Case Study 2

28 year old female, head-on collision, RLA-3 minimally responsive

2.1 Problem List

Q1. Develop a problem list for this patient.

2.2 Diffuse Axonal Injury

Q2. Describe Diffuse axonal injuries.

Q3. Describe some of the clinical features seen following DAI.

Q4. How does a DAI impact recovery and rehabilitation?

2.3 Ranchos Los Amigos Scale of cognitive Functioning

Q5. Describe the Ranchos Los Amigos Scale of Cognitive Functioning.

2.3.1 Ranchos Los Amigos Level-III (RLA-III)

Q6. The patient is an RLA-III. Describe how an RLA-III patient typically presents.

2.4 The Galveston Orientation and Amnesia Test (GOAT)

Q7. Describe the GOAT.

Q8. What are its advantages?

Q9. What are its limitations?

Q10. What does a Goat of 75 mean?

2.5 Management of Minimally Responsive and Hypoarousal State

Q11. How should this patient be managed from a cognitive standpoint?

2.5.2 Pharmacological Management of Minimally Responsive State

Q12. Patient is in a prolonged minimally responsive state: How might you try to wake her pharmacologically?

2.5.3 Pharmacological Management of Hypo-arousal States

Q13. How might you treat her hypo-arousal state?

Q14. Describe how these medications work?

Q15. What Evidence is there that they are effective?

2.6 Contractures Post ABI

2.6.1 Defining Contractures

Q16. Define contractures and their pathophysiology.

2.6.2 Location of Contractures

Q17. What are common locations for the development of contractures?

2.6.3 Prevention of Contractures

Q18. Discuss the prevention of contractures.

2.6.4 Treatment of Contractures

Q19. Discuss the treatment of contractures.

Q20. What evidence is there supporting the various treatments of contractures post ABI?

2.6.4.1 Serial Casting for Contractures Post ABI

Q21. What is the rationale behind serial casting for contractures post ABI?

Q22. What is the evidence that serial casting is effective?

2.6.4.2 Adjustable Orthoses for Contractures Post ABI

Q23.What is the rationale for using adjustable orthoses to treat contractures and what are the advantages over serial casting?

Q24. What evidence is there for the use of adjustable orthoses in the management of contractures post ABI?

2.7 Spasticity Post ABI

2.9.1 Definition of Spasticity

Q25. Define Spasticity.

2.7.2 Treatment of Spasticity

2.7.2.1 Indications for the Treatment of Spasticity

Q26. When is treatment of Spasticity indicated?

2.7.2.2 Treatment Approach to Spasticity

Q27. Describe a treatment approach to spasticity.

2.7.2.3 Oral Antispasticity Drugs Post ABI

Q28. When should oral antispastic drugs be used in the ABI patients?

Q29. What drugs are available?

Q30. What are some of the concerns with using these medications?

Q31. What evidence is there of oral antispasticity drugs in ABI?

2.7.2.4 Botulinum Toxin Injections for Spasticity Post ABI

Q32. How does botulinum toxin work in the treatment of spasticity?

Q33. When should it be used?

2.9.2.5 Intrathecal Baclofen for Spasticity Post ABI

Q34. What is the rationale behind use of intrathecal baclofen? What are the pros and cons of using it to treat spasticity post ABI?

Q35. What is the evidence for the use of intrathecal baclofen in the treatment of spasticity post ABI?

Case Study 2

Case Study

A 28-year old female belted driver was involved in a head-on collision with a transport truck during white-out conditions. She was suffering from hypothermia as a result of the long extrication at the scene with a GCS 3. Her injuries at the time included a severe diffuse axonal injury as well as a number of orthopedic injuries which were subsequently operated on and repaired.

The patient was admitted to rehabilitation 2 months post-injury. When reviewed she was able to occasionally follow single step commands (thumbs up) on Rt. Side. She had a flaccid left hemiplegia until recently and was now starting to demonstrate increased tone about the hand and elbow with decreased range of motion in all joints. She had bilateral plantar flexor posturing which could be reduced to neutral position. She was non-verbal, not even mouthing words as he had a tracheostomy. She made occasional eye contact especially with her family who feel she is "in there". There was very little responsiveness to staff but had been seen to track during personal care. She was a Rancho Los Amigos Level III.

2.1 Problem List

Q1. Develop a Problem List for this Patient

Answer

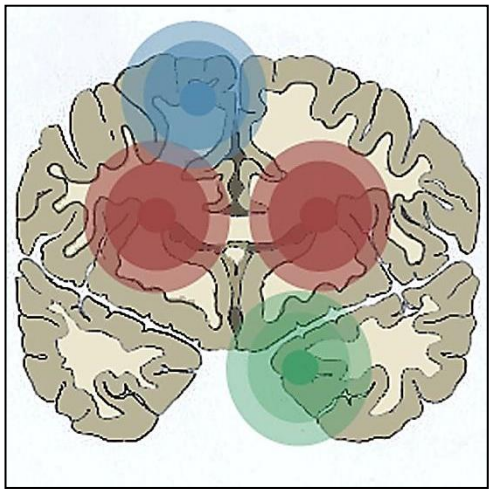
- Hypothermia and GCS 3 at scene
- Severe diffuse axonal injury
- Orthopedic injuries
- Flaccid left hemiplegia

In rehab:

- Occasionally follow single step commands (thumbs up) on Rt. Side
- Increased tone about the hand and elbow with decreased range of motion
- Bilateral plantar flexion with decreased range of motion (ROM)
- Non-verbal
- Tracheostomy
- Occasional eye contact with family
- Little responsiveness to staff

2.2 Diffuse Axonal Injury

Diagram 1: Diffuse Axonal Injury



Q2. Describe Diffuse Axonal Injuries

Answer

1. Diffuse axonal injury (DAI) is seen exclusively following TBI and results from acceleration-deceleration and rotational forces associated with high-velocity impact (motor vehicle accidents)
2. Results in shearing forces which causes the axon to twist and tear which in turn results in death of neurons.
3. Is responsible for the initial loss of consciousness seen in acute TBI.
4. Damage is most often seen in midline structures, in particular the corpus callosum, the parasagittal white matter, the interventicular septum, the walls of the third ventricle and the brainstem in particular the midbrain and the pons.

Q3. Describe some of the clinical features seen following a DAI.

Answer

1. Rostral brain stem involvement results in initial loss of consciousness, poor attention and concentration.
2. Corticospinal tract involvement results in hemiparesis.
3. Shearing of the grey-white matter junction results in slowed mental processing and fatigue.
4. Cerebellar peduncle involvement results in ataxia.
5. Brainstem injury involvement results in dysarthria and dysphagia.

Q4. How does a DAI impact recovery and rehabilitation?

Answer

1. Disrupted connections between nerves results in slowed mental processing, fatigue, poor attention and concentration.
2. Rehabilitation has to be paced and organized (very structured environment) in a manner which allows for these difficulties.

3. Good planning will consider that stamina to perform any skills (functional or otherwise) may be an issue.
4. Poor attention combined with memory difficulties and behavioural concerns may require attendant care.

2.3 Ranchos Los Amigos Scale of Cognitive Functioning

Q5. Describe the Rancho Los Amigos Scale of Cognitive Functioning.

Answer

1. Describes 8 stages of cognitive function that brain injury patients typically progress (see table)
2. Not an outcome measure but rather a global index used to describe awareness, environmental interaction and behavioural competence.
3. Used to monitor recovery.

For further details on the RLA-Cognitive Functioning Scale go to:
http://www.rancho.org/Research_RanchoLevels.aspx

Rancho Los Amigos Levels of Cognitive Functioning [1]	
I	No Response: *Total assistance*
II	Generalized response: *Total assistance*
III	Localized response: *Total assistance*
IV	Confused-agitated: *Maximal assistance*
V	Confused-inappropriate, non-agitated: *Maximal assistance*
VI	Confused-appropriated: *Moderate assistance*
VII	Automatic-appropriated: *Minimal assistance for daily living skills*
VIII	Purposeful-appropriate: *Stand-by assistance*

2.3.1 Ranchos Los Amigos Level III – RLA-III

Q6. This patient is an RLA-III. Describe how an RLA-III patient typically presents.

Answer

1. Patient reacts specifically but inconsistently to stimuli.
2. May follow commands.
3. May show a bias toward responding to some persons, in particular family and friends, but not others.

Discussion

RLA-III is referred to as ***Localized Response***:

- The patient will react specifically but inconsistently to stimuli presented
- The response by the patient is directly related to the stimuli present (patient will turn heard toward noise or focus on objects presented to them)
- If stimulus is painful, you may see the patient withdraw (pull away from the pain) or react vocally to it.
- The patient may show his or her awareness to any discomfort he/she is feeling by attempting to remove tubes, catheters, restraints etc.
- Simple commands may be followed; however the patient may do this in an inconsistent or delayed manner.
- The removal of external stimuli may result in the patient laying quietly.
- Patient may respond to family and friends but not to others [2].

2.4 The Galveston Orientation and Amnesia Test (GOAT)

Q7. *Describe the Galveston Orientation and Amnesia Test (GOAT).*

Answer

The GOAT consists of 10 items regarding orientation to:

1. person: name, address and birth date;
2. place: city/town and building they are in;
3. time: current time, date, month year and date of hospital admission;
4. memory of events both after and prior to the injury [3].

To view the Galveston Orientation and Amnesia Test (GOAT) click here.

Q8. What are the advantages of the GOAT?

Answer

1. The GOAT provides an objective rating of early cognitive recovery eliminating the need for sometimes ambiguous terminology used to describe mental status, such as “confused: [4].
2. Due to its design, the scale has been shown to be useful for assessing patients with a wide range of cognitive impairments [5].

Q9. What are its limitations of the GOAT?

Answer

1. The standard GOAT response format makes administration difficult with nonverbal patients [6].
2. The requirement for oral or written expression may result in penalizing patients who are experiencing deficits of expression but not in orientation or in the retrieval or consolidation of memory [7].
3. An aphasia-specific version of the GOAT has been created; however it requires further evaluation.

Note: While the GOAT does contain items intended to provide an assessment of memory, it is primarily a measure of disorientation. Eight of the 10 GOAT items evaluate orientation while only 2 examine memory [8].

Q10. What does a GOAT score of 75 mean?

Answer

1. The GOAT is scored from 0 to 100.
2. Scores that fall above 75 fall into the range considered normal within the reference group [4;5].
3. In order a patient to be out of PTA, the GOAT score must be above 75 on three consecutive administrations.

Discussion

- The GOAT was ***intended to evaluate orientation to time, place and person and to provide an estimation of the intervals prior to and following the injury for which there is no recall*** [4].
- The *duration of PTA is defined as the period following coma in which the GOAT score is less than 75* [4].
- PTA is considered to have ended if a score of 75 or more is achieved on 3 consecutive administrations [6-8].
- Assessment consists of 10 items regarding orientation to person (name, address & birth date), place (city/town and building they are in) and time (current time, date, month, year & date of hospital admission) as well as memory of events both after and prior to the injury [3].
- ***The GOAT is a brief and simple mental status examination developed for use by health professionals at the bedside*** or in the Emergency Department[4;5].

For a more detailed discussion on the GOAT post ABI please see **ERABI/Assessment of Outcomes Following Acquired/Traumatic Brain Injury**.

2.5 Management of Minimally Responsive and Hypoarousal State

2.5.1 Cognitive Management of Minimally Responsive and Hypoarousal State

Q11. How should this patient be managed from a cognitive standpoint?

Answer

1. Assessment should be conducted by a team with specialized experience to establish the level of awareness and interaction.
2. Patient will need specialized care wherever available.
3. Graded program to increase tolerance to sitting and standing.

ABIKUS Guidelines [9]

Cognitive Management of Minimally Responsive State

1. *"For all patients with a diminished level of consciousness, assessment should be undertaken by a team with specialized experience in profound brain injury to establish the level of awareness and interaction. (pg 28)*

2. *Where patients remain in coma or minimally conscious states, management in specializes tertiary centre should be considered if the local services are unable to meet their needs for specialized nursing or rehabilitation. (pg 29)*

3. *Every brain-injured patient who remains unconscious or is unable to sit themselves up should have a graded program to increase tolerance to sitting and standing. (pg 29)"*

2.5.2 Pharmacological Management of Minimally Responsive State

Q12. Patient is in a prolonged minimally responsive state: How might you try to wake her pharmacologically?

Answer

1. No reliable data to support the use of neurostimulants in the comatose (RLA-I) or vegetative (RLA-II) TBI patient (some evidence for bromocriptine although most clinicians do not find it helpful)
2. Level 2 evidence suggests Amantadine 150 mg BID improves outcomes in patients "emerging" from coma (RLA-III).

3. Neurostimulants such as Methylphenidate have been shown to improve attention (+/- function) in “responsive” patients (RLA IV-VIII) but don’t work so well with RLA-III.

2.5.3 Pharmacological Management of Hypoarousal State

Case Study (continued)

While in rehabilitation, the patient remains at an RLA-III level for several weeks and then is noted to be an RLA-IV whereby she is now responsive but suffers from hypoarousal and is obviously confused and disoriented.

Q13. How might you treat her hypoarousal state?

Answer

1. **Neurostimulants** such as Methylphenidate or dopaminergic medications have been shown to improve attention (+/- function) in “responsive” patients (RLA IV-VIII).
2. ***Methylphenidate*** in an indirect catecholamine agonist with a short half life requiring 2-4 doses per day. There is Level 4 evidence that it enhances recovery and functional status.
3. ***Amantadine*** is a dopamineric agent. It assists with recovery of under-responsive patients and distractibility. There is Level 2 evidence it improves consciousness and cognitive functioning in hypoarousal states.
4. ***Bromocriptine*** is a dopamine agonist. There is Level 4 evidence it improves recovery of TBI patients in the vegetative state and it is used in hypoarousal states although evidence for its use is lacking in this latter group.
5. ***Levodopa-Carbidopa*** increases cerebral dopamine. Has been suggested for hypoarousal state but there is very limited evidence of efficacy and side effects include dyskinesias and cognitive changes.
6. ***Selective serotonin re-uptake inhibitors (SSRIs)*** (Prozac, Zoloft, Paxil, Celexa) inhibit the reuptake of serotonin. Increase the dosage q 4-6 weeks and if treating depression need to commit to 12 month course (or increased likelihood of recurrence)
7. ***Other antidepressants*** such as Effexor and Wellbutrin inhibit serotonin, noradrenaline and dopamine reuptake. No evidence of efficacy

Q14. Describe how these medications work?

Answer

Treatment	Mechanism of Action	Dosage	Concerns

Methylphenidate **(indirect catecholamine agonist)**	Neurostimulant Presynaptic release of dopamine Inhibition of dopamine uptake Inhibition of monamine oxidase Improves learning and memory, attention and distractibility functions in TBI patients (RLA 4-7)	Initiate 5 mg @ 7am and 12 noon, increase by 5-10 mg/day to max 60 mg/day Half-life = 2-4 hours	If ineffective by 30 mg/day then wean in favour of another agent.
Amantadine **(dopaminergic agent)**	Potentiates dopamine (mechanism is unclear) Level 2 evidence of benefit for RLA-3	100-400 mg/day in BID dosing.	Elevated seizure risk when dose >300 mg/day. Hallucinations dose-limiting side effect.
Bromocriptine **(dopamine agonist)**	Dopamine receptor agonist. Been suggested for low level patients but of limited proven efficacy	2-15 mg/day in 2 doses.	High incidence of nausea/vomiting and headaches with increasing doses.

Q15. What evidence is there that these medication are effective?

Answer

Treatment	Level of Evidence	Action and Effects
Bromocriptine (Parlodel)	Level 4	Direct dopaminergic agonist to improve recovery of TBI patients in vegetative state.
Dextroamphetamine	Level 4	Enhances recovery and functional status.
Dexamethasone	Level 5	Decreases intracranial pressure.
Desipramine **Amitriptyline** **(tricyclic antidepressant are largely noradrenergic)**	Level 5	Blocks reuptake of norepinephrine and serotonin in the brain, promoting neurological recovery by improving arousal and initiation.

Protriptyline **(tricyclic antidepressant are largely noradrenergic)**	Level 5	Protriptyline is potential stimulant medication when traditional stimulant medications are ineffective.
Amantadine	Level 1	Improves consciousness and cognitive function.
Sensory stimulation	Level 2	Promotes emergence from coma or vegetative state.

2.6 Contractures Post ABI

Case Study (continued)
While on rehabilitation, the patient developed significant spasticity on the left side and to a lesser extent involving the right lower extremity. There is concern that she is beginning to develop a number of contractures in the affected limbs.

2.6.1 Defining Contractures

Q16. Define contractures and their 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."* [2].
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"* [2].

2.6.2 Locations of Contractures

Q17. 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 [2].

2.6.3 Prevention of Contractures

Q18. Discuss the prevention of contractures.

Answer

Contractures can be prevented with:
- Early mobilization
- Range of motion exercises
- Proper positioning
- Orthotic devices

Other important measures include:
- Antispasticity medication

Diagrams 2-4: Orthotic Devices to Treat Contractures post ABI

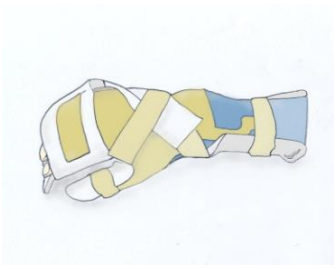


Diagram 2: Hand Splinting

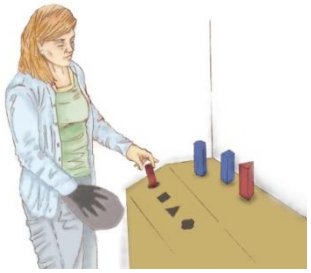


Diagram 3: Peg Board

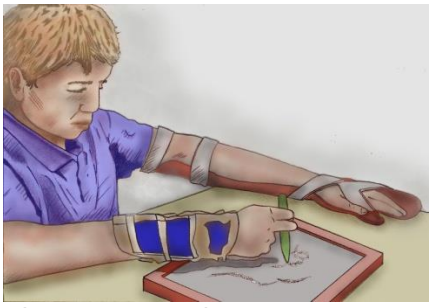


Diagram 4: Drawing

In both Diagrams 3 and 4, the hand that is unaffected is immobilized and the patient is encouraged to use the affected hand.

2.6.4 Treatment of Contractures

Case Study (continued)
While on rehabilitation, the patient went on to develop a left hip flexion contracture, a left knee flexion contracture, a left plantarflexor contracture and a right plantarflexion contracture.

Q19. Discuss the treatment of contractures.

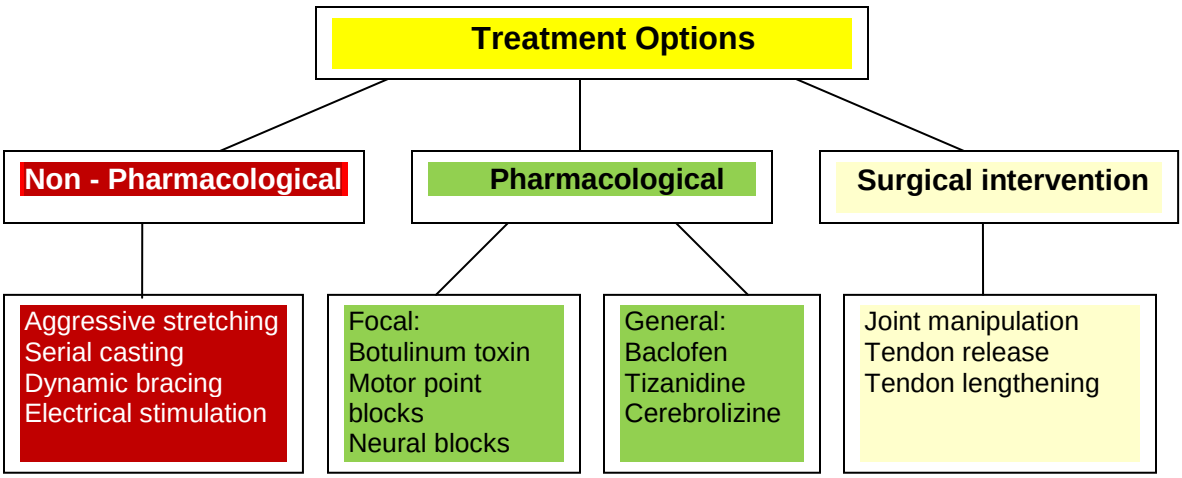
Answer

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 their severity [10].



- **Pharmacological treatments** address abnormal muscle tone and may help with reducing spasticity.
- However, the more general pharmacological treatments may have adverse effects on attention and cognition [10;11].
- Focal pharmacological treatment is effective in reducing localized tone without suffering the possible systemic side effects seen with more general treatments.
- **Non- pharmacological intervention** is effective in tone reduction in a specific muscle groups such as lower limb adductors or plantar flexors.
- Potential treatments and the evidence supporting their use are listed in the table below.

Q20. What evidence is there supporting the various treatments of contractures post ABI?

Answer

Treatment of Contractures	**Level of Evidence**	**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 [10]
	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.[12] 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 [11].
	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 procedures are generally regarded as last resort to be used in extreme cases to increase function and tend to be limited to more chronic patients [10].

ERABI (2010)[12] 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
4. Neurolytics (BOTOX, Phenol)
   - It is recommended that one proceed to the first three options before moving to the neurolytics.

- Physical management interventions such as range of motion, positioning, hygiene, etc. should be considered in rehabilitation for patients with disorders of consciousness [13].

2.6.4.1 Serial Casting for Contractures Post ABI

Q21. 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*** [14]**.**
- ***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*** [15]**.**
- There is also the potential that casting may be a reasonable adjunct to other therapies such as pharmacological interventions.

For a more detailed discussion on contracture post ABI please see **ERABI/Motor and Sensory Impairments Remediation Post Acquired Brain Injury**.

Q22. 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.***
- Based on multiple studies reviewed, overall it was found that ***serial casting did help to reduce plantar flexion contractures.***

For a more detailed discussion of serial casting post ABI please see **ERABI/ Motor and Sensory Impairments Remediation Post Acquired Brain Injury**.

2.6.4.2 Adjustable Orthoses for Contractures Post ABI

Q23. 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.

Q24. 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

- A single group (n=5) comparison study by Grissom and Blanton[16] found intervention with a ***fabricated ankle orthosis resulted in a significant improvement in ankle dorsiflexion after 2 weeks; mean gain in dorsiflexion of 20.1 degrees*** **(range: 6-36) (p=0.0078).**
- A significant concern was the relatively ***high complication rate of skin breakdown that occurred with splinting.***

For a more detailed discussion on adjustable orthoses post ABI please see **ERABI/ Motor and Sensory Impairments Remediation Post Acquired Brain Injury**.

2.7 Spasticity Post ABI

2.7.1 Definition of Spasticity (Jeremy to draw)

Q25. Define Spasticity

Answer

1. Spasticity is a common symptom encountered post acquired brain injury and is an element of the upper motor neuron syndrome [17].
2. 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"*** [18].
3. Common features of spasticity included increased muscle tone, exaggerated tendon jerks, and clonus.

2.7.2 Treatment of Spasticity

2.7.2.1 Indications for Treatment of Spasticity

Q26. 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 [19].

Discussion

- Spasticity is not unique to individuals who have sustained a brain injury.
- Spasticity results in involuntary contractions of synergistic muscles in the extremities, which are clinically manifested as flexor or extensor spasms [20;21].
- Improving patient function is the key to developing any plan to treat spasticity [19].
- For many developing plans to treat their spasticity is done in the early days of rehabilitation, however in those with an ABI, symptoms my not appear for several weeks or months post injury.

2.7.2.2 Treatment Approach to Spasticity Post ABI

Q27. Describe a treatment approach to spasticity

Answer

1. Remove those factors which may increase spasticity.
2. Oral antispastic medications.
3. Botulinum toxin for focal spasticity, when oral antispastic medications are ineffective.

4. Intrathecal baclofen should be used as a last resort for severe spasticity.

2.7.2.3 Oral Antispasticity Drugs Post ABI

Q28. When should oral antispastic drugs be used in the ABI patient, 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 [22].
2. Although anti-spasticity agents may be used with other medical conditions such as spinal cord injury or multiple sclerosis [23] the effectiveness should not be presumed to be similar for brain injury survivors.
3. One particular limitation is the associated cognitive and behavioral changes associated with brain injury.

Q29. What drugs are available?

Answer

1. Multiple medication have been evaluated to treat spasticity of both cerebral and baclofen, benzodiazepines, dantrolene sodium which affects ion flux and agents that affect alpha-2 adrenocrepeptors such as tizanidine and clonidine.

Discussion

Oral Tizanidine

- One RCT investigated the effect of trizanidine in the management of spasticity on individuals recovering from an ABI or stroke [20].
- 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.***

Oral Baclofen

- Oral baclofen was administered to patients post ABI to assist in the management of spasticity [24].
- **Lower extremity spasticity scores improved following** the administration of baclofen; however, upper extremity spasticitiy scores showed no significant improvement.
- A noted common adverse effect of the oral baclofen was the onset of considerable sleepiness in 6 (17%) patients.

For further details on the effectiveness of oral antispasticity drugs on individuals with an ABI see **ERABI/Motor and Sensory Impairments Remediation Post Acquired Brain Injury**.

Q30. What are some of the concerns when using these medications?

Answer

1. One particular limitation is the associated cognitive and behavioural changes associated with brain injury.

Q31. 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

2.7.2.4 Botulinum Toxin Injections for Spasticity Post ABI

Q32. How does botulinum toxin work in the treatment of spasticity?

Answers

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 [25].
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 [22].

Q33. When should it be used?

Answer

1. It has been suggested that BTX-A may be useful in the treatment of localized spasticity if oral treatment such as benzodiazepines, baclofen, dantrolene sodicum or tizanidine cause significant adverse effects.

Q34. 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

- Results of three studies suggest that botulinum toxin type A injections may be effective in the management of localized spasticity following ABI [26-28].
- Yablon et al. [26] 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. [27] noted 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, no significant improvements in overall a spasticity was found.
- van Rhijn et al. [28] reported that BTX-A was effective in improving MAS scores (in a pediatric population) up to 5 months post-treatment with concomitant improvements in range of motion.

For further details on the effectiveness of botulinum toxin injections on individuals with an **ERABI/ Motor and sensory Impairments Remediation Post Acquired Brain Injury**.

2.7.2.4 Intrathecal Baclofen for Spasticity Post ABI

Q35. 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 [23].
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 [23].

Q36. 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

- Meythaler et al. [29] in an RCT, ***confirmed the effectiveness of intrathecal baclofen to decrease upper and lower extremity spasiticity***
- **Subsequent studies, *found the effectiveness of intrathecal baclofen for decreasing upper extremity spasticity for up to 3 months*** [29;30] ***and 1 year*** [31] ***duration.***
- Investigations carried out by other research groups have reported similar findings [32-36].

- Of note: 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.

For further details on the effectiveness of intrathecal baclofen on individuals with an ABI see **ERABI/ Motor and sensory Impairments Remediation Post Acquired Brain Injury**

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