Educational Module 4

Motor and Sensory Dysfunction

4. Motor and Sensory Dysfunction

4.1 Upper Extremity Interventions Post Acquired Brain Injury

4.1.1 Constraint Induced Movement Therapy (CIMT)

Q1. 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

1. CIMT involves: a) motor restriction of the less affected upper extremity and; b) intensive motor training of the more affected upper extremity.
2. Works to counteract "learned non-use" of the most affected limb by removing dependence on the less affected limb.
3. Ideally the patient should be able to voluntarily extend their wrist and fingers in the affected hand.

Q2. What evidence is there for constraint induced movement therapy?

Answer

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

Discussion

Constraint induced movement therapy (CIMT) is an intervention directed at improving the function of the more affected upper extremity following brain injury. ***The 2 primary components involve: 1) intensive motor training of the more affected upper extremity (up to 6 hours per day); 2) motor restriction of the less affected upper extremity*** [1]. CIMT originates 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 [2].

Although there is evidence in the stroke population to suggest that CIMT is clinically effective, many stroke and ABI patients do not qualify for this type of therapy due to limited movement in the upper extremity. ***CIMT ideally requires that the patient can voluntarily extend their wrist and fingers in the affected hand which limits the number of patients for whom it is applicable.*** A further significant limitation of CIMT is the amount of resources required to implement it [2]. Due to this intense resource requirement, further study of the needed intensity for CIMT is required [1].

Two studies evaluating the effect of constraint induced movement therapy post acquired brain injury were identified:

In the initial study by **Page and Levine** [3], patients received 3 sessions of occupational therapy and physiotherapy weekly for 10 weeks with their less affected arm restrained for 5 hours daily. Each of the 3 patients demonstrated improvements in the amount and quality of use of the affected upper extremity.

The second study by **Shaw et al.** [4] reported significant improvements in both laboratory and real world spontaneous use of the more affected upper limb following two weeks of constraint induced movement therapy in 22 TBI patients.

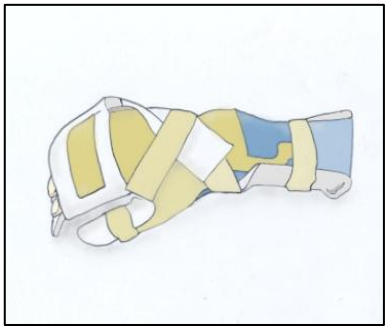
4.1.2 Hand Splinting

Q3. What is the purpose of hand splinting following an acquired brain Injury?

Answer

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

Diagram 1: hand splinting



Q4. What evidence is there for the benefit of nocturnal hand splinting post ABI?

Answer

1. There is Level 1 evidence based on a single RCT that nocturnal hand splinting does not improve range of motion, function or pain control post ABI.

Discussion

The purpose of hand splinting following an acquired brain injury is to prevent contractures and deformities and to reduce spasticity. Splints are not likely to be used for functional purposes [5]. There are biomechanical and neurophysiologic rationales for splinting the spastic hand [6]. 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 was identified evaluating the effectiveness of hand splinting post acquired brain injury. One study evaluated the effect of night time hand splinting in conjunction with conventional therapy compared to therapy alone [6]. 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 acquired brain injury as this practice is common in both acute and rehabilitation settings.

4.1.3 Improving Fine Motor Coordination in Adults with Brain Injury

Q5. What evidence is there for rehabilitation improving fine motor coordination post ABI?

Answer

1. Based on a single RCT, there is Level 1 evidence that functional fine motor control retraining activities results in improved fine motor coordination in addition to re-establishing life skills.
2. There is Level 2 evidence that visual feedback grip force training improved tracking and transfer performance.

Discussion

The negative symptoms of upper motor neuron syndrome, independent of spasticity, include: weakness, slowness of movement and loss of finger dexterity [7]. Although gross motor function may return early in the recovery following a brain injury, persistent fine motor deficits may persist and present a considerable challenge for both the individual and the clinicians treating them.

Two studies were identified that targeted fine motor coordination impairments in people who have experienced ABI. These studies highlight some of the treatment modalities that are being utilized to improve fine motor ability. Using tasks based in both functional and simulated activities and applying the principles of visual feedback the results of these two limited studies are insufficient to guide clinical practice in this area. There is room for both rigorous studies in the use of traditional methods as well as new innovative solutions.

Neistadt [8] examined the effects of puzzle construction and kitchen activities on fine motor coordination in a group of adult men with brain injury. Occupational therapists used two types of activities in coordination retraining for adults with brain injury: 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.

Kriz et al. [9] ***explored the effect of visual feed-back based training on grip force*** – a fundamental aspect of grasping and handling objects. Patients were selected from a larger group based on etiologies of their hand function. 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.

4.2 Spasticity

4.2.1 Definition of Spasticity

Q6. Define spasticity

Answer

1. Spasticity is a common symptom encountered post acquired brain injury and is an element of the upper motor neuron syndrome.
2. 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.
3. Common features include increased muscle tone, exaggerated tendon reflexes and clonus.

Discussion

Spasticity is a common symptom encountered post acquired brain injury and is part of the upper motor neuron syndrome [10]. Spasticity has been formally defined as *"a motor disorder characterized by a velocity-dependent increase in tonic stretch reflexes with exaggerated tendon reflexes, resulting from excitability of the stretch reflex"* [11]. Common features of spasticity included increased muscle tone, exaggerated tendon jerks, and clonus.

4.2.2 Treatment of Spasticity

Q7. When is treatment of spasticity indicated?

Answer

1. Spasticity may require intervention when it interferes with functional abilities such as mobility, positioning or hygiene, or when it is the cause of deformity or pain.
2. 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) and even the locus of injury[12].

Discussion

Some studies have found that spasticity of cerebral origin versus spinal cord injury respond differently to the same medications[13]. Typically, the clinical approach to spasticity is to first employ treatments that tend to be less interventional and costly. Management of spasticity is not unique to brain injury survivors, since it is often

associated with other conditions affecting the central nervous system such as spinal cord injury and multiple sclerosis. However, interventional strategies may differ between diagnoses based on the location of the spasticity (e.g. paraplegia in SCI versus hemiplegia in ABI) and other co-existing morbidities (e.g. cognitive impairment in ABI). Ultimately, multiple strategies may need to be administered concurrently.

4.2.3 Treatment Approach to Spasticity Post ABI

Q8. Describe a treatment approach to the treatment of spasticity

Answer

1. Remove those factors which may increase spasticity.
2. Oral antispastic medications.
3. Botulinum toxin for focal spasticity, where oral antispastic medications are ineffective.
4. Intrathecal baclofen should be used as a last resort for severe spasticity.

4.2.3.1 Oral Antispasticity Drugs Post ABI

Q9. When should oral antispastic drugs be used in the ABI patient, what drugs are available and what are some of the concerns with using these medications?

Answer

1. Oral agents are often used to manage spasticity particularly when a systemic agent to treat upper and lower extremity spasticity is required[14].
2. Although anti-spasticity agents may be used with other medical conditions such as spinal cord injury or multiple sclerosis[14], the effectiveness should not be presumed to be similar for brain injury survivors.
3. Multiple medications have been evaluated to treat spasticity of both cerebral and spinal cord origin. The more common medications include GABA agonists such as baclofen, benzodiazepines, dantrolene sodium which affects ion flux, and agents that affect alpha-2 adrenoreceptors such as tizanidine and clonidine.
4. One particular limitation is the associated cognitive and behavioral changes associated with brain injury.

Q10. What evidence is there for oral anti-spasticity drugs in ABI ?

Answer

1. There is level 1 evidence that oral tizanidine improves lower and upper extremity spasticity when compared to placebo.

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

Discussion

Oral Tizanidine

Meythaler et al. [15] completed a randomized, double blinded placebo controlled cross over trial of tizanidine in the management of spasticity in acquired brain injury. This study evaluated both stroke (53%) and traumatic brain injury (47%) survivors. For both lower and upper extremity, there was a significant decrease in spasticity scores compared to treatment with placebo. However, upper and lower extremity spasm and reflex scores did not improve compared to placebo. A common adverse effect was increased somnolence (41%) compared to placebo (0%). ***Oral tizanidine was found to be effective for improving upper and lower extremity spasticity.***

Study Snapshot

Meythaler JM, Guin-Renfro S, Johnson A, Brunner RM. Prospective assessment of trizanidine for spasticity due to acquired brain injury. *Archives of Physical Medicine and Rehabilitation* 2001;82:1155-1163

- 17 TBI and stroke subjects between 16 and 75 diagnosed with severe chronic spastic hypertonia in one or both lower extremities for at least 6 months were given either 4 mg of tizanidine or a placebo.
- Tizanidine was gradually increased to 36 mg over the next 6 weeks.
- Following a one week tapering off period, those on placebo began taking tizanidine and those on tizanidine began taking placebo.
- The average lower extremity Ashworth score significantly decreased from 2.3 ± 1.4 to 1.7 ± 1.1 ($p<0.0001$), spasms scores decreased rom 1.0 ± 0.9 to 0.5 ± 0.8 ($p=0.0464$). reflex scores decreased from 2.2 ± 1.0 to 2.0 ± 1.1 ($p=0.0883$), and motor tone improved significantly ($p=0.0006$).
- Upper extremity Ashworth scores also decreased significantly from 1.9 ± 1.1 to 1.5 ± 0.9 ($p<0.0001$); however no significantly changes in the upper extremity spasm and reflex scores were noted.
- Although changes in the Ashworth scores were noted while on placebo these changes were not as great as the changes seen while on tizanidine.
- Motor tone of both the upper ($p=.007$) and lower extremities ($p=.0006$) was significantly improved (a significant reduction was noted) while on the tizanidine resulting in an increase in motor strength ($p=.0089$).

Oral Baclofen

Meythaler et al. [16] completed a retrospective study evaluating the use of oral baclofen to manage spasticity in brain injury survivors. ***Pre and post testing of spasticity using the Ashworth scale revealed a significant decrease in lower extremity spasticity scores***; however, results were not significant for upper extremity spasticity scores or frequency of spasms. A noted common adverse effect of the oral baclofen was the onset

of considerable sleepiness in 6 (17%) patients. Although only one study was found, it appears as though oral baclofen does improve lower extremity spasticity.

4.3.2.2 Botulinum Toxin Injections for Spasticity Post ABI

Q11. How does botulinum toxin work in the treatment of spasticity? When should it be used?

Answer

1. Botulinum toxin type A (BTX-A) acts at pre-synaptic terminals to block acetylcholine release into the neuromuscular junction.
2. When selectively injected into a specific muscle, BTX-A is thought to cause local muscle paralysis thereby alleviating hypertonia due to excessive neural activity [17].
3. BTX-A is a relatively new treatment strategy for the management of spasticity in ABI.
4. 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 [14].

Q12. What is the evidence for the use of botulinum toxin to treat spasticity in ABI patients?

Answer

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

Discussion

Three single group intervention studies (2 conducted within the adult population and 1 with a pediatric population), specifically looked at the use of BTX-A for the management of spasticity following ABI.

Yablon et al.[18] reported that ***BTX-A injections into the upper extremities improved range of motion, and spasticity*** as measured by the modified Ashworth scale (MAS) in 21 ABI patients.

Fock et al.[19] reported that ***BTX-A injections into the lower extremities improved measures of walking performance including walking speed, stride length, cadence, dorsiflexion on contact with the ground and passive dorsiflexion***; however, there were no significant improvements in overall spasticity as measured by MAS scores.

In a study conducted in a pediatric cohort, **van Rhijn et al.**[20] reported ***that BTX-A was effective in improving MAS scores up to 5 months post-treatment with concomitant improvements in range of motion.*** Overall the findings suggest

botulinum toxin type A injections may be effective in the management of localized spasticity following ABI.

4.2.3.3 Intrathecal Baclofen for Spasticity Post ABI

Q13. What is the rationale behind use of Intrathecal Baclofen? What are the pros and cons of using it to treat spasticity post ABI?

Answer

1. A limitation of oral baclofen is the inability to achieve sufficient concentrations in the cerebrospinal fluid in order to modify spasticity without first causing significant sedation[14].
2. Intrathecal baclofen refers to direct administration of baclofen into the intrathecal space and cerebrospinal fluid at the lumbar level. For therapeutic treatment, a subcutaneously placed pump is required to provide continuous administration of the medication into the intrathecal space.
3. This treatment procedure is more invasive and is associated with complications including infection, pump failure and tube complications such as kinking or disconnection[14].

Q14. What is the evidence for the use of Intrathecal Baclofen in the treatment of spasticity post ABI?

Answer

1. There is level 1 evidence, based on a single RCT, that bolus intrathecal baclofen injections produce short-term (up to 6 hours) reductions in upper and lower extremity spasticity.
2. There is level 4 evidence prolonged intrathecal baclofen results in longer-term (3 months and one year) reductions in spasticity in both the upper and lower extremities following an ABI.
3. There is level 4 evidence, based on one study, that intrathecal baclofen results in short-term improvements in walking performance, particularly gait velocity, stride length and step width.

Discussion

Ten studies which investigated the efficacy of intrathecal baclofen for the management of upper and lower extremity spasticity following ABI were looked at in detail [21;21-30].

Meythaler et al. [23] confirmed the effectiveness of intrathecal baclofen for decreasing upper and lower extremity spasiticity 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 upper extremity spasticity***

for up to 3 months [22;23] ***and 1 year*** [30] duration. However, all of these studies used a single group intervention design which lacked a placebo control group during the phase when the subcutaneously placed pump was used to provide continuous administration of the medication into the intrathecal space.

Investigations carried out by other research groups have reported similar findings regarding the efficacy of intrathecal baclofen for the management of spasticity post-ABI [24;25;27-29]. However, these studies still lacked a control group thereby limiting the conclusions of their findings.

For the 154 participants in the ten studies identified by this review, ***it appears that intrathecal baclofen is an effective treatment for spasticity, however some adverse effects such as urinary hesitancy*** were reported. Only two of the nine studies examined the long-term effectiveness of the treatment. One study also evaluated the functional consequences by assessing walking performance following a bolus injection of intrathecal baclofen [26]. 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. ***Overall, the results from these 10 studies suggest: 1) bolus injections of intrathecal baclofen produce short-term reductions in upper and lower extremity spasticity post ABI; 2) prolonged Intrathecal baclofen reduces upper and lower extremity spasticity post ABI; 3) intrathecal baclofen may cause short-term improvements in walking performance.***

Table 1: Effects of Intrathecal Baclofen in Modifying Spasticity

Author/Year	N size	Intervention	Results
Meythaler et al., (1996) [23]	**N=11**	• placebo vs bolus injection of intrathecal baclofen intrathecal baclofen (50 ug) or placebo (normal saline)	+ significant reduction in scores on the spasm scale, Ashworth scale and the deep tendon reflex scale. + maximum reduction for all measures occurred 4 hours post-treatment.
Horn et al., (2005)[26]	**N=28**.	• single 50-µg intrathecal baclofen bolus injection was administered to subjects.	+ improvements in gait velocity stride length and step width + reductions in Ashworth scores at 2, 4 and 6 hours post-injection
Dario et al., (2002)[27]	**N=14**	• continuous intrathecal baclofen infusions was administered to patients.	+ decrease in Ashworth score in both lower and upper extremities + reduction in Spasm Frequency Scale scores
Becker et al., (1997)[25]	**N=18**	• Patients received continuous intrathecal baclofen infusion.	+ spasticity was reduced + Mean Ashworth and Spasm Frequency Reduction score was reduced Reduction in spasticity lead to a

Author/Year	N size	Intervention	Results
			reduction in pain.
Meythaler et al., (1997)[22]	**N=12**	• patients were fitted with an infusion pump for continuous intrathecal baclofen delivery for 3 months.	+ spasm frequency and reflex scores significantly decreased after 3 months of treatment
Meythaler et al., (1999) [30]	**N=17**	• patients were surgically fitted with a programmable infusion pump for continuous administration of baclofen	+ intrathecal baclofen treatment resulted in a decrease of Ashworth, spasm, and reflex scores in both upper and lower extremities
Meythaler et al., (1999)[21]	**N=6**	• patients were surgically fitted with a programmable infusion pump into the lower abdominal wall for continuous administration	+ a significant reduction in Ashworth scores, affected lower limb reflex score ,normal side of the lower extremities. + reductions in Ashworth scores on affected side of the upper extremities
Stokic et al., (2005)[24]	**N=30**	• patients received a single 50-µg intrathecal baclofen bolus injection.	+ Ashworth score on the more involved side decreased + H/M ratio decreased bilaterally + F-wave persistence decreased on the more involved side with no change in F/M ratio.
Francisco et al., (2005)[29]	**N=14**	• patients were surgically fitted with an infusion pump for continuous intrathecal baclofen delivery. • head injuries were sustained within the past year	+ reductions from baseline to follow up in upper and lower extremity MAS scores. - DRS scores
Francois et al., (2001)[28]	**N=4**	• intrathecal baclofen infusion was started within 1 month following injury onset.	+ reductions in spasticity, and lower limb Ashworth scores at 6 months post-treatment were reported in three of the four cases.

(+) Indicates statistically significant differences between treatment groups; (-) Indicates non-statistically significant differences between treatment groups.

4.3 Contractures

4.3.1 Definition of Contractures

Q15. Define contractures and pathophysiology

Answer

1. *"Contractures are defined as a fixed loss of passive joint range of movement secondary to pathology of connective tissue, tendons, ligaments, muscles, joint capsules and cartilage"* [31].
2. Trauma, inflammation, ischemia, infection can produce a 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 actively or passively functional range.
3. "*Alternatively, if the joint is immobilized, the collagen matrix will organize in a tightly packed manner, and a contracture will result*"[31].

4.3.2 Common Locations of Contractures

Q16. What are common locations for the development of contractures?

Answer

1. In the lower extremities, ankle plantarflexion, hip flexion, and knee flexion contractures are common.
2. In the upper extremities, elbow flexion and supination contractures are also seen as are adduction and internal rotation contractures of the shoulder.
3. Muscles that cross multiple joints, such as the biceps, hamstrings, tensor fascia lata, and gastrocnemius, are predisposed to contracture formation[31].

4.3.3 Prevention of Contractures

Q17. List important interventions in the prevention of contractures

Answer

Contractures can be prevented with:
1. Early mobilization
2. Range of motion exercises
3. Proper positioning
4. Orthotic devices

Other important measures include:

5. Antispasticity medications

4.3.4 Treatment of Contractures

Q18. List the interventions available for the treatment of contractures

Answer

Once a contracture has developed, a variety of interventions are available:
1. Factors that contribute to contracture formation should be treated, i.e. pain, spasticity, inflammation and improper positioning
2. Physical interventions include therapeutic heat (i.e. ultrasound) prior to a stretching program
3. Manual stretching: terminal sustained stretch is essential
4. Serial casting
5. Dynamic splinting
6. Phenol nerve blocks,
7. Botulinum toxin injections,
8. Intrathecal baclofen administration
9. Orthopedic surgical procedures, such as joint manipulation, tendon release and tendon lengthening

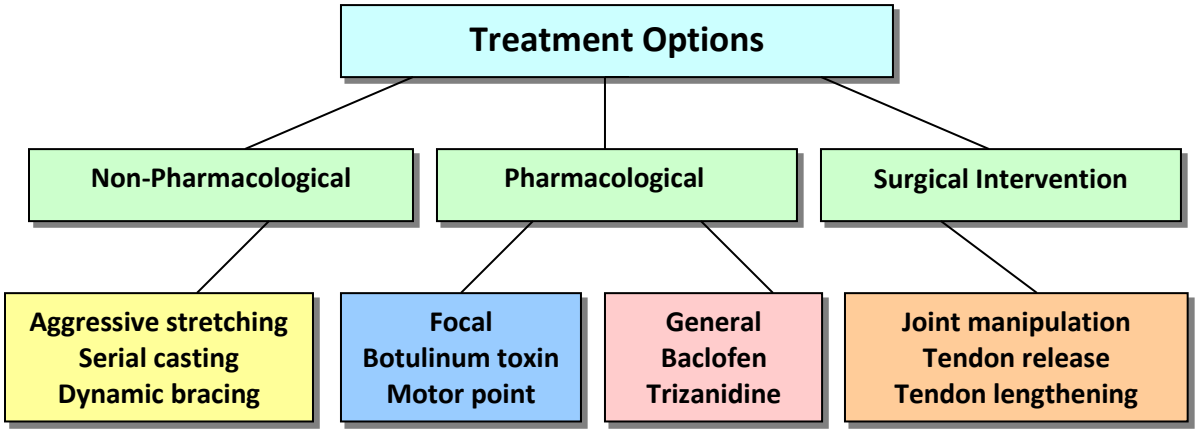
Discussion

Treatment of contractures depends on the severity (p. 295)[32].

Non- pharmacological interventions are effective in tone reduction in a specific muscle groups such as lower limb adductors or plantar flexors. Physical management interventions such as range of motion, positioning, hygiene, etc. should be considered in rehabilitation for patients with disorders of consciousness[33] Occupational Therapy and Physiotherapy programs have been developed to assist with Range of Motion (ROM) and positioning programs.

Pharmacological treatments address abnormal muscle tone and may help with reducing spasticity. However they may have adverse effects on attention and cognition ((p. 294-5)[32] and (p.634)[34]). Focal pharmacological treatment is effective in reducing localized tone. Pain needs to be managed as it can increase spasticity and reduce range of motion exercises.

Figure 1: Treatment Options for the Treatment of Contractures



Meythaler [35] recommends the ***following treatment protocol*** when dealing with the spastic limb prone to contractures:

1. Splinting and ROM
2. Modalities and/or casting
3. Medications:
   - Dantrolene sodium
   - Baclofen
   - Tizanidine
   - Neurolytics (Botulinum toxin, Phenol)
   - It is recommended that one proceed to the first three options before moving to the neurolytics.

Table 2: Evidence for Treatment of Contractures Post ABI

Treatment of Contractures	Level of Evidence (ERABI 2008)	Discussion
Electrical stimulation	Level 4	May be helpful in reducing lower extremity spasticity for up to 24 hours.
Serial Casting	Can be addressed to prevent and treat contractures (p295)[32]	
	Level 2	May reduce ankle plantar flexion contractures due to spasticity.
	Level 3	Short duration (1 to 4 days) has a significantly lower complication rate than longer duration (5 to 7 days)
Dynamic Splinting	Level 1	According to Marshall et al. [36] hand splinting is uses to prevent contractures and release spasticity after acquired brain injury.
Phenol Neural Blocks	Level 4	May temporarily reduce contractures and spasticity at the elbow, wrist and finger flexors for up to 5 months post injection.
Botulinum Neurotoxin Injections	Level 4	Effective for the treatment of localized spasticity and can be managed if oral treatments are associated with significant adverse effects

Intrathecal baclofen	Serves to reduce the side effects of oral baclofen treatment for patients who have arousal, attention and cognitive problems. It can also help control hypertension in ABI. The intrathecal route requires much smaller doses of oral baclofen. However, overdose of intrathecal baclofen can lead to coma and respiratory depression (p.635)[7].	
	Level 1	Reduce upper and lower extremity spasticity over the short-term (up to 6 hours).
	Level 4	Prolonged treatment results in long-term (3 months, and 1 year) reductions in spasticity of the upper and lower extremities.
Surgical intervention	Contractures may assist with skin care and hygiene, avert the development and advance the healing of pressure sores, decrease pain and advance transfers and ambulation. The procedure are generally regarded as last resort to be used in extreme cases to increase function and tends to be limited to more chronic patients (p.295)[32].	

4.3.5 Serial Casting for Contractures

Q19. What is the rationale behind serial casting for contractures post ABI

Answer

1. Musculoskeletal contractures are often associated with spasticity.
2. Spasticity may be reduced by the effect of prolonged stretch or the effects of neutral warmth and prolonged pressure reducing cutaneous sensory input to the spinal cord.
3. Muscles and connective tissues are elongated when immobilized in a stretched position.

Discussion

Musculoskeletal contractures often are associated with spasticity[7]. In a study conducted by Yarkony and Sahgal [37] the incidence of spasticity was has high as 87%. The theoretical premises for the effect of casting on hypertonia and joint mobility are neurophysiologically and biomechanically based [38]. Spasticity may be reduced by the effect of prolonged stretch or possibly the effects of neutral warmth or prolonged pressure which may in turn reduce the cutaneous sensory input to the spinal cord. From a biomechanical perspective, it is likely that muscle and connective tissues are elongated when immobilized in a stretched position[38]. There is also the potential that casting may be a reasonable adjunct to other therapies such as pharmacological interventions.

Q20. What is the evidence that serial casting is effective?

Answer

1. There is level 2 evidence, based on a single RCT, that serial casting reduces ankle plantar flexion contractures due to spasticity of cerebral origin.

2. These is level 3 evidence, based on a single RCT, that casting alone is as effective as casting and Botulinum toxin injections for treating plantar flexion contractures due to spasticity of cerebral origin.
3. There is level 2 evidence, based on a single RCT, that casting alone is as effective as casting and Botulinum injections for treating plantar flexion contractures due to spasticity of cerebral origin.

Discussion

Serial casting has been utilized by physiotherapists for more than 40 years and although there is consensus that this is a useful adjunct to other therapies for the management of spasticity and contracture there has been little empirical data to support it. Overall it was found that serial casting did help to reduce plantar flexion contractures. Results from the seven studies reviewed indicate the following (see Table 3) [39].

Table 3: Summary of the Effect of Serial Casting Techniques in Managing Spasticity

Authors/ Year	n	Intervention	Result
Pohl et al. [40]	105	Conventional (5-7days) vs shorter (1-4 days) serial casting change intervals	Range of Motion (+ improvement post treatment and at 1 month follow-up in both groups) (- differences between groups)
Moseley [41]	9	Short term effects of no casting or stretching (control) vs casting combined with stretching (experimental) using crossover design.	Passive Ankle Dorsiflexion (+ short term)
Kent et al. [42]	18	Effect of unilateral lower extremity serial plaster casting on ambulatory function during post-acute phase of rehabilitation	Holden Scale (- ambulatory improvement between treatment group and matched retrospective controls)
Singer et al. [43]	16	Below knee plaster casts re-applied weekly to increase joint range and muscle extensibility	Ankle Passive Range of Motion (+) Transfer Assistance (+) Rancho Los Amigos Scale (- before compared to 3 months post intervention)
Singer et al. [44]	9	Serial casting to correct spastic ankle equinovarus deformity	Maximal Ankle Passive Range of Motion (+ post intervention and at 6 months) Passive Resistance Torque Angle (+ over casting period)
Verplancke et al. [45]	28	Effects of Standard physical therapy (control) compared to lower leg casting plus saline injections (treatment group 1) and lower leg casting plus Botox injections (treatment group 2) on the development of calf contracture.	Maximal Ankle Passive Range of Motion(- control vs saline) (+ control vs Botox) (- saline vs Botox) Glasgow Outcome Scale (+ treatment groups) Modified Ashworth Scale (+ treatment groups, - control group)

Conine et al. [46]	10	Serial Casting within 14 days of injury for prevention or correction of equinus	Dorsiflexion Range of Motion (+)
Hill [47]	15	Effects of traditional therapy (control) vs. serial casting combined (experimental) using crossover design.	Passive ROM (+) Point of stretch reflex angle elicitation (+) Rapid alternating motions (-) Performance on functional tasks (-)

(+) Indicates statistically significant differences between treatment groups; (-) Indicates non-statistically significant differences between treatment groups. For further details on spasticity and casting post ABI please see Marshall et al.,[39]

4.3.6 Adjutable Orthoses for Contractures Post ABI

Q21. What is the rationale for use of adjustable orthosis to treat contractures and what are the advantages over serial casting?

Answer

1. Similar to casting, an adjustable pre-fabricated orthosis would potentially provide prolonged stretching of an ankle plantar flexion contracture.
2. 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.

Q22. What evidence is there for the use of adjustable orthoses in the management of contractures post ABI

Answer

1. There is level 4 evidence that a pre-fabricated ankle foot orthosis does reduce ankle plantar flexion contractures due to spasticity of cerebral origin.

Discussion

In a single group comparison study conducted by Grissom and Blanton[48] they found ***intervention with a fabricated ankle orthosis demonstrated a significant improvement in ankle dorsiflexion after 2 weeks***; mean gain in dorsiflexion of 20.1 degrees (range: 6-36) (p=0.0078). One significant concern was the relatively high complication rate of skin breakdown that occurred with splinting. Another concern is the very small sample (n=5) of individuals with an ABI who participated in the study.

4.4 Apraxia

4.4.1 Definition of Apraxias

Q23. Define and classify apraxias

Answer

1. Apraxia is a disorder of voluntary movement where one cannot execute a purposeful activity despite the presence of adequate mobility, strength, sensation, coordination and comprehension.

Discussion

Apraxia is common in patients with left hemispheric strokes, especially in lesions involving the left frontal and parietal lobes. Apraxia is a disorder of voluntary movement where one cannot execute willed, purposeful activity despite the presence of adequate mobility, strength, sensation, coordination and comprehension. In other words, patients are unable to perform previously learned tasks and the inability is not explained by weakness, aphasia, or sensory loss (p. 97)[49]. Alternatively, Caplan [49] refers to it as an inability to perform previously learned tasks when such an inability cannot be explained by weakness, aphasia, or sensory loss. The difficulty can be spontaneous and noted during everyday activities (e.g. difficulty with dressing, using utensils, starting the car, turning keys to open doors, and lighting a cigarette). In others the difficulty in performing motor tasks becomes evident when the patient is asked to do something (p. 97)[49].

Table 4: Types of Apraxias

Type	Site of Lesion	Manifestation
Motor or Ideomotor	Often left hemisphere.	Can automatically perform a movement but cannot repeat it on demand.
Ideational	Often bilateral parietal.	Can perform separate movements but cannot co-ordinate all steps into an integrated sequence.
Constructional	Either parietal lobe but right more often than left.	Unable to synthesize individual spatial elements into a whole (e.g., cannot draw a picture).
Dressing	Either hemisphere, right more often than left.	Inability to dress oneself despite adequate motor ability.

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