3. Motor and Sensory Dysfunction

On behalf of the ERABI Research Group

3.1 Upper Extremity Interventions Post Acquired Brain Injury

Constraint Induced Movement Therapy (CIMT)

Q. What is constraint induced movement therapy? How does constraint induced movement therapy work? What are the minimal requirements to use it in the most affected upper extremity?

Answers

- An intervention directed at improving the function of the more affected upper extremity following brain injury.
- CIMT involves: 1) intensive motor training of the more affected upper extremity (up to 6 hours per day), and 2) motor restriction of the less affected upper extremity (Dettmers et al. 2005).
- Works to counteract "learned non-use" of the most affected limb by removing dependence on the less affected limb.
- CIMT ideally requires that the patient can voluntarily extend their wrist and fingers in the affected hand (20° of wrist extension and 10° of finger extension at the metacarpophalangeal and interphalangeal joints).

Q. What evidence is there for constraint induced movement therapy?

Answers

- There is Level 4 evidence for the effectiveness of constraint induced movement therapy in improving upper extremity use post ABI.

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. 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 and use of the affected limb decreased by 21% at two years post treatment. Given these two studies, CIMT for the upper extremity looks promising.

Hand Splinting

Q. What is the purpose of hand splinting following an acquired brain Injury?

Answer

- Hand splinting (see diagram 1) following an acquired brain injury serves to prevent contractures and deformities and to reduce spasticity.

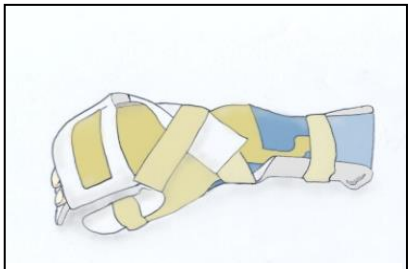

Figure 1. Hand Splinting

The purpose of hand splinting following an ABI is to prevent contractures and deformities and to reduce spasticity. Splints are not likely to be used for functional purposes (Djergaian 1996). 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. 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. Only one randomized controlled trial (RCT) was identified evaluating the effectiveness of hand splinting post ABI.

Q. What evidence is there for the benefit of nocturnal hand splinting post ABI?

Answer

- There is Level 1b evidence that nocturnal hand splinting does not improve range of motion, function or pain control post ABI.

One study evaluated the effect of night time hand splinting in conjunction with conventional therapy compared to therapy alone (Lannin et al. 2003). Overall results did not demonstrate significant benefits of nocturnal hand splinting. There is a need to further research both the biomechanical and neurophysiologic effects of splinting in the individuals with ABI as this practice is used in both acute and rehabilitation settings.

Improving Fine Motor Coordination in Adults with Brain Injury

The negative symptoms of upper motor neuron syndrome, 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 following a brain injury, fine motor deficits may persist and present a considerable challenge for both the individual and the clinicians treating them.

Q. What evidence is there for rehabilitation improving fine motor coordination post ABI?

Answer

- There is Level 1b evidence that both functional and tabletop fine motor control retraining activities result in improved fine motor coordination; however functional retraining activities were more effective in improving fine motor tasks in the dominant hand.
- There is Level 2 evidence that visual feedback grip force training improved tracking and transfer performance.
- There is Level 2 evidence that gesture recognition biofeedback leads to greater improvements in fine motor function of the hand compared to standard repetitive training without feedback.

Discussion

Neistadt (1994) examined fine motor coordination in a group of adult men with brain injury 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 suggests that functional activities may be slightly more effective than table top activities in promoting fine motor coordination in persons with brain injury. Another study found that visual feedback-based training of grip force is useful 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.

The most recent fine motor coordination study compared the use of gesture recognition biofeedback to standard repetitive training without feedback; results showed a significant decrease in task completion time for those who received feedback (Yungher & Craelius 2012). 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.

3.2 Spasticity

Definition of Spasticity

Q. Define spasticity

Answer

- Spasticity is 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).

Spasticity is a common symptom encountered post ABI and is part of the upper motor neuron syndrome (Mayer et al. 1997). Common features of spasticity include increased muscle tone, exaggerated tendon jerks, and clonus.

Treatment of Spasticity

Q. When is treatment of spasticity indicated? What factors should be taken into account when proposing treatment?

Answer

- 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 (Gormley et al. 1997), as well as comorbidities.
- 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.

Oral Anti-spasticity Drugs Post ABI

Q. When should oral anti-spasticity drugs be used for patients with ABI? What drugs are available and what are some of the concerns with using these medications?

Answer

- Oral agents are often used to manage spasticity particularly when a systemic approach to generalized upper and lower extremity spasticity is required (Gracies et al. 1997).
- Oral Baclofen and tizanidine.
- The use of any of drugs must be weighed against potential side effects, such as sedation, which are complicated by the cognitive and behavioural changes associated with brain injury.

Q. What evidence is there for oral anti-spasticity drugs in ABI?

Answer

- There is level 4 evidence that oral baclofen improves lower extremity spasticity but not upper extremity spasticity.

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).

Oral Tizanidine

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 treatments 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.

Botulinum Toxin Injections for Spasticity Post ABI

Q. How does botulinum toxin work in the treatment of spasticity? When should it be used?

Answer

- Botulinum toxin type A (BTX-A) acts at pre-synaptic terminals 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 due to excessive neural activity (Jankovic & Brin 1991).
- It has been suggested that BTX-A may be useful in the treatment of localized spasticity if oral treatments such as benzodiazepines, baclofen, dantrolene sodium or tizanidine cause significant adverse effects (Gracies et al. 1997).

Q. What is the evidence for the use of botulinum toxin to treat spasticity in ABI patients?

Answer

- There is Level 2 evidence that botulinum toxin type A injections are effective in the management of localized spasticity following ABI.
- There is Level 1b evidence to suggest that patients receiving botulinum toxin type A through a single motor point or through multisite distributed injections both show a reduction in spasticity regardless of the drug administration method.

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 treatment. Although pain was also significantly reduced, no significant improvements in function were shown, 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 greater than one year post (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).

For the lower extremity, Fock et al. (2004) reported that BTX-A injections improved measures of walking performance including walking speed, stride length, cadence, dorsiflexion on contact with the ground and passive dorsiflexion. In terms of the administration of BTX-A, Meyer 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-treatment.

Intrathecal Baclofen for Spasticity Post ABI

Q. What is the rationale behind the use of Intrathecal Baclofen? What are the pros and cons of using it to treat spasticity post ABI?

Answer

- A limitation to the use of oral baclofen is the inability to achieve sufficient concentrations in the cerebrospinal fluid in order to modify spasticity without first causing significant sedation (Gracies et al. 1997).
- Intrathecal baclofen is directly administered into the intrathecal space and cerebrospinal fluid at the lumbar level. A subcutaneously placed pump regulates the continuous delivery of medication into the intrathecal space.
- This treatment is more invasive as it requires surgical placement of the pump and is associated with complications including infection, pump failure and tube complications such as kinking or disconnection (Gracies et al. 1997).

Q. What is the evidence for the use of Intrathecal Baclofen in the treatment of spasticity post ABI?

Answer

- There is Level 1b evidence that bolus intrathecal baclofen injections produce short-term (up to six hours) reductions in upper and lower extremity spasticity.
- There is level 4 evidence that prolonged intrathecal baclofen administration results in longer-term (3 months and 1 year) reductions in spasticity in both the upper and lower extremities following an ABI.
- There is Level 4 evidence that intrathecal baclofen results in short-term improvements of walking performance in ambulatory patients, particularly gait velocity, stride length, and step width.

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 (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; Dario et al. 2002; Francisco et al. 2005; Hoarau et al. 2012b; Posteraro et al. 2013; Stokic et al. 2005). 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; however, 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; infections at the operative site was the most frequent complication (20.9%), followed by overdosed with profound flaccidity, sedation, and vomiting (16.3%; Hoarau et al. 2012a)

Two studies also evaluated the functional consequences by assessing walking performance, gait speed and range of motion following a bolus injection of intrathecal baclofen (Horn et al. 2010; Horn et al. 2005). Horn et al. (2005) and Horn et al. (2010) found that although the injections produced changes in joint range of motion during gait, only ankles showed a significant result. Future studies should be conducted using prospective controlled trials or RCTs that include control or placebo groups to further establish the efficacy of intrathecal baclofen for the management of spasticity post ABI.

3.3 Contractures

Definition of Contractures

Q. Define contractures and the underlying pathophysiology.

Answer

- Contractures are the loss of passive movement in joints due to pathology in connective tissue (tendons, ligaments, muscles, joint capsules or cartilage; Backonja 2003).
- Trauma, inflammation, ischemia, infection can produce collagen proliferation. Initially, these collagen fibers may be deposited in a disorganized manner but the collagen can be organized in a linear fashion if the joint is taken through full active or passive functional range. If the joint is left immobile, collagen will tightly pack, resulting in the formation of a contracture. (Backonja 2003).

Common Locations of Contractures

Q. What are common locations for the development of contractures?

Answer

- In the lower extremities, ankle plantar flexion, hip flexion, and knee flexion contractures are common.
- In the upper extremities, elbow flexion and supination contractures are seen, as are adduction and internal rotation contractures of the shoulder.
- Muscles that cross multiple joints, such as the biceps, hamstrings, tensor fascia lata, and gastrocnemius, are predisposed to contracture formation (Backonja 2003).

Prevention of Contractures

Q. List important interventions in the prevention of contractures.

Answer

- Early mobilization
- Range of motion exercises
- Proper positioning
- Orthotic devices
- Antispasticity medications

Treatment of Contractures

Q. List the interventions available for the treatment of contractures

Answer

- Factors that contribute to contracture formation should be treated, i.e. pain, spasticity, inflammation and improper positioning.
- Physical interventions include therapeutic heat (i.e. ultrasound) prior to a stretching program.
- Manual stretching: terminal sustained stretch is essential.
- Serial casting.
- Dynamic splinting.
- If spasticity is a contributing factor: Phenol nerve blocks, botulinum toxin injections, and intrathecal baclofen administration can be considered.
- Orthopedic surgical procedures, such as joint manipulation under anaesthesia, tendon release and tendon lengthening.

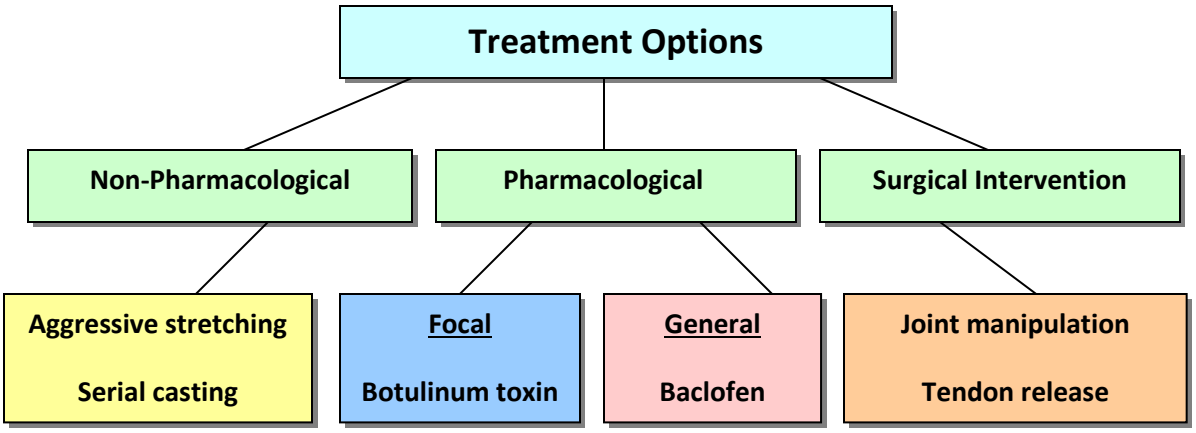

Figure 2. Options for the Treatment of Contractures

Serial Casting for Contractures

Q. What is the rationale behind serial casting for contractures post ABI?

Answer

- Musculoskeletal contractures are often associated with spasticity.
- The theoretical premise for the effect of casting on hypertonia and joint mobility is neuro-physiologically and biomechanically based (Mortenson & Eng 2003).
- From a biomechanical perspective, muscle and connective tissues are likely elongated when immobilized in a stretched position (Mortenson & Eng 2003).

Q. What is the evidence that serial casting is effective?

Answer

- There is Level 1b evidence that serial casting does induce increases in range of motion; however, these effects began to diminish one day post treatment.
- There is Level 2 evidence that serial casting does reduce ankle plantar flexion contractures due to spasticity of cerebral origin.
- There is Level 3 evidence that short duration (one to four days) serial casting has a significantly lower complication rate than longer duration (five to seven days) serial casting; however, there was no difference in range of motion outcome.
- There is Level 2 evidence that casting alone is as effective as the combination of casting and Botulinum toxin injections for treating plantar flexion contractures due to spasticity of cerebral origin.

Seven studies were identified that evaluated the effect of serial casting on change in range of motion of the casted joint. Moseley et al. (2008) found that those patients receiving elbow serial casting showed a greater reduction in elbow contractures post treatment than individuals who receiving passive stretching; however, this improvement diminished quickly (mean reduction was 22°, then 11° one day later). Follow up assessments found no significant difference in improvements between the groups. Evidently, this treatment increases range of motion but the effects are unfortunately not maintained (Moseley et al. 2008). In a another study of casting, Hill (1994) reported that, compared with traditional therapies, casting was effective in improving range of motion and joint angle at which the stretch reflex was elicited in the upper extremity; however there was no difference between groups in performance on functional tasks or in rapid alternating motions. It should also be noted that this RCT received a poor methodological score (PEDro=3); thus, the findings should be interpreted with caution.

For lower extremity, 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 plantar flexion whereas the control condition tended to have overall deterioration of ankle range of motion (Moseley 1997). Verplancke et al. (2005) found that casting was more effective in improving range of motion than passive stretching. This study found that active prophylaxis of leg spasticity using casting is beneficial; however there was no difference comparing persons casted with or without Botulinum toxin (Verplancke et al. 2005). Future studies, with a larger sample size, are needed to examine this further. These studies are promising as greater ankle mobility has been shown to be associated with improved transfer independence (Singer et al. 2003).

In a retrospective case comparison study, Pohl et al. (2002) compared short, one to four days, of casting to a longer duration, five to seven days, 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. From these studies, casting seems to be beneficial.

Adjustable Orthoses for Contractures Post ABI

Q. What is the rationale for use of adjustable orthoses to treat contractures and what are the advantages over serial casting?

Answer

- Similar to casting, an adjustable pre-fabricated orthosis would potentially provide prolonged stretching of an ankle plantar flexion contracture, for example.
- Advantages of the orthosis could include the ease of adjustability and the ability to remove it for short periods of time on a daily basis.

Similar to casting, an adjustable pre-fabricated orthosis would potentially provide prolonged stretching of an ankle plantar flexion contracture. Advantages of the orthosis include the ease of adjustability and the ability to remove it for short periods of time on a daily basis. 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 dorsiflexion passive range of motion. There was 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). More research is needed with an ABI population before conclusions on adjustable orthoses can be made.

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