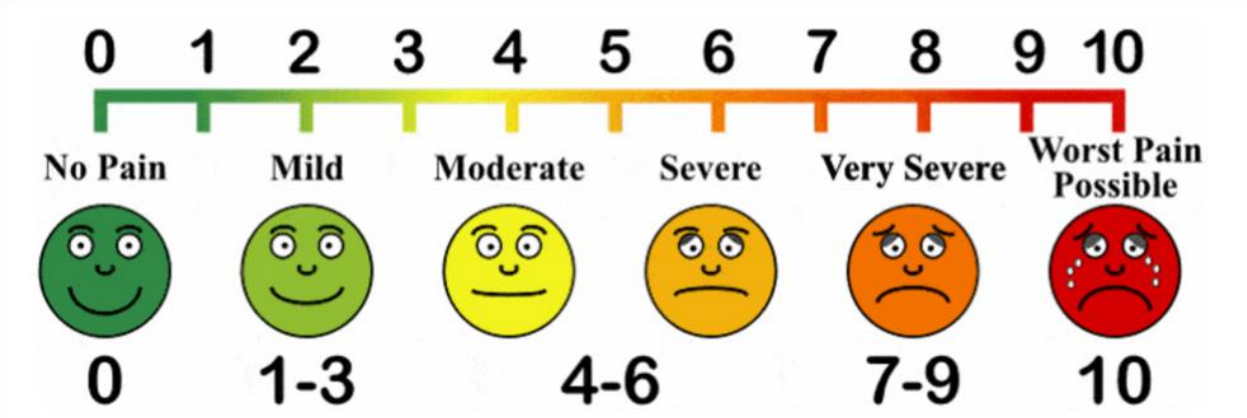

Figure 6.6 The Visual Analog Scale which is a subjective measure of pain intensity.

6.3.4 Modified Ashworth Scale

The Modified Ashworth Scale (MAS) is a clinician administered assessment of spasticity and tone. The MAS measures the resistance during passive stretching in eight areas of the body from the fingers to the soleus. Each area is measured on a nominal scale from 0-4, with 0 representing normal tone and 4 representing extreme rigidity, and is conducted in the supine position. It is recommended that each joint be assessed more than once per assessment but not more than three times to avoid passive stretching benefits from skewing the results. Concurrent validity of this assessment has been seen to be moderate to high, with moderate inter-rater reliability (Benz et al., 2005; Tederko et al., 2007).

The MAS is an open source assessment and can be accessed for free here.

6.3.5 Functional Independence Measure

The Functional Independence Measure (FIM) is a tool to assess the quality of life of patients with a disability. Primarily, the FIM analyzes the functional independence of patients through assessment of various physical and cognitive domains (FIM Instrument Manual, 2014).

The FIM contains 18 items, 13 of which asses motor function (eating, grooming, bathing, dressing, toileting, bowel and bladder control, transfers and locomotion) and five which asses cognitive (communication and social cognition). Each item ranked from 1 – 7 (1= total assistance to complete task and 7= total independence). The lower the score, the more dependent a patient is for that particular task.

The FIM requires a trained assessor to administer it, on average lasting approximately 15-20 min per assessment (A license to use the FIM instrument may be obtained at www.udsmr.org) The FIM can be used to guide discharge decisions or to objectively quantify the progress an intervention or rehabilitation program is making in patient's recovery. The FIM further provides benchmarks which to compare patient progress to, as standardized charts outlining expected function exists for different disability patterns (i.e. brain and spinal cord dysfunction, stroke rehabilitation, etc.).

The FIM has high interrater reliability (0.95), test-retest and equivalence reliability (0.95, 0.92 respectively; (Ottenbacher et al., 1996)), and internal test reliability (α=0.93-0.95, (Dodds et al., 1993)).

6.3.6 Six Minute Walk Test

The six minute walk test (6MWT) consists of an individual walking as far as they can within six minutes back and forth along a marked path (Balke & Civil Aeromedical Research, 1963). The individual should walk at a pace that is comfortable for them and take breaks when needed. Specific considerations for the 6MWT include the use of encouragement, safety, and the number of trials performed. If encouragement is given the administration of it should be standardized and recorded as encouragement has been shown to increase the distance walked (Harada et al., 1999). There is support for individuals having at least 1-2 practice trials before completing any trials where measurements are taken. At least two studies have shown large improvements in total distances between the first three trials performed and therefore practice trials may be useful to help eliminate natural variability in functioning (Domenico Pinna et al., 2000; Du et al., 2009). As this is assessment can be physically exerting for some individuals it is recommended that the administrator have basic life support certification. Normative values for healthy adults range between 400-700m on the 6MWT (Enright, 2003), however, normative values are inconsistent across studies due to differences in methodologies and therefore these values should be interpreted with reasonable flexibility.

6.3.7 Fugl-Myer for Assessment of Sensorimotor Function

The Fugl-Meyer Assessment (FMA) was originally developed as a measure for individuals with stroke to assess motor functioning, balance, range of motion, and sensation (Fugl-Meyer et al., 1975) and can be accessed here. The FMA contains five areas of assessment: motor function, sensation, balance, joint range of motion, and joint pain, for a total of 155 items. Administration of the complete FMA may take approximately 30-35 minutes or even longer in cases where individuals have significant impairment. Individuals are given verbal instructions and scored on a nominal scale with 0=cannot perform, 1=performs partially, and 2=performs fully" for possible score of 226. The FMA requires several different tools to complete, such as a reflex hammer, a goniometer, a blindfold, etc. and should be administered by either an occupational therapist or physical therapist. Proposed interpretation of scores is presented in Table 6.5.

Table 6.5 FMA impairment classification (adapted from Duncan et al. (1994)).

Impairment Severity	Score Range
Mild	>79
Moderate	56-79
Severe	36-55
Very severe	0-35

The inter-rater reliability of the FMA is strong (r=0.93), however, under the sensation domain items related to "light touch" have been found to have poor to moderate item-level agreement (Lin et al., 2004).

6.4 Criteria for Diagnosis

A diagnosis of a motor deficit should be made in the context of the assessment performed and is critical to confirm since this will lead to guiding and informing treatment/intervention decisions. In the above examples each measure has a scoring interpretation, which should be used to determine the presence and severity of a motor deficit.
Similarly, sensory and pain deficits may be more challenging to diagnose since examination is typically more subjective. However, distinguishing the problem is also important to guide management.

Clinical Tip!
Click HERE to see a video on how to complete the Dix-Hallpike Manoeuvre for the assessment of vertigo by BMJ Learning.

6.5 Interventions for Motor Deficits

As previously discussed, upper motor deficits are common following an ABI. The Clinical Practice Guideline for the Rehabilitation of Adults with Moderate to Severe TBI provide two assessment recommendations to support the rehabilitation of motor deficits, which are presented below.

Motor Function and Control Rehabilitation Principles (Section M1 and M3. From www.braininjuryguidelines.org

- *A trained professional with neurological expertise should assess, design, implement and supervise therapy to improve the motor functions of individuals with traumatic brain injury.*

- *Individuals with traumatic brain injury with spasticity should be assessed and provided with a coordinated plan for interdisciplinary management including:*
- *Identification and management of aggravating factors such as pain, bladder or bowel distention, skin irritation and infection.*
- *Use of specific treatment modalities such as serial casting or removable splints*
- *Use of anti-spasticity medications*
- *Rehabilitation interventions that consider range of motion, flexibility and positioning routine.*

- *Any physical treatment approaches provided following traumatic brain injury should take into account any associated orthopedic or musculoskeletal injuries.*
- *Individuals should be given an opportunity to practice their motor skills outside of designated therapy.*

- *Motor therapy programs should be adapted to accommodate the normal environment and activities of the person with traumatic brain injury as much as possible.*

6.5.1 Upper Extremity Interventions

When assessing possible rehabilitation interventions for upper extremities following an ABI there are a few stroke resources that may be clinically relevant to clinicians caring for individuals with ABI. The reader is encouraged to review the via therapy application https://www.viatherapy.org/. This is a mobile application that provides best practice and evidence-based recovery interventions for upper extremity stroke rehabilitation. Clinically it is thought that many of the interventions would be suitable for the ABI population as well. Further, there is a useful clinical algorithm developed by Stinear et al. (2007) for individuals post stroke that may also be of use. This algorithm represents a potential management strategy when dealing with symptoms of UMNS. It should be noted that this algorithm makes use of motor-evoked potentials (MEPs) present in the motor cortex as the initiating point of this trajectory.

Constraint Induced Movement Therapy

Q1. What are the three principle components of constraint induced movement therapy?

1. Intensive motor training of the affected limb.
2. Motor restriction of the less affected limb.
3. Training of behavioral techniques which help translate gains to real world function.

Constraint induced movement therapy (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 CIMT is more commonly used and studied in stroke populations (strong evidence), there is limited evidence of its positive effects on the ABI population. To be eligible for CIMT the following minimum motor criteria is recommended: 10° wrist extension, 10° thumb abduction, and 10°finger extension (Taub & Uswatt, 2006). **CIMT is supported by INESSS ONF guidelines**.

Figure 6.7 An example of an individual performing a task while receiving CIMT.

Functional Fine Motor Skills

Q2. What tasks can be considered interventions for the rehabilitation of functional fine motor skills?

1. Meal preparation
2. Finger sequence tasks
3. Block assembly

Symptoms experienced following an ABI which affect the upper extremities can include weakness, loss of dexterity, and slow movement (Mayer, 1997). Although gross motor function may return in the acute stage after injury, fine motor skills may take much longer to recover and can cause significant frustration during that time period. There are many functional motor tasks which can be practiced to improve fine motor control over time. Both meal preparation (dexterity) and finger sequencing tasks (speed) have moderate evidence to support their use for improving fine motor skills following an ABI (Korman et al., 2018; Neistadt, 1994). Interventions like meal preparation also align with the Clinical Practice Guideline for the Rehabilitation of Adults with Moderate to Severe TBI in that they are functionally relevant to the individual.

Click here to see the complete evidence for Interventions for Fine Motor Coordination in ERABI

Virtual Reality

Q3. What areas of upper motor function has virtual reality (VR) been shown to improve specifically in ABI populations?

1. Dexterity
2. Reaching accuracy
3. Dominant hand speed
4. Range of motion

There is moderate evidence from two studies (Mumford et al., 2012; Sietsema et al., 1993) that VR has been effective for improving some areas of motor function specifically in individuals with an ABI. It should be noted that both of these studies combined VR training with usual care. VR programs are challenging to evaluate in that they can take on many forms (tracking, reaching, etc.) and are not often applied consistently for easy comparisons. However, if there is an opportunity to participate in VR therapy and usual care is not impacted, there is no observed harm to participation.

Unfortunately, to our knowledge there are no guidelines for the use of VR therapy in the rehabilitation of upper motor deficits following an ABI. However, if virtual reality is going to be used as an intervention for motor rehabilitation, Levin et al., (2015) highlight that virtual reality should be functionally relevant, incorporate principles of neural plasticity, and provide feedback to enhance learning.

6.5.2 Lower Extremity Interventions

A few general rehabilitation principles have been recommended by the Clinical Practice Guidelines for the Rehabilitation of Adults with Moderate to Severe TBI and are presented below. Additionally, it should be noted that partial body weight supported gait training does not provide any additional benefits compared to traditional rehabilitation.

Motor Function and Control (Section M2. From www.braininjuryguidelines.org)

- *Gait re-education is recommended to improve mobility after traumatic brain injury*

- *Specific repetitive training interventions to increase functions post traumatic brain injury are recommended, such as sit-to-stand, functional reaching and balance, and gross motor coordination of the lower extremities*

- *For individuals with traumatic brain injury who are unable to ambulate over ground, gait training with partial support with a harness and/or hydrotherapy should be considered.*

Sit-to-Stand Exercises

Q4. What strength of evidence exists for sit-to-stand exercises in the rehabilitation of lower extremities following an ABI?

1. Moderate. There is evidence from one randomized control trial (Canning et al. (2003) that sit-to-stand exercises are effective at increasing the number of sit-to-stand actions within a given time frame, but not exercise capacity, or efficiency.

Sit-to-stand exercises can be modified in a variety of ways to increase or decrease the difficulty of this exercise and are commonly used in senior populations to assist in mobility. In the basic sit to stand exercise an individual will sit on the edge of a stable chair with their feet behind their knees and stand (with or without the use of arm rests). This exercise can be made easier by providing physical support, or made more challenging by asking the individual to cross their arms while standing and sitting, or placing a pillow below the feet to work on areas of balance.

Clinical Tip!
Mobility aids such as canes and walkers are NOT recommended to be used during sit-to-stand exercises as they can move and cause serious falls

Gait Training

Q5. How can individuals of different mobilities participate in gait training?

1. Individuals who are able to ambulate can participate in treadmill training and over-the-ground gait training with no adjustment.
2. Individuals who have difficulty ambulating may use partial body weight supported gait training and/or hydrotherapy to help improve gait.
3. Supports such as handrails and walkers can also be used to help individuals with difficulty ambulating
*It should be noted that partial body weight supported gait training has not been shown to have any added benefits compared to conventional gait training, however it may be used when an individual requires extra mobility support to participate in gait training.

Figure 6.8 An example of a gait training exercise which can be administered by a physiotherapist.

Gait training can involve the use of a treadmill or be done "over-the-ground" and in addition to gait exercises, it can also involve other areas of physical therapy such as balance exercises, muscle strengthening, and range of motion exercises. Treadmill training is typically supervised by a physiotherapist and used in conjunction with physical therapy to help improve gait, muscle strength, and coordination. There is strong evidence (Brown et al., 2005; Esquenazi et al., 2013; Wilson et al., 2006) to suggest that there are limited-to-no benefits for the use of partial body weight supported gait training for those with an ABI. In instances where individuals are not able to sufficiently ambulate to participate in any other kind of gait training, partial body weight support gait training (Figure 6.9) may be used. This recommendation is supported by the Clinical Practice Guideline for the Rehabilitation of Adults with Moderate to Severe TBI. For those who are able to ambulate but have unsteady gaits, gait training has been shown to be effective (Clark et al., 2012; Peters et al., 2014). In an RCT by Clark et al. (2012), it was observed that partial body weight supported gait training resulted in the smallest improvements overall, while the therapist administered gait training protocol resulted in the closest gait parameters to healthy controls.

Key Study

Author/Year/ Country/Study design/PEDro/N	Methods	Outcome
Clark et al. (2012) Australia RCT PEDro=3 N=42	**Population:** *Experimental Group (n=17)*: TBI=11, Stroke=5, Multiple Sclerosis=1; Mean Age=38.7yr; Gender: Male=10, Female=7; Median Time Post Injury=9 mo. *Control Group (n=25)*: Healthy controls; Mean Age=27.8yr; Gender: Male=16, Female=9. **Intervention:** All participants performed 7 alternative gait training methods in a	1. Body weight-support treadmill training without any additional support resulted in greater amplitude, altered timing, and reduced movement stability compared with non-pathologic gait. 2. Manual facilitation by the therapist most closely matched non-pathologic gait for timing and stability.

	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. **Outcome Measure:** Mediolateral Center of Mass Movement, Stride Time, Stability of Movement.	3. 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.

Individuals should be assessed by a physical therapist and prescribed the appropriate frequency, intensity, and supported level of gait training that best aligns with their current mobility status. As individuals progress, adjustments should be made to reduce the number of supports to encourage continued progress.

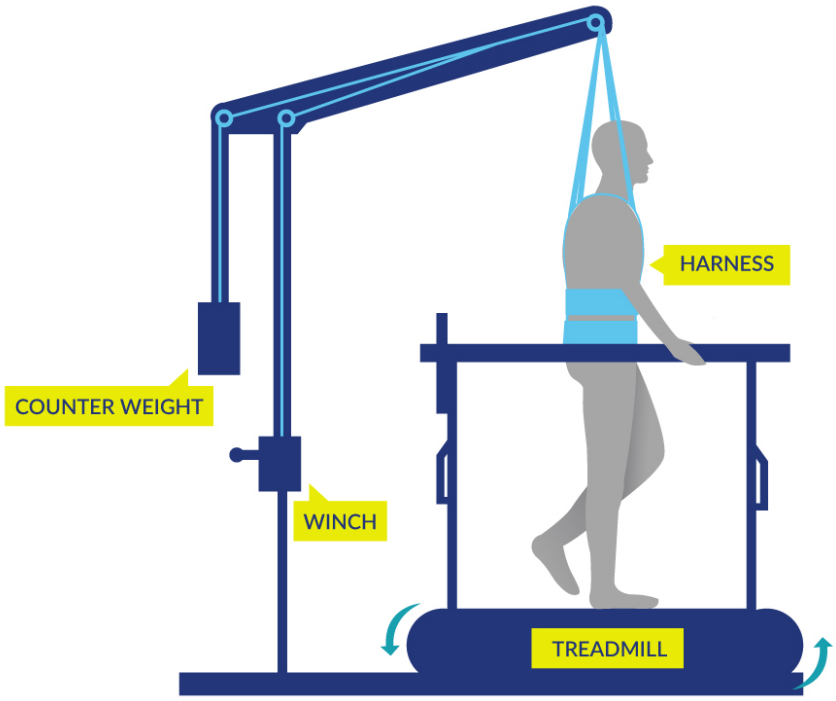

Figure 6.9 The primary components involved in the apparatus used for partial body weight supported gait training (taken from www.scireproject.com).

6.5.3 Combination Upper and Lower Extremity Interventions

Virtual Reality

With the advancement of technology, virtual reality has become mainstream for both recreational and rehabilitation purposes. Although there are a variety of potential interventions (Wii Fit, Balance Board, etc.), moderate to severe ABI specific literature remains limited.

Key Study

Author/Year/ Country/Study Design/N	Methods	Outcome
Ustinova et al. (2014) USA Pre-Post N=30	**Population:** TBI; Mean Age=30.6yr; Gender: Male=10, Female=5; Mean Time Post Injury=6.1yr. **Intervention:** 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 5-6wk). **Outcome Measure:** Berg Balance Scale (BBS), Functional Gait Assessment (FGA), Functional Reaching Test (FRT).	1. BBS scores increased by a mean of 4.5 points (45.6±5.15 to 50.2±4.4, $p<0.01$). 2. FGA scores improved by a mean of 4.6 points (20.3±5.6 to 24.9±4.6, $p<0.05$). 3. FRT scores increased by a mean reaching distance of 2.3 inches (12.5±2.3 to 14.8±2.3, $p<0.01$).

Virtual reality training programs have been shown to improve balance, gait, and reaching in individuals with an ABI (Ustinova et al., 2014). In another study, individuals with an ABI significantly increased their reach as a result of virtual reality training (Schafer & Ustinova, 2013). Although the literature is limited, overall there is moderate evidence to support the use of virtual reality in the rehabilitation of both upper and lower limbs following an ABI (Figure 6.10). However, it should be noted that no specific virtual reality training program has been compared to traditional rehabilitation such as physical therapy and therefore it is undetermined if virtual reality training adds any additional benefits over standard therapies. **The Clinical Practice Guidelines for the Rehabilitation of Adults with Moderate to Severe TBI make a priority recommendation that "Either virtual-reality-based balance retraining program or a conventional balance retraining program can be used to improve balance post traumatic brain injury."**

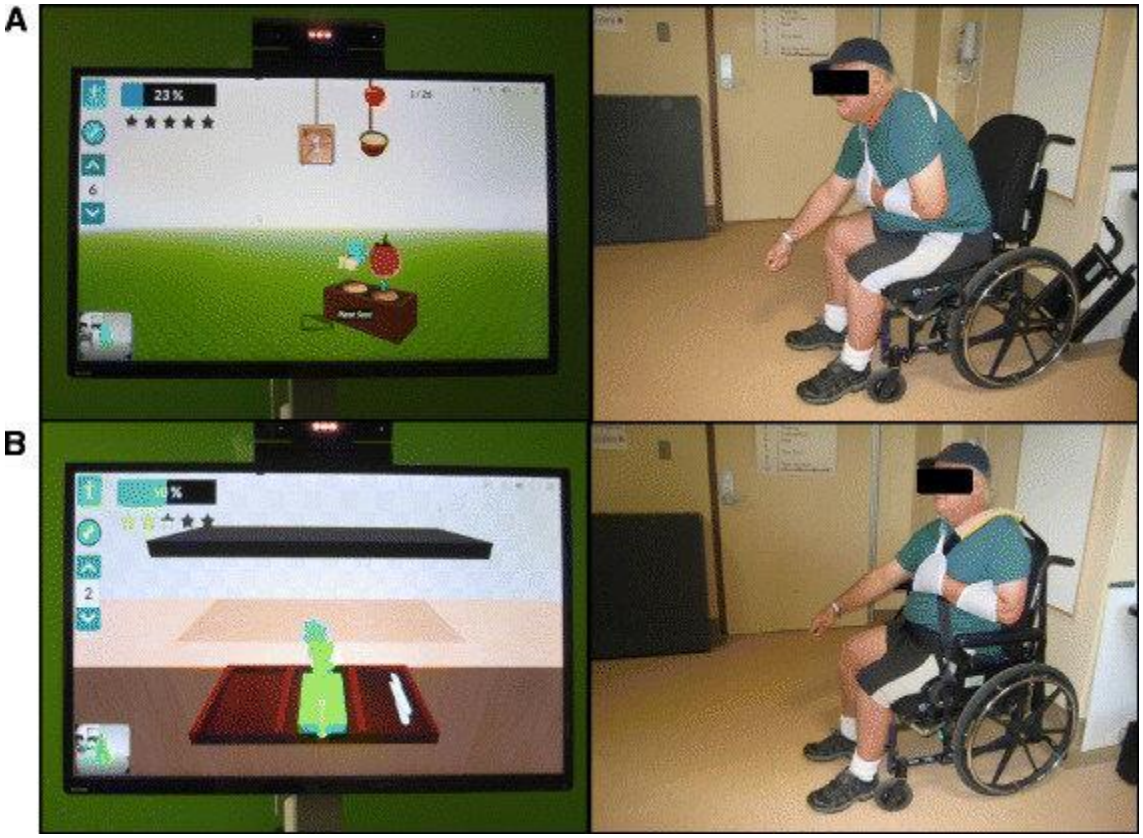

Figure 6.10 An individual completing a virtual reality-based balance exercise as part of their rehabilitation therapy (Sheehy et al., 2016).

Click here to see the Clinical Practice Guideline for the Rehabilitation of Adults with Moderate to Severe TBI recommendations for Motor Rehabilitation

6.5.4 Spasticity Interventions

Oral Medications

Q6. What oral medications have been tested for individuals with an ABI in an attempt to reduce symptoms of spasticity, AND been found to be effective?

1. Oral baclofen.
2. Dantrolene Sodium.
3. Tizanidine.

Oral baclofen can be used to treat spasticity as well, however, the strength of its results have been inconsistent in the literature. As mentioned above, the exact mechanism of action for baclofen is not known, however, it is theorized to be a GABA agonist (Glenn & Whyte, 1990). Currently there is limited evidence supporting its use for those with an ABI, although some positive effects have been seen

(Meythaler et al., 2004). In order to determine the appropriate dosage, titration is required and doses should not exceed 80mg a day.

Key Study

Author/Year/ Country/Study Design/N	Methods	Outcome
Meythaler et al. (2004) USA Case Series N=35	**Population:** TBI=22, ABI=6, Stroke=7; Mean Age=31yr; Gender: Male=22, Female=13. **Intervention:** Oral baclofen regimen beginning at 5 mg 3x/day increased per protocol to 80mg/day. Follow-up occurred between 1 and 3mo after initiation of oral baclofen. **Outcome Measure:** Ashworth Rigidity Scale (ARS), Spasm Frequency Scale (SFS), Deep Tendon Reflexes (DTR).	1. Mean dose was 57±26mg/day for all patients and 55 ± 28mg/day for patients with TBI. 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. 3. No significant changes in lower extremity spasm scores were observed. 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>0.05). 5. Upper extremities showed no significant changes for tone, spasm frequency, or reflexes (p>0.05).

Other medications which have been used to treat spasticity following an ABI are dantrolene sodium, tizanidine, and diazepam. Dantrolene sodium directly effects skeletal muscle fibers by modifying the release of calcium from the sarcoplasmic reticulum resulting in reduced intensity of muscle contraction (Mayer & Esquenazi, 2013). Unfortunately, treatment with dantrolene sodium is only recommended for short term low-intensity treatment of spasticity as those with significant muscle over activity have not been shown to respond well to it (Mayer et al., 1973; Mayer & Esquenazi, 2013). Tizanidine (TZD) has been shown to decrease muscle response to passive stretching in those with ABI (Meythaler et al., 2001). However, another study identified that onabotulinumtoxinA was safer than TZD, produced a greater decrease in spastic symptoms and could be considered as a first line of therapy for spasticity (Mayer & Esquenazi, 2013; Simpson et al., 2009) . Negative side effects associated with TZD are dry mouth, hypotension, sedation and generalized fatigue (Taricco et al., 2000). Diazepam has been used to treat spasticity due to its general highly sedating effects, which increase central inhibition; it is not used as a front-line intervention due to the extreme sedation it can cause (Mayer & Esquenazi, 2013).

The Clinical Practice Guideline for the Rehabilitation of Adults with Moderate to Severe TBI indicate that oral baclofen, tizanidine, or dantrolene sodium may be used to manage symptoms of spasticity following an ABI, however clinicians should monitor individuals for potential side effects (INESSS-ONF, 2015).

Q7. What interventions for spasticity are recommended by the Clinical Practice Guidelines for the Rehabilitation of Adults with Moderate to Severe TBI?

1. Botulinum toxin therapy (BoNT) combined with physical/occupation therapy when appropriate.
2. Oral baclofen.
3. Tizanidine.
4. Dantrolene.

5. Intrathecal baclofen *Should only be considered when spasticity is severe and other treatment options have been exhausted.

Botulinum toxin type A

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 induces local muscle paralysis, thereby alleviating hypertonia caused by excessive neural activity (Jankovic & Brin, 1991). Typically, BTX-A is used in situations where the primary challenged is focal spasticity. **Although there are limited studies evaluating the use of BTX-A in individuals with an ABI specifically, BTX-A has been shown to reduce the symptoms of spasticity in both upper and lower extremities** (Clemenzi et al., 2012; Intiso et al., 2014; Mayer et al., 2008; Yablon et al., 1996) **and is supported by the Clinical Practice Guildeline for the Rehabilitation of Adults with Moderate to Severe TBI.**

Key Study

Author/Year/ Country/Study Design/PEDro Score/N	Methods	Outcome
Intiso et al. (2014) Italy Pre-Post N=22	**Population:** ABI=16, Cerebral Palsy=6; Mean Age=38.1yr; Gender: Male=12, Female=10; *Brain Injury*: Mean Time Post Injury=3.8yr. **Intervention:** Patients with severe spasticity of the upper and lower limbs received injections of onabotulinum toxin A (BoNT-A; up to 840 IU). **Outcome Measure:** Modified Ashworth Scale (MAS), Glasgow Outcome Scale (GOS), Frenchay Arm Test (FAT), Barthel Index (BI), Visual Analog Scale, Visual Analogue Scale–Pain (VAS).	1. Seventeen patients had spastic hemiparesis and 5 had paraparesis. 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 $p<0.05$) and ankle ($p<0.03$). 3. No significant improvements were seen on the GOS, BI, or FAT at 4 or 16 wk. 4. A significant reduction in pain was seen from baseline (7.6±1.1) to 4 (3.5±0.7) and 16 wk (3.6±0.5) post intervention ($p<0.001$).

Three common botulinum toxin derivatives which can be used are Abobotulinum toxin A, Incobotulinum toxin A, and Onabotulinum toxin A. For focal spasticity a maximum dose of 300-400 Units of BOTOX© is recommended by Allergan. The most commonly reported adverse side effects according to Allergan are pain, muscle weakness, fatigue, and upper respiratory tract infections.

Intrathecal Baclofen

As highlighted by the Clinical Practice Guidelines for the Rehabilitation of Adults with Moderate to Severe TBI, intrathecal baclofen should be reserved for instances where spasticity is severe and other treatment options have been exhausted. 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., 1997), while intrathecal baclofen is able to reach these concentrations. The exact mechanism of action for baclofen is not fully known, however, it is capable of inhibiting mono and poly synaptic reflexes resulting in hyperpolarization of afferent terminals, which is thought to enhance its effects.

There is moderate evidence as to the efficacy of intrathecal baclofen and its ability to reduce symptoms of spasticity (Chow et al., 2015; Margetis et al., 2014; Wang et al., 2016). With a majority of studies finding significant reductions on Modified Asworth Scale scores (Chow et al., 2015; Margetis et al., 2014; Meythaler et al., 1999; Meythaler et al., 1997; Wang et al., 2016) and increased range of motion (Horn et al., 2010; Horn et al., 2005). However, due to the nature of its administration (continuous subcutaneous pump) intrathecal baclofen therapy is associated with significant adverse effects and complications such as infection, pump failure, and tube disconnections and kinking (Gracies et al., 1997). In a study of 42 individuals receiving intrathecal baclofen therapy, 62% of patients experienced complications including pump-site infections and overdoses resulting in sedation and vomiting (Hoarau et al., 2012), which should be taken into consideration with the possibility of managing spasticity with intrathecal baclofen.

Nerve Blocking Agents

If spasticity symptoms are localized, nerve blocking agents may be an effective management strategy. A nerve block involves injecting an agent which compromises and impairs nerve functioning, this effect may be temporary or permanent (Katz et al., 2000). Nerve blocks can be categorized as diagnostic or therapeutic. Short-acting local anesthetics can be used to help diagnose the location of spastic symptoms such as range of motion, muscle stiffness, and motor control (Katz et al., 2000). While therapeutic nerve blocks are tyically more long-acting and are designed to relieve symptoms of spasticity to improve pain and function. A common nerve block agent is phenol which denatures proteins and causes denervation when injected near neural structures. The denervation stems from “loss of cellular fatty content, separation of the mylein sheath from the axon, and axonal edema.” (D'Souza & Warner, 2019). Other common nerve block agents are ethanol, glycerol, ammonium salt solutions, and hypertonic saline (Swerdlow, 1978).

No consensus guidelines exist for the use of nerve block agents for those with an ABI. As with intrathecal baclofen, it is recommended that an individual exhaust all other means of therapy before turning to nerve blocking agents as there can be significant negative side effects. Anatomical candidates for successful chemical neurolysis include: peripheral nerves, saddle blockade, lumbar sympathetic blocks, the celiac plexus, and the neuraxis (D'Souza & Warner, 2019). Contraindications for use are patient refusal, active infection, bleeding disorders, or use of anticoagulant therapy (D'Souza & Warner, 2019). Complications of this procedure are pain at the injection site, bleeding, infection, damage to surrounding muscle or other tissues, and occassionally neuritis (D'Souza & Warner, 2019).

Casting

Casting or serial casting is used when there is limited range of motion due to increased muscle tone or spasticity. In serial casting, muscles are passively stretched and then casted for an extended period of time in an attempt to release/stretch soft tissues. Serial casting has been shown to be effective and safe in both adults and children with spasticity according to the Cincinnati Children’s Hospital Medical Center 2009 Evidence-based care guideline**. Indications for the use of serial casting are decreased range of motion, position of the extremity impairs activities of daily living, prevention of contractures, normalization of muscle tone, and an overall reduction in spasticity. If heterotopic ossification (HO) is suspected casting is not indicated as there will be no resulting increase in range of motion and the severity of HO may increase.** Once a cast is applied it is important to monitor closely for skin integrity, loss of sensation in cast limb, discomfort, and psychological frustration. With respect to frustration, not only should the individual receive as much education as possible (based on their level of cognitive functioning) but family and care

givers should also be educated on what to expect. Familial and caregiver support are strong factors in creating positive outcomes.

Key Study

Author/Year/ Country/Study Design/PEDro Score/N	Methods	Outcome
Moseley et al. (2008) RCT PEDro=8 N=26	**Population:** TBI; *Positioning Group (n=12)*: Gender: Male=11, Female=1; Mean Age=30.8yr; Median Time Post Injury=71d; Median Glasgow Coma Scale (GCS) score=3. *Serial Casting Group (n=14)*: Gender: Male=12, Female=2; Mean Age=32.3yr; Median Time Post Injury=59d; Median GCS score=4.5. **Intervention:** 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 2wk with the elbow in a stretched position. Casts were changed to progress the stretch. After 2wk, the cast was removed, and the participants underwent passive stretching 1hr/wk for 4wk. The second group had passive stretch applied to the elbow flexor muscles for 1hr/day, 5 x/wk. **Outcome Measure:** Torque controlled passive elbow extension, Modified Tardieu Scale.	1. Stretching group received a mean of 13 hr of stretching during the intervention and the serial 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 22º ($p<0.001$) 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 ($p<0.05$).

Overall, there is strong evidence that casting can reduce symptoms of spasticity and contractures in those with an ABI. Short term, casting has been seen to be more effective than passive stretching, however results may not be long lasting (Moseley et al., 2008). Other studies have found casting to be effective for both upper and lower limbs and extremities (Moseley, 1997; Pohl et al., 2002; Singer et al., 2003) and the use of casting is supported by the Clinical Practice Guideline for the Rehabilitation of Adults with Moderate to Severe TBI in cases where contractures and deformity is progressive.

Cannabis

Although there is currently no literature evaluating the effects of cannabis on spasticity for those with ABI meeting our inclusion criteria, a collection of five studies on those with other neurogenic forms of spasticity such as multiple sclerosis (MS), spinal cord injury (SCI), and amyotrophic lateral sclerosis (ALS) have found positive effects. In three randomized controlled trials on the use of Sativex (1:1, CBD to THC) for those with MS and spasticity, significant reductions in spasticity were seen (Collin et al., 2007; Collin et al., 2010; Novotna et al., 2011). A review by the American Academy of Neurology (Koppel et al., 2014) examined 34 studies evaluating the effects of medical marijuana on neurologic disorders and concluded that there is strong evidence for the use of medical marijuana to reduce spasticity and pain. However, there are three primary considerations based on the results of this review. First, this review consisted of large mixed populations with motor and pain symptoms arising from multiple neurologic conditions and therefore the extrapolation of these results to any one population is limited. Second, multiple routes of administration (oral versus inhaled) were compared, it is important to evaluate the specific route as a potential moderating factor when evaluating the likelihood of successful therapeutic benefits. Last, marijuana contains approximately 60 pharmacologically active compounds (Koppel et al., 2014), with different active ingredients demonstrating different levels of efficacy. When considering the use of medical marijuana, the specific active ingredient should be of significant consideration. To assist with the therapeutic use of medical marijuana, The National Academies of Sciences, Engineering, and Medicine published *"The Health Effects of Cannabis and Cannabinoids: The current state of evidence and recommendations for research"* in 2017 which can be used to support clinical practice on a variety of topics.

6.6 Interventions for Sensory Deficits

General principles for the best-practice management of sensory deficits begin with proper assessment. It is recommended that all individuals with an ABI be screened for visual impairment, perceptual deficits, and vestibular function (INESSS-ONF, 2015). For pain syndromes, special attention should be paid to non-verbal signs of pain if an individual has difficulty with oral communication (INESSS-ONF, 2015).

6.6.1 Interventions for Visual Dysfunction

As a variety of visual deficits can occur following an ABI it is important to identify the specific visual intervention that will support a positive outcome based on the nature of the dysfunction. Individuals should always be screened for visual dysfunction following an ABI by an appropriate clinician such as opthalmologists, orthoptists, and other clinicians with expertise in visual rehabilitation.

Prisms

Prisms are lenses that bend light. The usefulness of prisms for those with visual dysfunction is that prisms have the ability to shift visual field perception so that the visual world aligns between both eyes. This can be very useful for individuals with double vision, strabismus (cross-eyed or wall-eyed), or spatial imbalance and other visual processing problems (Padula, 2013). For those with Visual Midline Shift

Syndrome (VMSS), yoked prism lenses are appropriate (Gottlieb et al., 1998). Additionally, to increase visual field and spatial deficits, multiple prisms can be layered or mounted to increase the size of the visual field (Gottlieb et al., 1998; Padula, 2013).

Patching

Patching is also used to treat double vision. A patch can be placed over one eye to block either the central visual field or the peripheral visual field in an attempt to allow the deviating eye to align information with the sensorimotor systems of the body (Padula, 2013). In order for patches to be effective, diplopic field measurement is required to determine the precise size, placement, and shape of the patch needed.

However, patching can lead to its own challenges. Wearing a patch leaves an individual with monocular vision (which can reduce the visual field by up to 25%) and impairs visual acuity, hand-eye coordination, and spatial navigation making everyday tasks such as driving, walking, and eating challenging (Padula, 2013). If an individual is receiving a patch, they should be thoroughly educated on what visual perception changes to expect and how they can maintain their (and others') safety while wearing a patch.

Vison Therapy/Orthoptics

Vision therapy can involve a variety of exercises, tools, and strategies, usually executed under optometric supervision, intended to improve visual processes. There are a variety of interventions that can be a part of vision therapy such as visual tracking training, prisms, occupational therapy, and surgery (Figure 6.11).

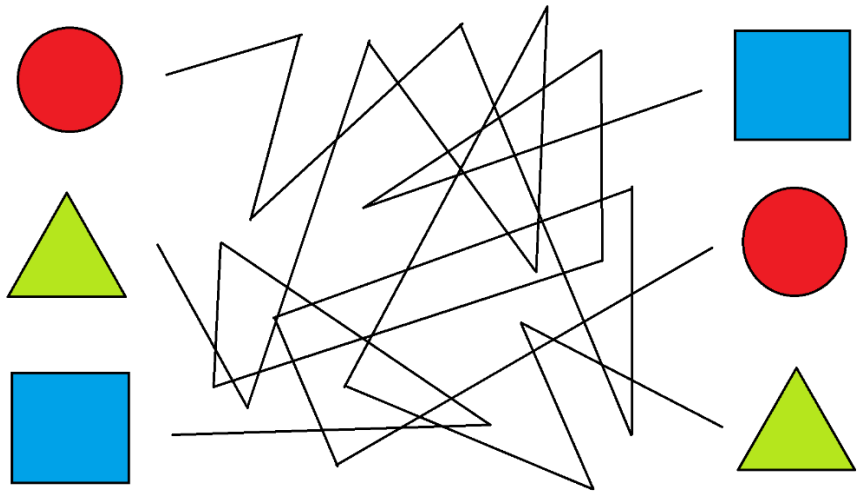

Figure 6.11 An example of a visual tracking training exercise where individuals need to track and follow the path of one shape to its pair on the other side of the trail.

Visual tracking training involves exercises designed to train the brain to align visual processing systems and return to an equilibrium. Visual tracking involves control moving the eyes around the visual field (left to right, up and down) and being able to track moving objects through space. Difficulty with visual tracking can strongly manifest through reading challenges. If an individual frequently needs to restart reading a sentence, skips words, and/or has poor reading comprehension, those may all be indicators of poor visual tracking. Visual tracking training addresses the coordination required for visual tracking through a variety of potential modalities such as computer-based programs, physical activity, occupational therapy, games, and paper and pencil tasks.

Key Study

Author/Year/ Country/ Study design/ PEDro Score	Methods	Outcome
Conrad et al. (2016) USA Pre-Post $N_{Initial}$=19, N_{Final}=13	**Population:** TBI=15, Stroke=3, Organic Brain Syndrome=1; Mean Age=45.2yr; Gender: Male=12, Female=7; Time Post Injury=2.2 yr. **Intervention:** Participants were prescribed home-based computer vergence therapy using software provided (5d/wk for 12wk). Participants were assessed at baseline, 4, 8 and 12wk. **Outcome Measure:** Negative Fusional Vergence, Positive Fusional Vergence, Near Point of Convergence, Vergence Facility, Convergence Insufficiency Symptom Survey (CISS).	1. Negative fusional vergence showed significant improvement over 12wk in blur (p=0.037), break (p=0.003) and recovery (p=0.006). 2. Positive fusional vergence showed significant improvement over 12wk in blur, break and recovery (p<0.0001). 3. Near point of convergence showed significant improvement over 12wk in break (p=0.002) and recovery (p<0.001). 4. Vergence facility showed a significant improvement from baseline to 12wk (p<0.0001). 5. CISS scores improved significantly from baseline to 12wk (p=0.0001).

Overall, there is strong evidence to support the efficacy of visual tracking training, particularly with technology-based programs (Conrad et al., 2016; Kasten et al., 2000; Kasten et al., 1998). One recent study showed that home-based computer visual training significantly improved convergence over time. It should be noted that this protocol was particularly intense with participants completing training five days a week for 12 weeks. Programs which provide feedback have also been shown to be successful (Ciuffreda et al., 2006) and are supported by the Clinical Practice Guideline for Adults with Moderate to Severe TBI (INESSS-ONF, 2015).

Click here to see the ERABI module and complete literature on Motor and Sensory Deficits Following an ABI

6.6.2 Interventions for Vestibular Dysfunction

As a general model of care, it is recommended that all individuals with a traumatic brain injury be screened for vestibular dysfunction, and if appropriate be formally assessed "by a professional specializing in vestibular function." (INESSS-ONF, 2015).

Vestibular and Balance Rehabilitation Therapy

Vestibular and balance rehabilitation therapy (VBRT) is the most common management strategy for vestibular dysfunction following an ABI (Shepard et al., 2013). VBRT programs are usually significantly comprehensive due to the fact that an ABI can cause damage to both primary and secondary structures responsible for balance. There are four broad categories of VBRT which are outlined below (Table 6.6).

Table 6.6 Categories of VBRT (Shepard et al., 2013).

Type	Description
Habituation	Habituation exercises operate under the principle that the body will eventually reduce its response to unpleasant or noxious stimuli through exposure.

Adaptation	Encouraging long-term plastic change is part of the adaptive strategy, exercises are designed to reinforce a reorganization of the vestibular system through similar means of exposure.
Substitution	This approach involves using different eye-movement systems to substitute the eye-movement strategies normally used on specific tasks (such as saccade versus smooth eye movement strategies).
Treatment of benign paroxysmal positional vertigo (BPPV)	As BPPV is thought to be caused by the inappropriate movement of otoconia (bio-crystals within the semicircular canals). Treatment for BPPV involves a variety of maneuvers attempting to make the crystals responsive to gravity again (e.g., Canalith Repositioning Maneuver).

Key Study

Author/Year/ Country/Study Design/PEDro Score/N	Methods	Outcome
Naguib & Madian (2014) Egypt RCT PEDro=5 N=60	**Population:** TBI; Mean Age=30yr; *Group 1 (n=20):* Gender: Male=14, Female=6; Severity: Mild=8, Moderate=7, Severe=5. *Group 2 (n=20):* Gender: Male=14, Female=6; Severity: Mild=8, Moderate=8, Severe=4. *Group 3 (n=20):* Gender: Male=15, Female=5; Severity: Mild=6, Moderate=8, Severe=6. **Intervention:** Participants were randomized to receive betahistine dihydrochloride (48mg/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 2wk, and then every month until recovery. **Outcome Measures:** Recovery time.	1. Group 3 showed the earliest recovery time: complete recovery within 2mo. 2. For Group 2, 80% had complete recovery within 2 months and 20% within 3mo. 3. For Group 1, 85% had complete recovery within 2-3 months, and 15% in more than 3 months. 4. Mean recovery time was significantly longer in Group 1 (62.1d) than in Group 2 (37.6d) and Group 3 (34.4d; $p<0.050$), but there was no significant difference between Group 2 and Group 3 ($p>0.05$).

There is strong evidence to support the use of vestibular recovery programs (Dault & Dugas, 2002; Naguib & Madian, 2014; Peirone et al., 2014) specifically for those with an ABI. A study specifically examining the effects of betahistine dihydrochloride, a physical vestibular rehabilitation program, and the two combined found that individuals recovered significantly faster in the conditions that included the physical rehabilitation program (Naguib & Madian, 2014). Although those in the pharmacological condition also improved over time, there is (limited) evidence to suggest that there is no added benefit for the use of betahistine dihydrochloride in addition to vestibular therapies. Unfortunately, to our knowledge no moderate to severe brain injury guidelines discuss specific therapies for vestibular recovery.

6.6.3 Interventions for Auditory Dysfunction

When there is auditory dysfunction, formal assessment by audiology is indicated. For hearing deficit whether sensorineural of conductive will often has some benefit from use of a hearing aid. For other symptoms such as Tinnitus, there is less evidence for management of this chronic, often debilitating problem. Some strategies for managing this are listed below:

Tinnitus Treatment Recommendations (Henry & Manning, 2019):

- *Sound therapy (use of background sounds of a pleasant and relaxing nature)*
- *TRT – Ear level, broad band sound generator*
- *Masking relief therapy*
- *Hearing aid follow-up or prescription*
- *Relaxation and stress management*
- *Otoacoustic emissions*
- *Tinnitus follow-up*
- *Annual hearing test*
- *Desensitization (hyperacusis)*
- *Speech reading/Aural rehabilitation classes*
- *Consideration of cognitive strategies such as those presented in the book called Tinnitus: A Self-Management Guide for the Ringing in Your Ears, by Jane Henry and Peter Wilson (through group workshop or actually reading the book)*
- *Auditory Brainstem Testing at request*
- *Consideration of other retrocochlear evaluation*
- *ENT consult / follow-up-Consultation*

6.6.4 Interventions for Pain Syndromes

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). General considerations for a model of pain management are outlined below (INESSS-ONF, 2015).

Recommendations for the Management of Pain and Headaches following a TBI (taken from Section P2 at www.braininjuryguidelines.org (INESSS-ONF, 2015).

- *Rehabilitation programs for individuals with traumatic brain injury should have pain management protocols in place, which include:*

- *Regular review and adjustment of mechanisms.*
- *Handling, support and pain relief modalities appropriate to the person's needs.*

- *Education of healthcare professionals and caregivers about appropriate handling of paretic upper limbs during transfers, hypersensitivity and neurogenic pain.*

Click here to see the Clinical Practice Guideline for the Rehabilitation of Adults with Moderate to Severe TBI recommendations for pain management

Cognitive Behavioral Therapy

Cognitive Behavioral therapy (CBT) 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). For more details on specific CBT strategies please see section 5.5.1.1 of Chapter 5 (Mental Health Following Acquired Brain Injury).

Key Study

Author/Year/ Country/Study Design/N	Methods	Outcomes
Gurr and Coetzer (2005) UK Pre-Post $N_{Initial}$=41, N_{Final}=20	**Population:** TBI; Mean Age=44.05yr; Gender: Male=28, Female=13; Mean Time Post Injury=78.7mo. **Intervention:** The CBT program consisted of 3 weekly relaxation group sessions, which were followed by six 30min 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. **Outcome Measure:** Structured Diagnostic Interview, Headache Disability Inventory, Headache Needs Assessment (HANA), Nottingham Health Profile (NHP), Chronic Pain Index (CPI).	1. 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. 2. Headaches occurred a mean 14d per month and the mean length was 10.46hr. 3. Following the intervention, headache intensity (CPI) and frequency decreased significantly (p=0.004). 4. Headache disability, according to results on the HDI and HANA, were significantly reduced (p=0.001 and p=0.02 respectively). 5. According to the NHP, pain was reduced but this was not significant. 6. Emotional well-being as measured by the HDI-emotion and the NHP-emotion subscales was also significantly improved (p<0.05).

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. **Overall, it should be noted that there is limited evidence from ABI specific studies as to the efficacy of CBT in reducing pain post-ABI however the Clinical Practice Guideline for the Rehabilitation of Adults with Moderate to Severe TBI do endorse it as a therapy for pain when appropriate (INESSS-ONF, 2015).**

Other Non-Pharmacological Therapies for Pain Management

A variety of other non-pharmacological interventions have been explored to reduce pain and headaches following an ABI. They are biofeedback, relaxation training, acupuncture, cryotherapy, yoga and mindfulness. Unfortunately, all of these interventions have very limited evidence to support their efficacy in those with an ABI specifically. Although their use may be appropriate in specific cases for individuals with ABI they should be considered on a case by case basis.

Pharmacological Therapies for Pain Management

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. Table 6.7 highlights common oral medications which can be used to manage post-traumatic pain.

Clinical Tip!

An effort should always be made to identify pain generators and treat them as directly as possible before pursuing generalized pain management therapies.

Table 6.7 Common oral pharmacological agents used to treat pain following an ABI (Zasler, 2013)

Analgesics	Antidepressants	Steroids	Short-acting Opioids	Anticonvulsants
- Acetaminophen	- Amitriptyline	- Prednisone	- Morphine	- Carbamazepine
- Aspirin	- Desipramine	- Dexamethasone	- Hydromorphone	- Valproic acid
- Ibuprofen	- Nortriptyline		- Codeine	- Phenytoin
	- Fluoxetine		- Hydrocodone	- Gabapentin
	- Venlafaxine		- Tramadol	- Clonazepam
	- Paroxetine			- Oxcarbazepine
				- Lamotrigine
				- Topiramate
				- Pregabalin
				- Levetiracetam

Pain management is a significant public health issue, with the goal of managing not only an individual's pain, but also the associated psychological effects of pain (Zasler, 2013). For this reason, it is valuable to assess the individual as a whole, including lifestyle and psychosocial factors, before determining the appropriate pharmacological treatment to help manage their pain. While antidepressants and anticonvulsants are typically used to treat neuropathic pain, severe pain may be cautiously managed by narcotics (Zasler, 2013).

The administration of anticonvulsants to treat pain post brain injury has also 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 6.8 summarizes several antiepileptic medications that are used to treat pain post ABI.

Table 6.8 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®)	200mg q 8hr; 100-1600mg/day	Drowsiness, bone marrow suppression, kidney stones
Valproic acid (Depekene®)	250mg q 8hr; 600-2400mg/day	Drowsiness, headache, sleepiness, agitation, mood swings, memory loss
Phenytoin (Dilantin®)	100mg q 8hr; 200-600mg/day	Double vision, imbalance, slurred speech
Gabapentin (Neurontin®)	600mg q 8hr; 200-3600mg/day	Drowsiness, dizziness
Clonazepam (Klonopin®)	0.5mg q 8 hr; 2-7mg/day	Drowsiness, disequilibrium, abnormal behavior
Oxcarbazepine (Tripeptal®)	300-600mg q 12hr; 150-1800mg/day	Drowsiness, lightheadedness, dizziness
Lamotrigine (Lamictal®)	50-100mg q 12hr; 50-200mg/day	Rash, fatigue, stomach upset
Topiramate	25mg q 12hr; 200-400mg/day	Ataxia, impaired concentration, confusion, dizziness, fatigue, speech disturbances, language problems.
Pregabalin (Lyrica®)	300-450mg/day; 150-600mg/day	Drowsiness, dizziness
Levetiracetam (Keppra®)	250-500mg q 12hr; 250-1500mg/day	Drowsiness, dizziness

Unfortunately, there are limited recommendations for the management of pain and post-traumatic headache following an ABI. Pregabalin is the only pharmacological agent supported by the Clinical Practice Guideline for the Rehabilitation of Adults with Moderate to Severe TBI (INESSS-ONF, 2015). As some pain management strategies carry the risk of dependence and addiction it is important to always monitor and adjust pain management strategies appropriately while keeping in mind both the physical and cognitive/psychological consequences of treatments for pain.

6.7 Case Study

Patient Snapshot:

Mr. J…
Is a 42 year-old male who was involved in a high speed MVC resulting in a moderate brain injury and orthopedic injuries (fracture to his right tibia and fibula and right wrist). He was admitted to your outpatient Rehabilitation Program for ABI 10-months post-injury for further therapy.
Lifestyle Factors: Mr. J has a history of a previous MVC (two years past) that resulted in a number of orthopedic injuries and chronic pain. He completed a BSc and had just recently returned to work following recovery from the 1st MVC. He is recently single and has a supportive family who live in another city. He currently uses medical marijuana to manage pain and assist with sleep.
Medical History: Mr. J had an initial GCS of 12, and his duration of post-traumatic amnesia was about four hours and has since resolved. His MRI showed diffuse axonal injury and cognitive screening at the time was suggestive of mild impairment. He had an open reduction and internal fixation for his tibia and fibula fractures and closed reduction of his wrist fracture. There is no history of alcohol or substance abuse, and he has chronic neuropathic pain.
Signs & Symptoms: Mr. J reports ongoing somatic pain symptoms in his right wrist and right leg that include: pain with gripping and pain with walking, chronic headache, light and noise sensitivity, subjective impairment of memory and executive functions, and low mood and easy irritability. At the time of referral, Mr. J expressed that his main goal was to improve his functional independence, irritability, and low mood.

Mr. J's orthopedic surgeon referred him to your out-patient clinic for the management of his motor and sensory challenges. What do you do next?

Assess each area of motor and sensory functioning to further determine where his deficits exist (i.e. *Upper and lower limbs, pain level, vision, and hearing*)

***Note: Mr. J's neurobehavioural management is continued in the Neurobehavioral Case Study which is a part of Chapter 5 of this guidebook.**

Q1. What are some considerations to keep in mind while addressing Mr. J's deficits?

1. What is the real-world impact of Mr. J's symptoms? Which areas of his life do they impact?
2. Are there any temporal relationships to his pain? (i.e. activity related, or only at night)
3. What has Mr. J used to try and manage his chronic pain in the past? Was it effective?
4. What devices, such as a walker, and home modifications in place?
5. Other medications that he's being prescribed by his care team.
6. Mr. J has expressed that he will continue to use medical marijuana during his recovery.

Baseline testing:

Q2. What assessments can you use to further examine the extent of Mr. J's symptoms?

1. History and medication review.
2. Berg balance scale.
3. Visual analog scale.
4. Physical exam.
5. Functional independence measure.

You conduct all these assessments.

History → Mr. J explains that he has pain in his right wrist with activity, especially gripping and using the brake on his four-wheeled walker. He also reports that his left foot tends to stick to the floor and he has pain in his right leg that has a burning quality around the surgical incision and fracture sites. His pain does not stop him from sleeping. For pain, he is using acetaminophen 500 mg TID. He uses ice and heat and finds these can be helpful. He has not had any falls, but is worried about falling when he does not use his walker and when getting in or out of the shower.

Berg balance scale → Mr. J scores a 40. Indicating that he has some balance issues and there's a minor risk of falling.

Visual analog scale → Mr. J rates his pain as a 7/10. The pain is localized to his orthopedic injuries, headache, and is mostly activity related.

Physical exam → Mr. J's physical exam reveals a sensitivity to light, however, his visual acuity is fine. His fractures have healed as expected and you observe that he has some challenges ambulating long distances due to pain in the right leg. You determine he has plantarflexor spasticity in the left leg, and right wrist pain as a result of his fracture. He has good strength and normal sensation in all four limbs.

Clinical Tip!

If Mr. J's light sensitivity was severe or he had other visual concerns, you could refer him to a neuro-optometrist.

Functional independence measure → Mr. J's motor FIM score is 74 indicating he presently has some challenges completing daily activities and self-care.

Q3. Based on these results, what potential therapeutic treatments can you recommend to Mr. J?

1. Physical therapy.
2. Occupational therapy.
3. Pharmacotherapy for his pain.

You determine that he should see a physiotherapist for spasticity and mobility issues, an occupational therapist should evaluate his home for modification, and prescribe him pharmacotherapy for his pain.

Therapy Breakdown:
Physical therapy → Mr. J is receiving gait retraining, range of motion exercises, strength training, and passive stretching as part of his physical therapy.

Occupational therapy → An occupational therapist assesses his home environment and provides Mr. J with a modified walking aid with a single left-handed brake, and a shower bench.

Pain management → You prescribe Mr. J gabapentin starting at 100 TID up to 300 TID to manage his right leg burning pain. You instruct his family doctor that the dose of gabapentin can be increased further if needed for pain. You suggest trying over-the-counter topical diclofenac for his wrist pain. He can continue with acetaminophen, ice, and heat as needed.

***Note: At this time, you would inform other members of Mr. J's care team as to his medications and treatments.**

You've addressed all of Mr. J's symptoms at this point and agree to follow-up in 2-3 months and check on his progress.

***Note: During your break before Mr. J's follow-up , one of his other treating physicians has informed you that they've started treating him with venlafaxine for depression. You refer to your treatment plan and determine that he can continue his use of gabapentin and medical marijuana.**

You're following up with Mr. J three months after you initiated his treatment, what are your next steps?

At this time point you want to reassess the severity of his motor and sensory symptoms and make adjustments to his treatment is necessary. You can use reports from his physical therapy team to add insight into his progress.

Mr. J has improved overall in his mobility and balance, and currently uses walking poles after graduating from the use of a walker. The occupational therapist has provided him with a shower bench, which he feels comfortable using and has not had any significant home accidents. At this point you are no longer concerned about falls. His headache and sensitivity to light has almost disappeared along with most of his pain (this may be due to a combination of generalized recovery, the gabapentin, and/or the venlafaxine). However, he continues to have localized pain at the site of his wrist fracture, and you notice he is still dragging his left leg as a result of his spasticity.

Q4. How can you adjust your management strategy based on Mr. J's current status?

1. Based on the localized pain in his wrist you refer Mr. J to an orthopedic surgeon to consider steroid injections.
2. For the continued spasticity in his left leg you recommend either bracing or botulinum toxin injections. Mr. J decides to make use of a brace (over-the-counter ankle foot orthosis).

After reassessment and adjusting your strategy to address Mr. J's remaining concerns you schedule another follow-up three months from now.

***Note: One of Mr. J's other treating physicians has informed you that they've switched him from venlafaxine to sertraline for his depression. You refer to your files and determine that he may continue on the course of treatment you have prescribed him.**

At your next follow-up Mr. J expresses that his pain is being sufficiently managed, his mobility is functional for his daily life, and he continues to progress nicely. Now what?

After discussing with Mr. J you conclude that he will continue to follow the direction of his physical therapist for his mobility needs. He tried the brace but feels he no longer needs it and he continues to use the walking poles in the community. He doesn't appear to need an adjustment to his medication at this time and you will continue to see him periodically for the continued management of his pain. However, the majority of his recovery has taken place and he is functioning well.

Q5. Identify your 'successes' in managing Mr. J's care?

1. Mr. J has a safe home which he can navigate and use with ease.
2. You have successfully managed both his initial right leg pain and the specific pain related to his wrist fracture.
3. He is walking better with the use of walking poles and a brace to prevent further spasticity.
4. You have communicated with other members of his care team to ensure a coordinated and safe management strategy.

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